Alan Davies · Alwyn Scott

Starting to Read ECGs

The Basics

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This book is dedicated to the memory of:
Valerie Jane Davies
1955–2000

Foreword

There are several textbooks comprehensively covering the field of ECG interpretation and this book does not attempt to replicate these texts. The purpose of this book is to provide a concise, practical and systematic guide to interpretation of ECGs for the beginner. It not only covers how to perform an ECG but also basic interpretation of a normal ECG before progressing onto the effects of anatomical abnormalities of the heart on the ECG. More complex rhythm abnormalities are described and illustrated to ease understanding. The ECG should always be interpreted whilst considering the clinical presentation of the individual subject from whom it was recorded. The ECG is part of diagnosis and management. Clinical scenarios affecting the ECG are also described in the last two chapters. Each chapter concludes with a quiz for reflective learning and a summary of key points from the chapter. The authors should be congratulated for producing a well presented and easy to understand text which will be useful to medical students, nurses and other allied professionals not only as a starter text but also as an immediate bedside reference manual.

Cambridge, UK — Sarah Clarke

Preface

The ECG is one of the most widely available diagnostic tests used in clinical practice today. Since the first use of the ECG there has been a wealth of books available on the subject, aimed at all sorts of different experience levels and healthcare practitioners. With such a wealth of material already available you may ask yourself what is special or different about this book.

The authors have tried very hard to write a book that is aimed at the absolute beginner. Many make this claim, but we have really tried to strip everything back to essential basics. We pick simple methods that can be used easily in clinical practice. We do not assume any prior knowledge. Above all we wanted the book to be easy to read and attractive, using many photos, images and diagrams to illustrate points and aid in memory retention. We constantly revisit and remind the reader of information already covered to reinforce knowledge. We gradually build on the information given throughout the book, so as not to overload the reader with too much in one go.

This book aims to give the beginner just what they need to know, including information about how to record good quality ECGs. We hope to avoid information overload, although extra information and points of interest are included in information boxes.

We hope you will find this book easy to read, informative, and a useful aid in building your ECG knowledge and confidence in interpretation, whatever your clinical role may be.

Plymouth, UK — Alan Davies

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Chapter 1
How to Record a 12-Lead ECG

Abstract The heart is located in the chest between the lungs in the mediastinum. It is surrounded by a protective sac called the pericardium (Fig. 1.1). Essentially the heart is split into four functional chambers; a left and right atrium, and a left and right ventricle (Fig. 1.2). Deoxygenated blood (blood with no oxygen in it) is emptied into the right atrium via the vena cava. The inferior vena cava returns blood from the lower portion of the body as the superior vena cava returns blood from the higher portion. This blood is then pumped through the tricuspid valve into the right ventricle. Blood is then passed into the lungs via the pulmonary artery where it is oxygenated. Oxygenated blood then returns from the lungs into the left atrium where it can be pumped to the rest of the body by the powerful left ventricle, via the aorta (Fig. 1.3). The cells responsible for the contraction of the heart muscle are called myocytes. Apart from the hearts mechanical function as a pump it also has an electrical system governing the rate at which the heart beats, controlling in turn how slow or fast the blood and oxygen gets pumped to all the organs and tissues in the body.

Keywords Electrophysiology • Cardiac anatomy • Electrode placement • Recording

Physiology

The heart is located in the chest between the lungs in the mediastinum. It is surrounded by a protective sac called the pericardium (Fig. 1.1). Essentially the heart is split into four functional chambers; a left and right atrium, and a left and right ventricle (Fig. 1.2). Deoxygenated blood (blood with no oxygen in it) is emptied into the right atrium via the vena cava. The inferior vena cava returns blood from the lower portion of the body as the superior vena cava returns blood from the higher portion. This blood is then pumped through the tricuspid valve into the right ventricle. Blood is then passed into the lungs via the pulmonary artery where it is oxygenated. Oxygenated blood then returns from the lungs into the left atrium where it can be

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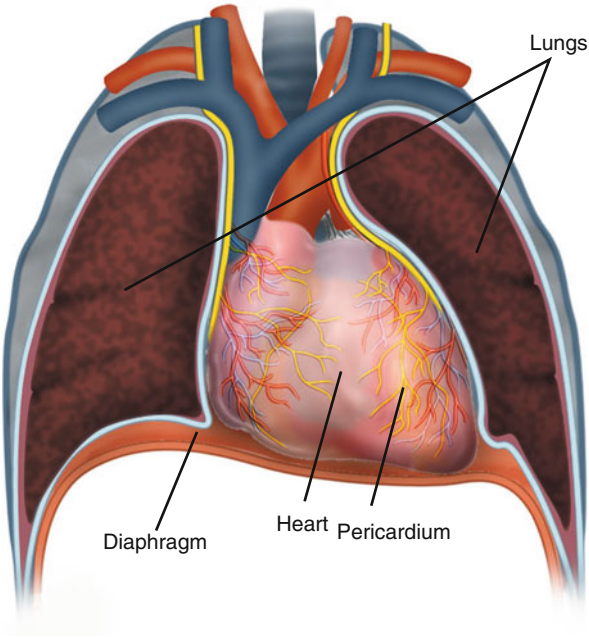


Fig. 1.1 The location of the heart in the thoracic cavity

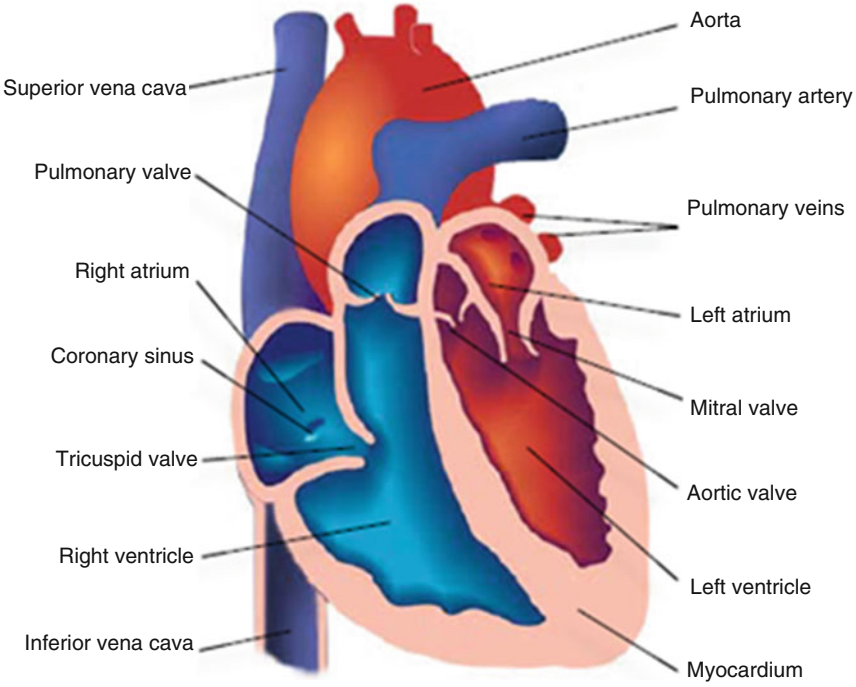


Fig. 1.2 Diagrammatic view of the chambers and vessels of the heart

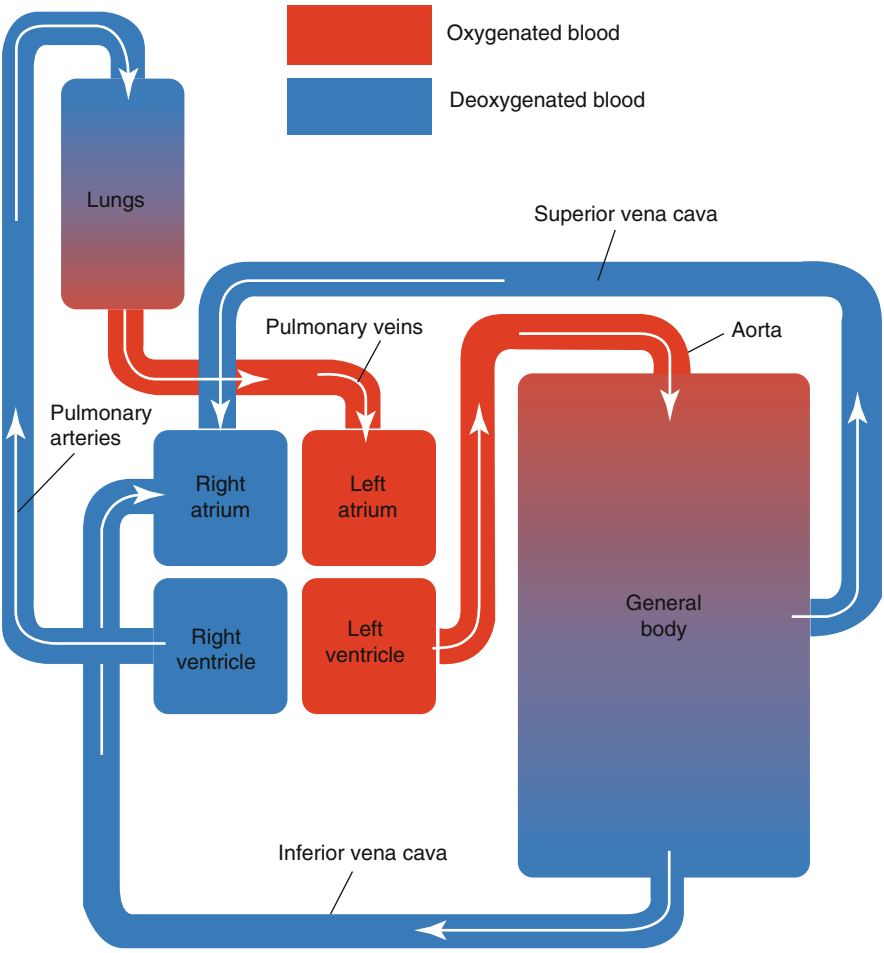


Fig. 1.3 Schematic diagram showing the mechanical function of the heart

pumped to the rest of the body by the powerful left ventricle, via the aorta (Fig. 1.3). The cells responsible for the contraction of the heart muscle are called myocytes. Apart from the hearts mechanical function as a pump it also has an electrical system governing the rate at which the heart beats, controlling in turn how slow or fast the blood and oxygen gets pumped to all the organs and tissues in the body.

In addition to the myocyte cells, there are also specialised conduction cells in the heart. These cells possess a quality know as automaticity. This is the ability to spontaneously depolarise via an electromechanical gradient. Depolarisation is a process where by a resting cell becomes gradually more positively charged (Fig. 1.4). This is accomplished by a sudden influx of positively charged sodium and calcium ions into the cell Alternatively, Repolarisation is the returning of the cell to its resting state following a brief refractory (recovery) period.

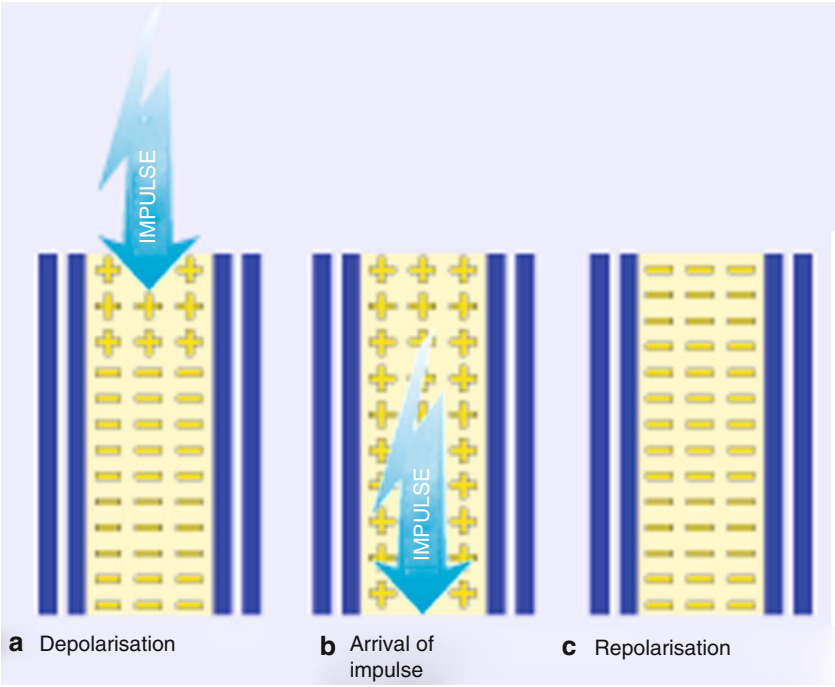


Fig. 1.4 Depolarisation and repolarisation

These specialised conduction cells are distributed throughout the heart forming specialised conduction pathways (Fig. 1.5). Depolarisation occurs in the Sinoatrial node (SAN). This is a collection of self-excitory (pacemaker) cells that normally fire at a rate of between 60 and 100 Beats Per Minute (BPM). The "wave" of Depolarisation moves from the SAN through an intra-atrial tract called Bachmanns bundle into the left atrium and to the Atrioventricular (AV) node. From here the impulse travels down the bundle of His into the right and Left bundle branches and finally into the Purkinje fibres activating the ventricles.

We will now examine the components of the conduction system in isolation to better understand their function. It is also worth noting that every cell in the conduction system can act as a pacemaker when called upon to do so. This provides a backup system should the SAN fail. The lower down the conduction system is activated, the slower the heart rate.

Sinoatrial Node (SAN)

[pacemaker rate approx: 60–100 BPM] The SAN is located in the right atrium, near the join of superior vena cava with the atrial mass (Fig. 1.6).

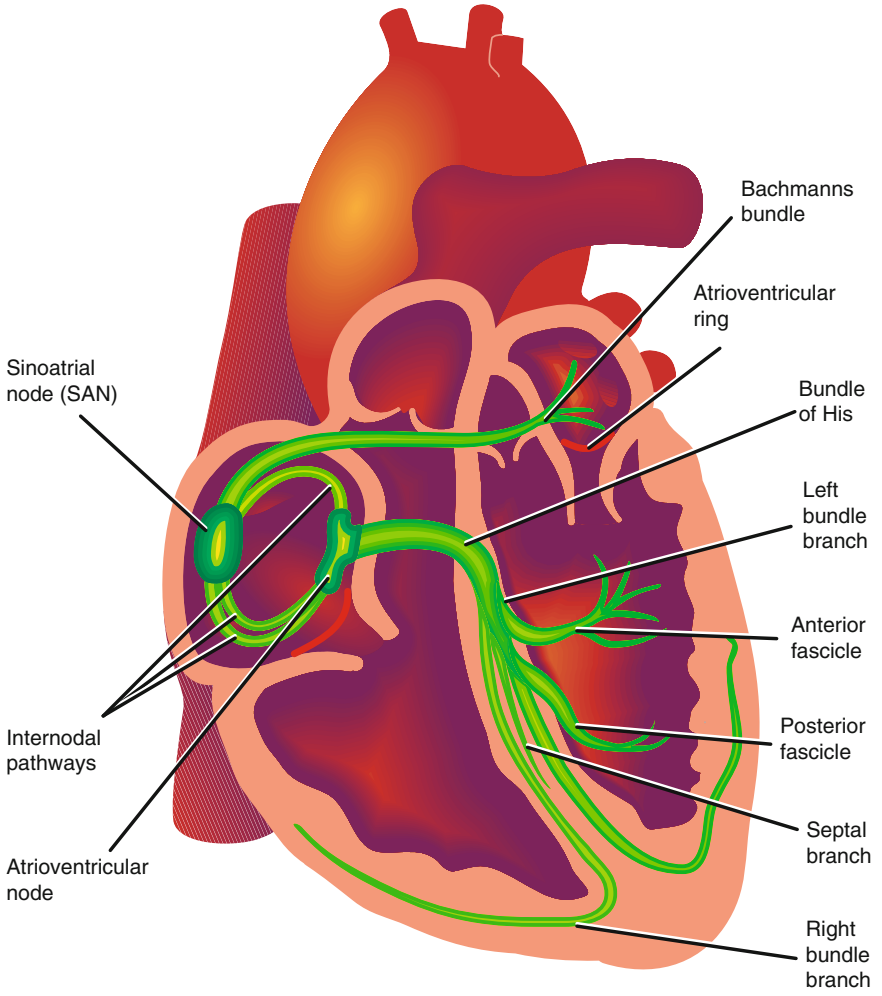


Fig. 1.5 Cardiac conduction system

The SAN acts as the hearts primary pacemaker. The 'firing' rate of the SAN is where the 'normal' heart rate figure is derived from. Anything above 100 BPM is termed a 'tachycardia', conversely anything below 60 BPM is referred to as a 'bradycardia'.

Note
Blood supply to the SAN originates from:

- Right coronary artery in 59 % of people
- Left coronary artery in 38 % of people
- Right and left coronary arteries in 3 % of people

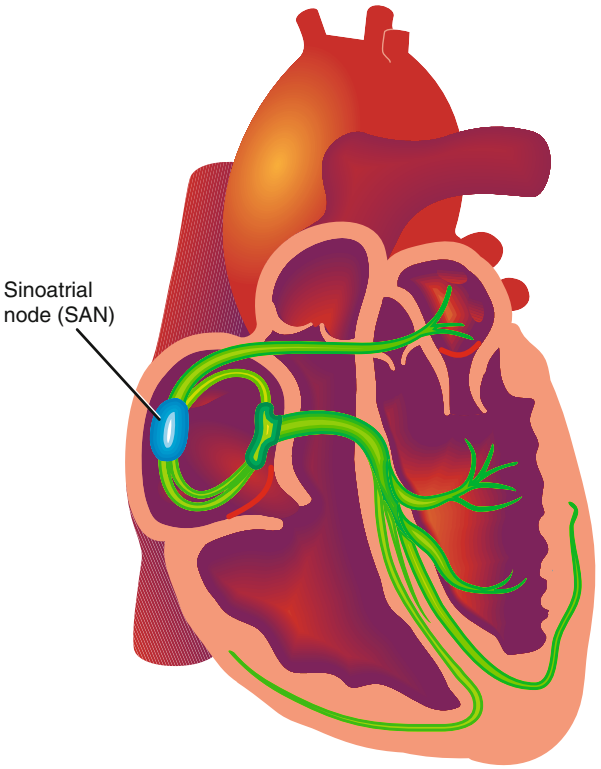


Fig. 1.6 Sinoatrial node (SAN)

Interatrial/Internodal Tracts

The Bachmanns bundle and iternodal tracts allow the rapid transmission of electrical impulses from the SAN to the left atrium and AV node.

Note
Some authors argue about the existence of the internodal pathways and/or the Bachmanns bundle and instead believe that impulses generated in the SAN are transmitted through normal cardiac tissue in waves. The analogy of a stone dropped into water creating electrical ripples that eventually reach the AV node is often sighted.

Atrioventricular Node (AV)

[pacemaker rate approx: 40–60 BPM] The AV node deliberately delays the impulses from the atria allowing the ventricles time to finish filling and to optimise cardiac

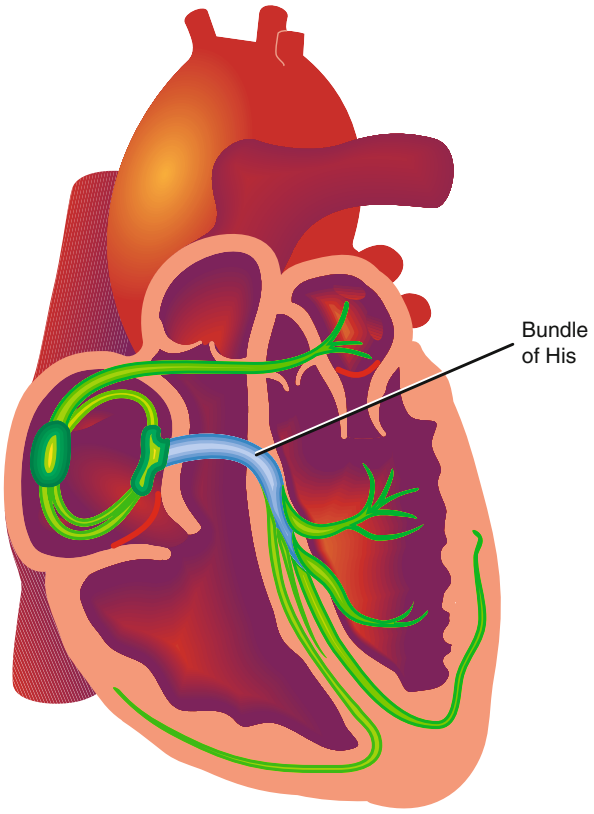


Fig. 1.7 The bundle of His

output. The atria and ventricles are isoelectrically insulated by the atrioventricular ring. The AV node allows electrical impulses generated in the atria to pass into the ventricular region.

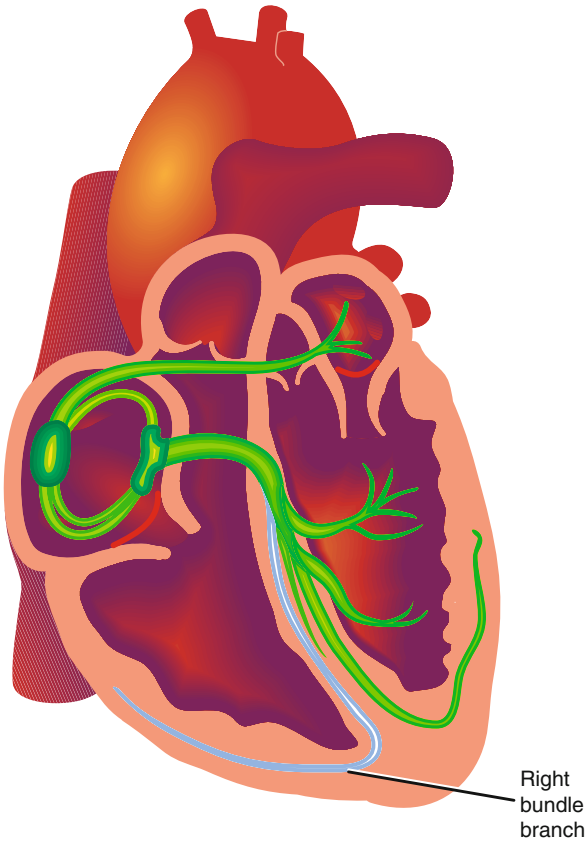
Bundle of His

[pacemaker rate approx: 40–45 BPM] Located primarily in the intraventricular septum (Fig. 1.7). The bundle of His allows the impulse to travel from the atria to the ventricles. The bundle of His bifurcates into the left and right bundle branches.

Right Bundle Branch

[pacemaker rate approx 40–45 BPM] Allows the electrical impulse to travel from the common bundle branch into the right ventricle where the impulse is transmitted through the Purkinje fibres attached to the Right bundle branch (Fig. 1.8).

Fig. 1.8 Right bundle branch



Left Bundle Branch

[pacemaker rate approx.: 40–45 BPM] The Left bundle branch is more complex and has two fascicles protruding from it. This is because the left ventricle is much larger than the right, so by contrast there are more elements to the conduction system of the Left bundle branch. The two fascicles are referred to as the anterior and posterior fascicles (Fig. 1.9).

What Is an ECG and How Are They Recorded?

The ECG, short for electrocardiogram is a graphical representation of the electrical activity generated by the heart. This can be of help in diagnosing or supporting the presence of cardiac rhythm disturbances, structural heart disease, acute cardiac emergencies and a variety of other medical conditions. The ECG is a cheap and easily repeatable test. The wide availability of the ECG means that it is available

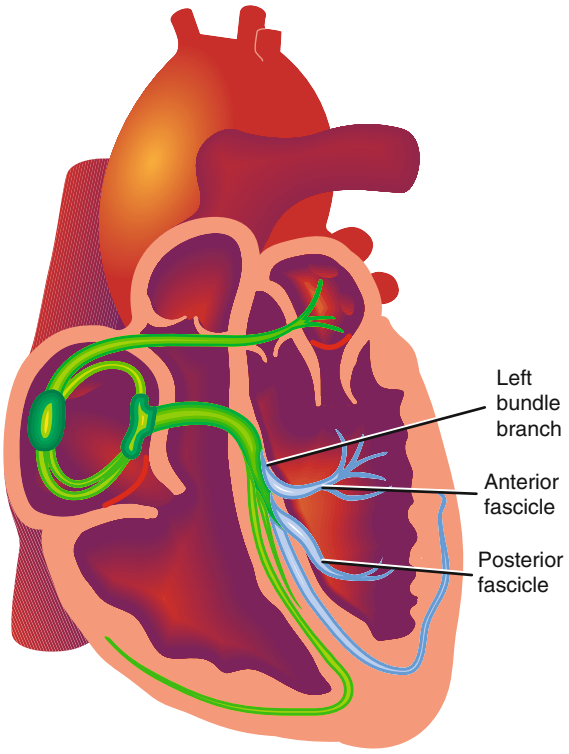


Fig. 1.9 Left bundle branch

outside of cardiology areas, and is now found on many general wards, GP surgeries and other clinical areas. Electrical activity from the heart is picked up by cables called leads that are attached to a patient. The electrical activity of the heart muscle is then represented by the ECG machine on pre-printed graph paper.

Patient Positioning

Prior to recording a 12-lead ECG, the procedure should be explained to the patient and the patient's consent obtained (Fig. 1.10). The patient should then be laid back at an angle of around 30–45°. This helps to open up the intercostal spaces, allowing easier placement of the electrodes. If the patient is in any other position, including sitting upright, it should be documented on the ECG i.e. 'recorded with patient sitting upright'.

Sometimes it is necessary to prepare the skin prior to attaching electrodes. If there is visible dirt (blood, soil, water, oil, etc.) this should be removed prior to obtaining a recording. If the patient is perspiring, the adhesion of the electrodes may be affected. In this case an alcohol wipe (or soap and water) can be used to clean the area. It may also be necessary to shave off any excessive chest hair, to ensure better adhesion of the electrodes.

Fig. 1.10 Nurse explaining procedure to patient and seeking consent

Note
Before recording the ECG, check the equipment, including:

- Paper is loaded
- Power supply
- Presence of all cables/clips

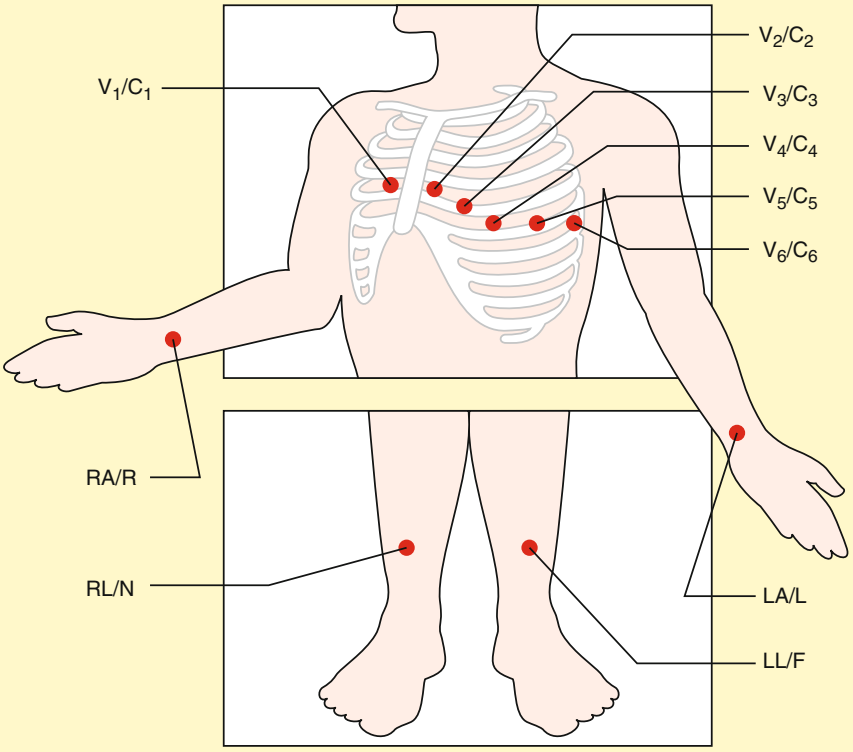
Electrode Placement (Fig. 1.11)

Step 1: Prior to attaching the electrodes and ensuring good patient position, all clothing on the top half of the body should be removed. The trousers can be rolled up to allow access to the legs. If the patient is wearing any tights they should also be removed prior to attaching electrodes. The skin is then prepared as necessary (as discussed earlier).

Step 2: First start by attaching the limb electrodes to the arms and legs. When attaching the electrodes to the legs it helps to place them with the tab facing towards the torso so the cables don't pull (Figs. 1.12 and 1.13).

Step 3: The electrodes can then be attached to the torso, starting with V_1, which is placed in the fourth intercostal space, just to the right of the sternum.Electrodes should be placed with the middle of the electrode in the middle of the intercostal space.

One of the problems with the position of V_1 is that practitioners sometimes miscount the number of intercostal spaces due to the gap between the clavicle and the start of the rib cage. To avoid this, the patient's sternum can be felt from the top



Chest leads

V_1/C_1 – 4th Intercostal space, right side of the sternal border
V_2/C_2 – 4th Intercostal space, left side of the sternal border
V_3/C_3 – Diagonally between V_2 and V_4
V_4/C_4 – 5th Intercostal space, midclavicular line
V_5/C_5 – Anterior axillary line in horizontal line with V_4
V_6/C_6 – Midaxillary line in horizontal line with V_5

Limb leads

LA/L – Left wrist
RA/R – Right wrist
LL/F – Left leg
RL/N – Anywhere, but usually on the right leg for symmetry.

Fig. 1.11 Electrode positions

down until contact is made with the 'angle of Louis' (Fig. 1.14). From here, the fourth intercostal space can be located by feeling diagonally down from the bottom of this point by two intercostal spaces.

The next electrode to be placed is V_2, this is positioned in the same horizontal line as V_1 but on the opposite side of the sternum. Next to be positioned is V_4, this is done out of sequence as the position of V_3 is relative to that of V_4. The V_4

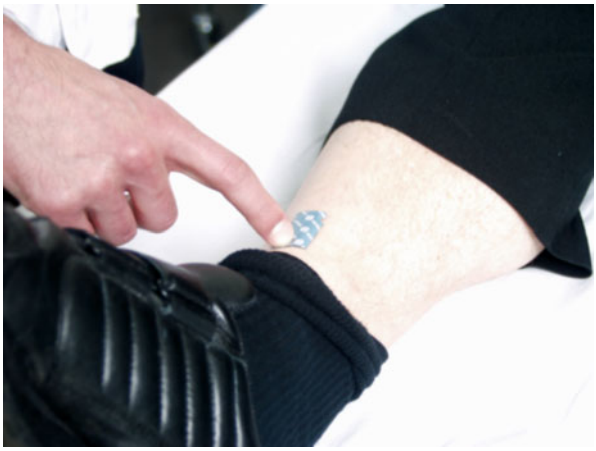


Fig. 1.12 Electrode placed with tab facing towards torso

Fig. 1.13 Electrode placed on wrist

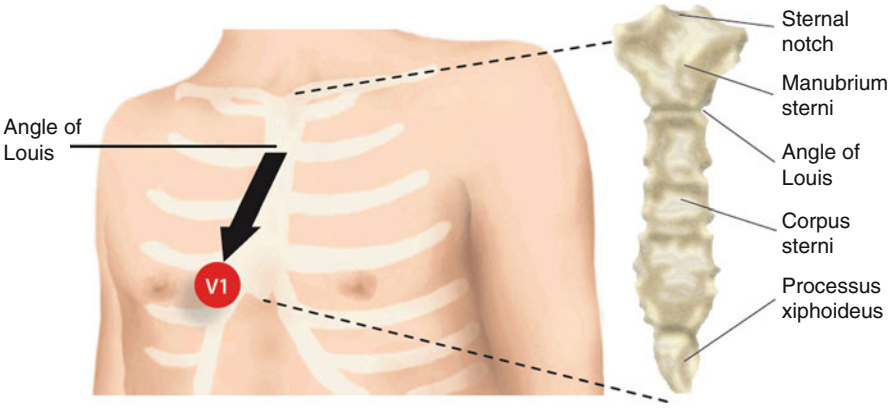


Fig. 1.14 The angle of Louis

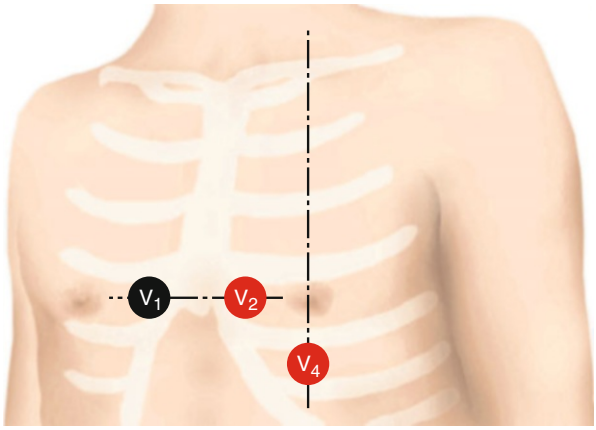


Fig. 1.15 Electrode position for V_4 (5th intercostal space/ mid clavicular line)

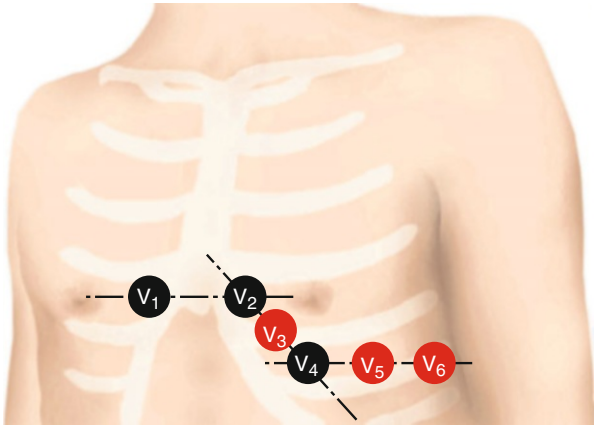


Fig. 1.16 Electrode positions for V_3, V_5 and V_6

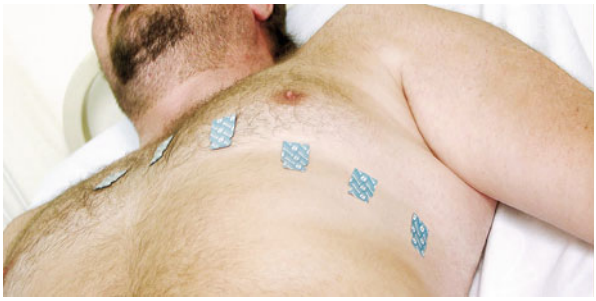


Fig. 1.17 Electrode positions V_1–V_6

electrode is placed in the 5th intercostal space in line with the middle of the clavicle (Fig. 1.15) this is normally located approximately just under the left nipple.

Now V_3 can be positioned, diagonally in between V_2 and V_4. The V_5 electrode is then placed in line with the anterior axilla line and V_6 in line with the middle of the armpit (Figs. 1.16 and 1.17).

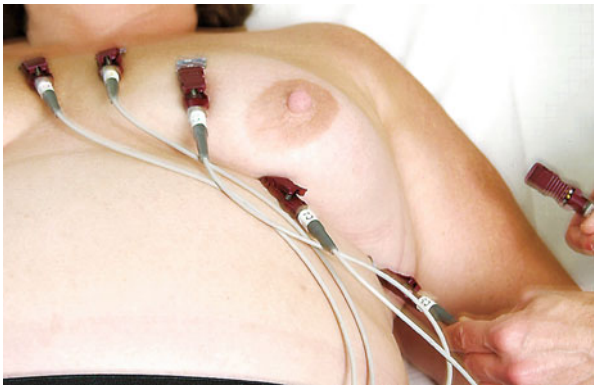


Fig. 1.18 Attaching of cables to electrode sites

Women

There is sometimes confusion about the electrode positions in the case of women due to the breast tissue. If the breast tissue is relatively thin or there is a considerable breast droop, then the electrodes may be placed on top of the breast. In most cases and certainly with younger women the electrodes are placed in exactly the same positions, with the exception of V_4 which is placed under the breast, taking due care to maintain dignity and seek consent prior to placement. The cables can then be attached to the electrode pads (Fig. 1.18).

Attaching the Cables

When attaching the leads to the electrodes (Figs 1.18, 1.19 and 1.20):

- Be wary of pinching the patient's skin
- Pressing down on the top of the electrode helps to lift the tab, making it easier to attach the clip
- Make sure the cables are not twisted or dangling over the edge of the bed. The patient should be encouraged to relax their arms, shoulders, neck and head prior to recording as this can cause interference on the ECG tracing.

General Tip
Turn the box from where the cables protrude so the top (usually labelled) faces upwards. From this position the cables fan out in the correct order for attachment. i.e. the two cables on the left of the machine are R/RA and N/RL and the last two are L/LA and F/LL, leaving the six in the middle V_1–V_6 in order. This can help you to record an ECG more rapidly.

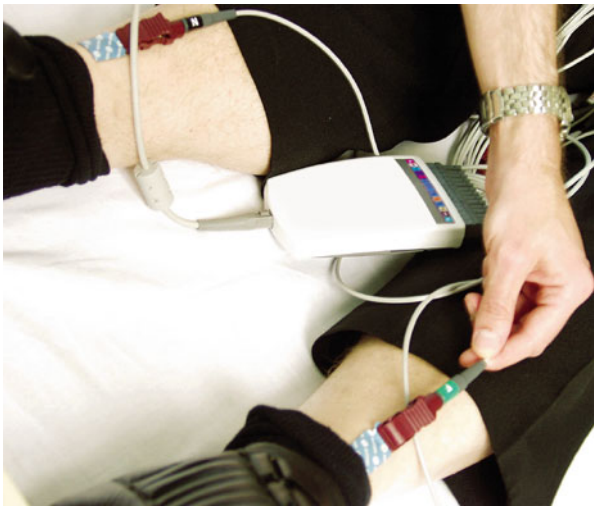


Fig. 1.19 The cables can be attached to the electrode tabs

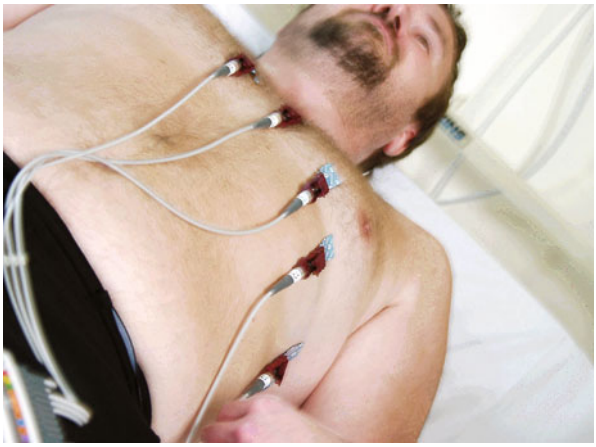


Fig. 1.20 Attaching chest leads

The Machine

There are many different types of 12-lead ECG recording device. Most share similar features. Specific details can be found in the operator's manual that comes with the ECG machine.

Check the machine to ensure all leads are being recorded. On modern machines the tracing can be seen on the monitor, older machines usually have a light which activates if a lead is not being recorded. In such an event, recheck the electrodes are still attached to the patient and the clips are attached to the electrodes.

It is also important to ensure that the machine is running at the standard calibration and speed and to adjust them as necessary if they are not.

Standard Recording Settings
- Speed 25 mm/s
- Amplitude 10 mv/mm

The patient should then be asked to lie still and not move or speak until instructed. This will aid in recording a good quality ECG, and reduce potential interference on the recording.

What to Write on the ECG

If you have recorded an ECG it is helpful for diagnosis and/or future reference to document certain pieces of information on the ECG including:

- Patients name, sex, DOB and hospital ID number
- The date and time recorded
- Any relevant observations or symptoms i.e. patient's blood pressure, heart rate or symptoms i.e. 'chest pain >30 min', 'palpitations'
- Any alterations to recording or position i.e. 'patient sat upright'.

Summary of Key Points

- Accuracy in electrode positioning is vital for a good quality diagnostic ECG.
- Patients should be relaxed and informed prior to the recording of an ECG, and dignity maintained throughout.
- Relevant information should be documented on the ECG about the patient, including identifying details, symptoms and observations along with the date and time of the recording.
- As a backup system, any part of the conduction system can take over the role as primary pacemaker. The lower down the conduction system the slower the rate.
- It is important to ensure that the cables are attached correctly to the electrodes with no twisting or dangling.

Quiz

Q1. The V_4 electrode should be positioned…

(A) 4th intercostal space mid-clavicular line
(B) 5th intercostal space mid-clavicular line
(C) 5th intercostal space mid-axilla

Q2. The neutral lead 'N' must be placed on the right leg

(A) True
(B) False

Q3. The specialised cells of the conduction system are said to possess…

(A) Extra electricity
(B) Action potentials
(C) Automaticity

Q4. How many fascicles does the left bundle branch have?

(A) 2
(B) 3
(C) 1

Q5. What should be documented on the ECG after recording?

(A) Patients name DOB and unit number
(B) Relevant observations and symptoms
(C) Date and time of recording
(D) All of the above

Q6. The ECG is best recorded with the patient…

(A) Laid down
(B) Sat bolt upright

Answers: Q1 = B, Q2 = B, Q3 = C, Q4 = A, Q5 = D, Q6 = A

Chapter 2
ECG Basics

Abstract The 12-lead ECG is a graphical representation of the electrical activity produced by myocardial excitation. It works by detecting this electrical activity by means of a set of passive terminals called leads, which are located in specific positions on top of the patient's skin. This signal is translated into the familiar ECG graph. This electrical information is displayed in 12 different views based on the position of the electrodes on the body.

Keywords Waveforms • Complexes • Leads • Intervals • Segments • Deflection

How Does the 12-Lead ECG Work?

The 12-lead ECG is a graphical representation of the electrical activity produced by myocardial excitation. It works by detecting this electrical activity by means of a set of passive terminals called leads, which are located in specific positions on top of the patient's skin. This signal is translated into the familiar ECG graph. This electrical information is displayed in 12 different views based on the position of the electrodes on the body.

ECG Paper

The paper used in standard ECG machines comes with the grid pre-printed. The standard grid conforms to specific dimensions when machines are in standard setting. The gridded paper is split into large squares and small squares (Fig. 2.1).

The online version of this chapter (doi: 10.1007/978-1-4471-4962-0_2) contains supplementary material, which is available to authorized users.

Please note some of the figures within this chapter have been reproduced in full size online at Extra Materials (extras.springer.com) for ease of viewing

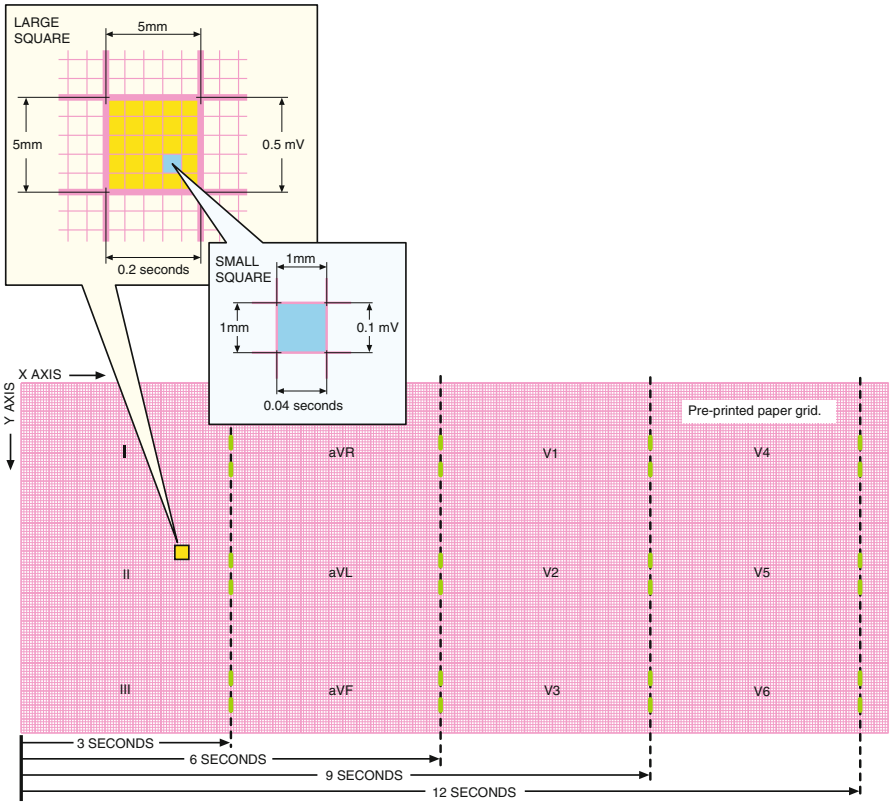


Fig. 2.1 ECG paper

Large Squares
Contain five small squares in height and width

- Are 5 mm by 5 mm in height and width
- Along the X axis a large square represents 0.20 s (seconds of time)
- Along the Y axis a large square represents 0.5 mV (millivolts)
- Five large squares represent 1 s of time

Small Squares

- Are 1 mm by 1 mm in height and width
- Represent 0.04 s along the X axis
- Represent 0.1 mV along the Y axis

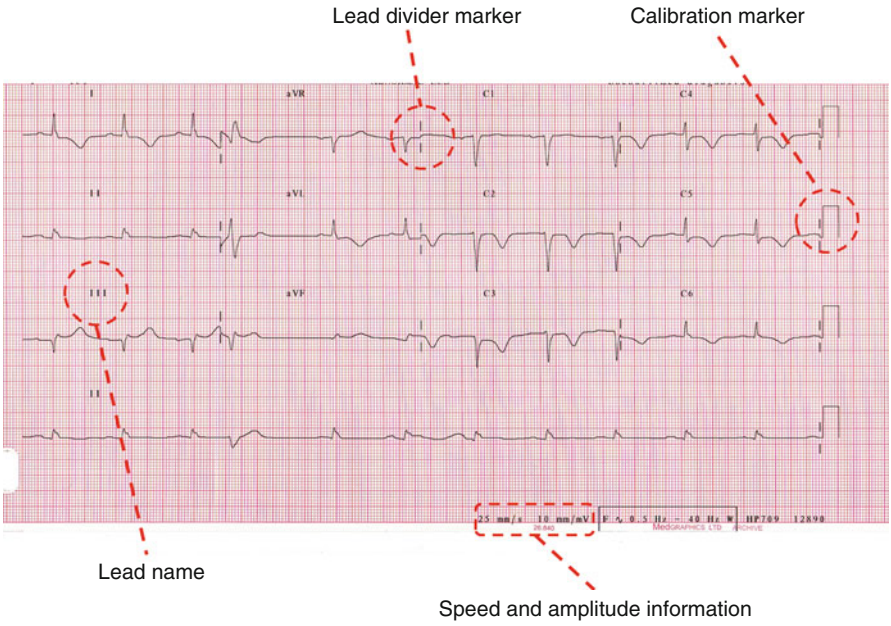


Fig. 2.2 Standard details found on the 12-lead ECG

Details Found on a Standard 12-Lead ECG

Each of the 12-leads are displayed on the ECG paper with the appropriate lead name (I, II, III, aVR, aVL, aVF, V_1, V_2, V_3, V_4, V_5 and V_6). Each lead is separated by a lead separator/divider marker. A calibration marker is also found at the beginning or end of each line of the ECG. The speed and amplitude may also be found along the bottom the ECG (Fig. 2.2). On many ECGs one of the leads (usually lead II or V_1) is repeated along the bottom of the ECG for the full 12 s. This is used as a rhythm strip.

Many modern ECG machines may also display additional data such as various intervals and timings; some even attempt to provide a possible diagnosis (Fig. 2.3).

12-Lead ECG Leads

The ECG leads shown on the 12-lead ECG are made up of a combination of the ten electrodes placed on the body. These leads can be referred to either as unipolar or bipolar leads. It is important to understand the difference between the 12 leads (views) of the heart shown on the ECG, and the ten cables attached to the electrodes, also referred to as leads.

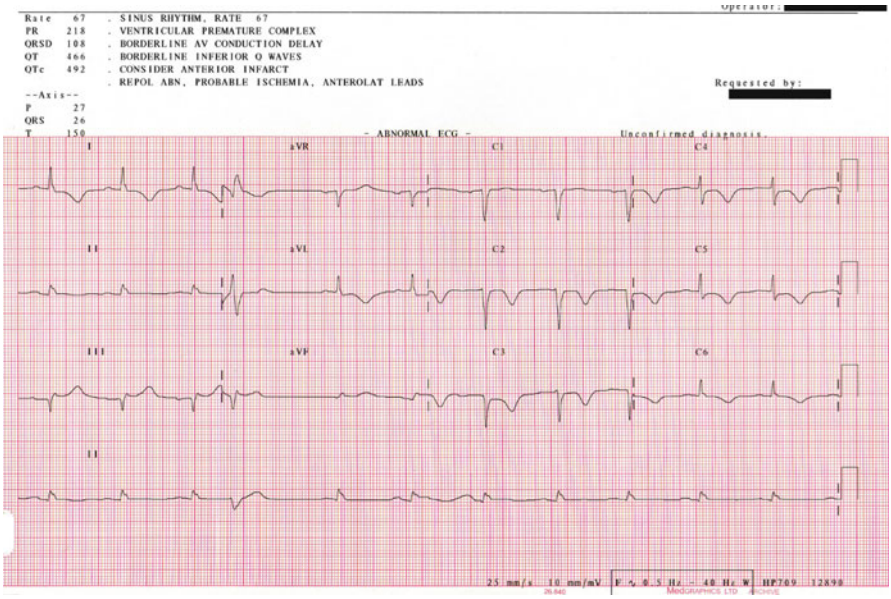
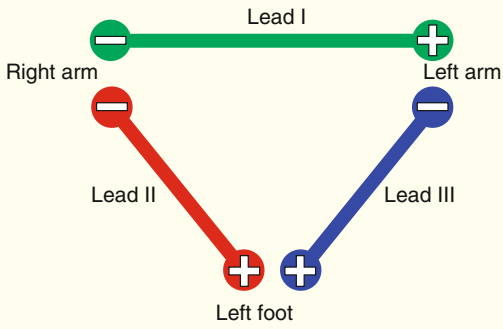


Fig. 2.3 ECG showing various additional information, including the machines attempt to derive a diagnosis

Fig. 2.4 Einthoven's triangle



Bipolar Leads

The three limb leads; I, II and III combine to form the bipolar leads. By bipolar we mean that the lead has both a positive and negative pole. Willem Einthoven established this standardised lead system, which was named after him, called 'Einthoven's Triangle' (Fig. 2.4). Different electrode positions were also tested but produced little or no measurable results in comparison.

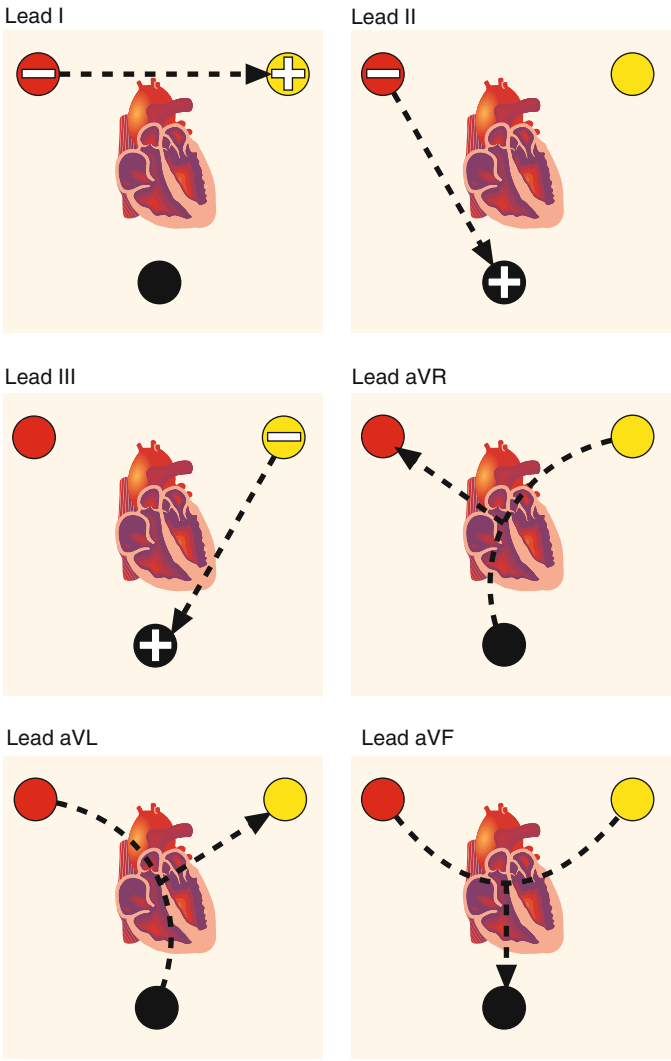


Fig. 2.5 Leads I, II, III, aVR, aVF and aVL

Unipolar Leads

The unipolar leads are leads with only one pole. After the creation of the bipolar leads the unipolar leads were created to detect the heart's electrical activity from anywhere on the body, called; VF, VR and VL. They work by taking an average from between any two of the bipolar leads (Fig. 2.5).

The voltage was later boosted creating the augmented voltage leads known as aVR, aVL and aVF. Subsequently the six unipolar chest leads were also created to view the heart from the frontal plane in a horizontal view (Fig. 2.6).

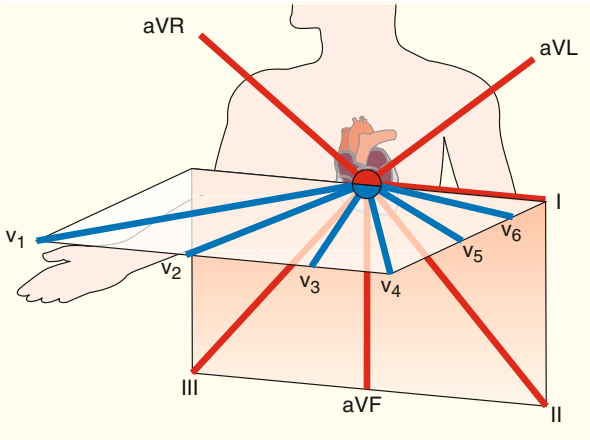


Fig. 2.6 Chest leads V_1–V_6 (unipolar)

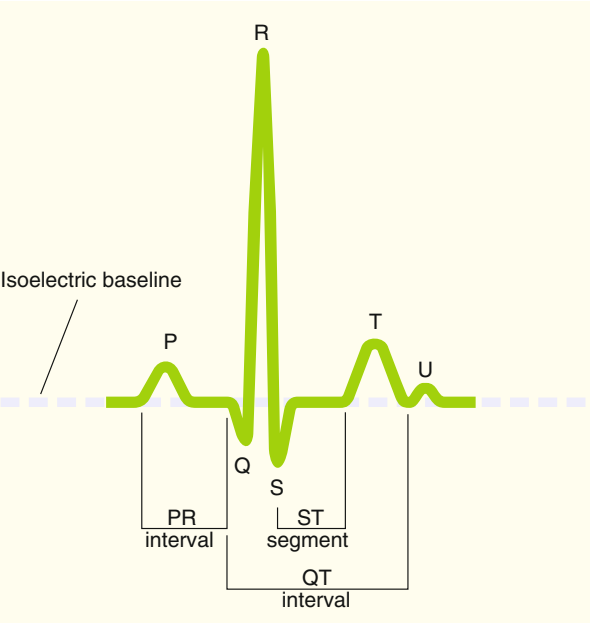


Fig. 2.7 ECG PQRST waveform and intervals

The PQRST Waveform

The standard ECG is made up of PQRST and sometimes U waves. Each PQRST waveform represents a single heart beat (Fig. 2.7).

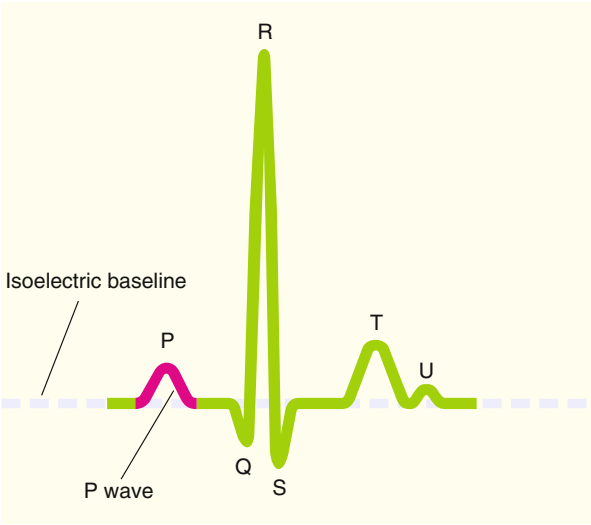


Fig. 2.8 The P wave

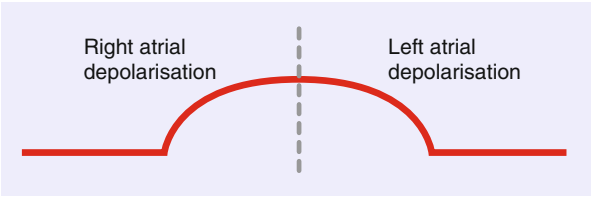


Fig. 2.9 P wave showing left and right atrial depolarisation

The P Wave

The P wave represents the electrical activation of both atria (atrial depolarisation). The P wave is formed when the electrical impulse (usually) formed in the SAN reaches both atria initiating atrial depolarisation (Figs. 2.8 and 2.9).

The first half of the P wave corresponds to the depolarisation of the right atria, the second half corresponds to the left atrial depolarisation (Fig. 2.10).

The PR Interval

The PR interval, measured from the start of the P wave to the start of the QRS complex. This is the time taken from the electrical signal activating the atria then travelling down the His bundle via the AV node to the left and right bundle branches, and finally to the Purkinje fibres extending from the bundle branches (Figs. 2.11 and 2.12). This delay allows for blood to pass from the atria into the ventricles and is a silent event (not seen) on the 12-lead ECG.

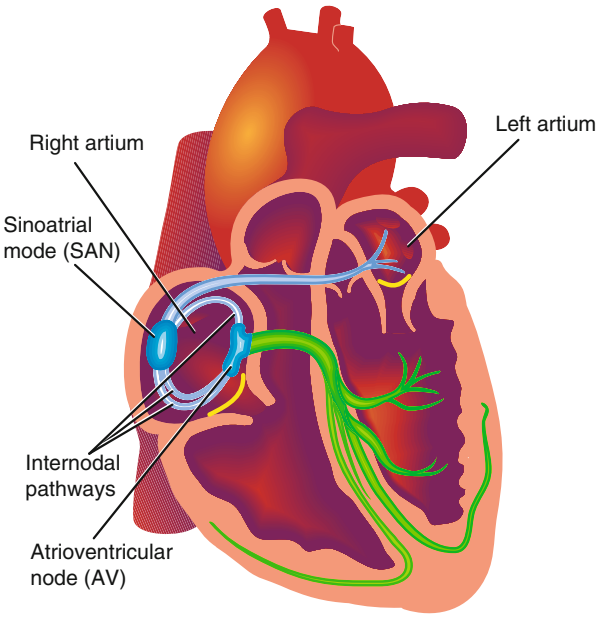


Fig. 2.10 P wave formed representing atrial depolarisation

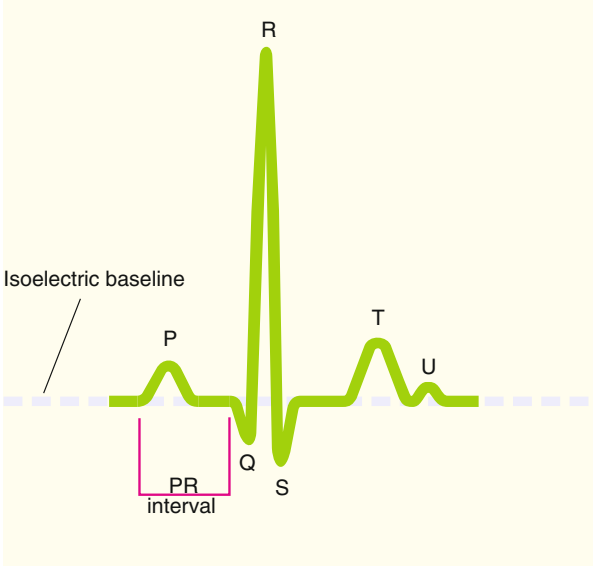


Fig. 2.11 The PR interval

The QRS Complex

The larger QRS complex represents the activation of the ventricles (ventricular depolarisation) (Fig. 2.13). After the electrical impulse arrives in the ventricles, the

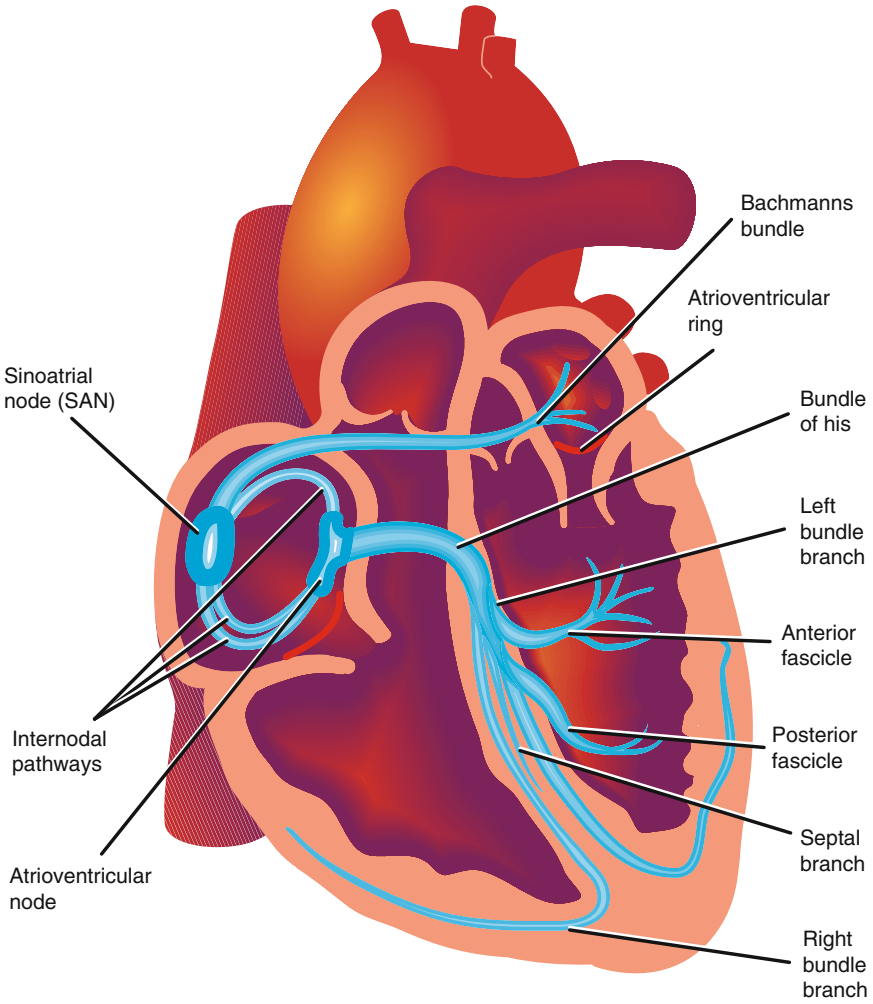


Fig. 2.12 PR interval represents time taken for impulse to travel from SAN to Purkinje fibres at the end of the bundle branches

ventricles contract, pumping blood around the body. As the ventricles are larger than the atria the QRS has a greater amplitude (is taller) than the P wave. The QRS complex is made up of three separate waves, the Q, R and S waves.

There can also be quite a variety in appearance of the QRS complex, as seen in Fig. 2.14. When a wave has an amplitude greater than 5 mm it is represented by an uppercase letter. When the amplitude is less than 5 mm a lower case letter is used.

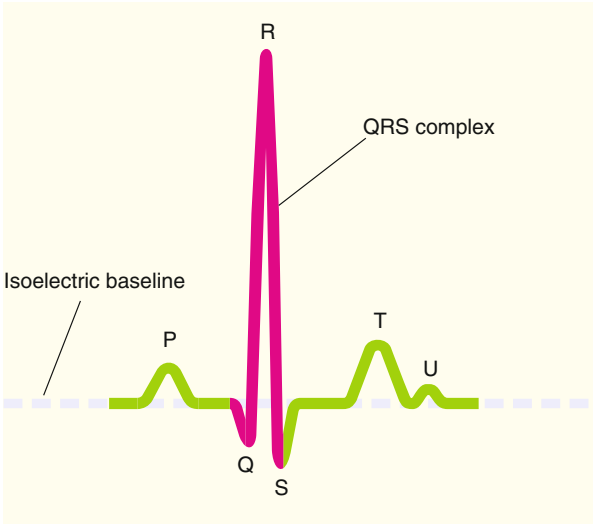


Fig. 2.13 The QRS complex

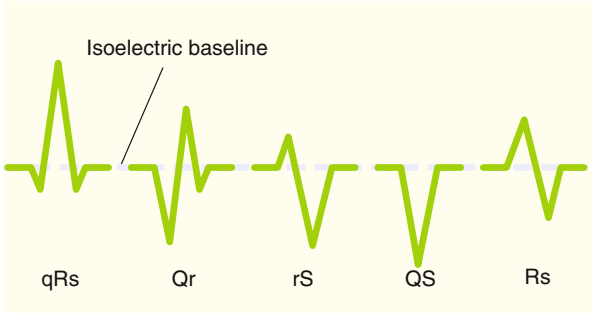


Fig. 2.14 Some variations in QRS appearance

QRS Complex

- The first downward pointing wave in the QRS complex is called the Q wave
- The first upward pointing wave is called the R wave
- The downward pointing wave following the R wave is called the S wave

Note

- Not all of the waves of the QRS complex may be visible
- The QRS complex also looks different when viewed by different leads.

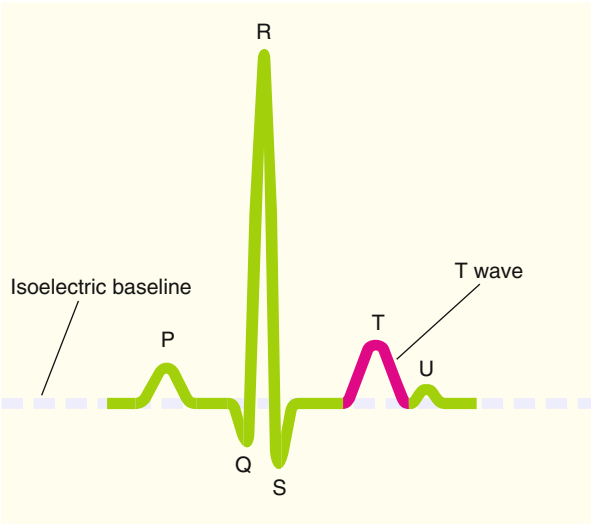


Fig. 2.15 The T wave

The T Wave

The T wave represents the repolarisation of the ventricles (Fig. 2.15). During this time the ventricles return to their resting electrical state.

The U Wave

U waves are not always seen on the ECG (Fig. 2.16). Opinion is divided over what exactly the U wave represents. Many consider the U wave to represent repolarisation of the ventricular septum or Purkinje fibres. These waves can be difficult to see and are usually smaller than P waves.

Note
The atrial repolarisation is not detectable on the ECG.

The ST Segment

The ST segment is measured from the end of the S wave to the start of the T wave (Fig. 2.17). The J point represents the junction where the S wave meets the isoelectric baseline. The ST segment represents the gap between ventricular depolarisation and repolarisation. During this period no additional electrical signals can pass through the myocardium.

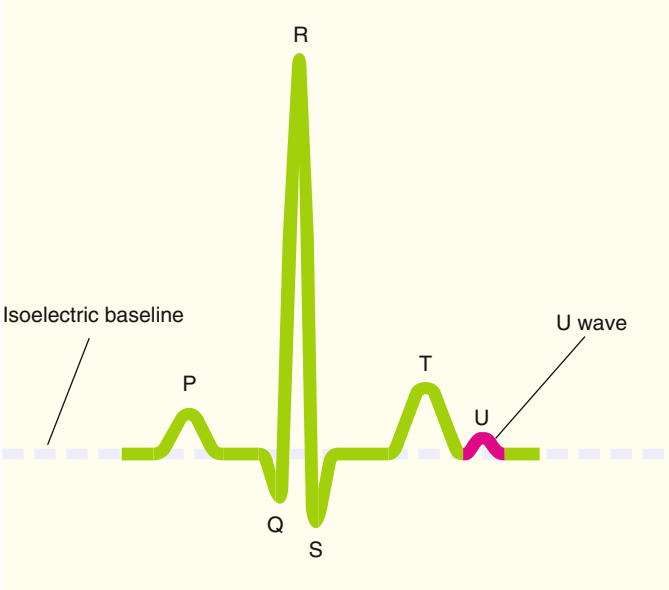


Fig. 2.16 The U wave

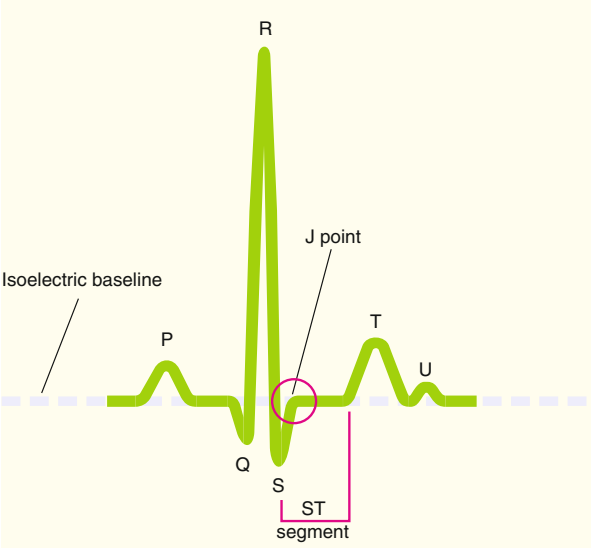


Fig. 2.17 The ST-segment

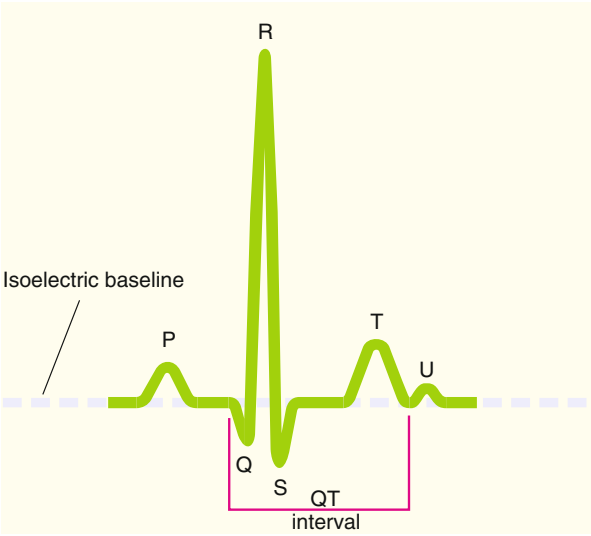


Fig. 2.18 The QT interval

The ST segment should be isoelectric and not be raised or depressed above or below the isoelectric baseline.

The QT Interval

The QT interval is measured from the start of the QRS complex to the end of the T wave, and represents the total time taken from depolarisation of the ventricles to their repolarisation (return to resting state) (Fig. 2.18).

Deflection

It is important to understand that when the heart's electrical impulse is viewed through different leads, it looks different depending on the angle at which it is viewed. If the wave of depolarisation is moving toward a lead then it is positively deflected (points upwards above the baseline). If the wave is moving away from the lead then it is negatively deflected (points down from baseline). If the lead is at right angles then the wave is equiphasic (deflected as positively as it is deflected negatively). If the lead is somewhere between these positions then its deflection will be biphasic, predominately either up or down depending on its position relative to the direction of the wave of depolarisation. This is summarised in Fig. 2.19.

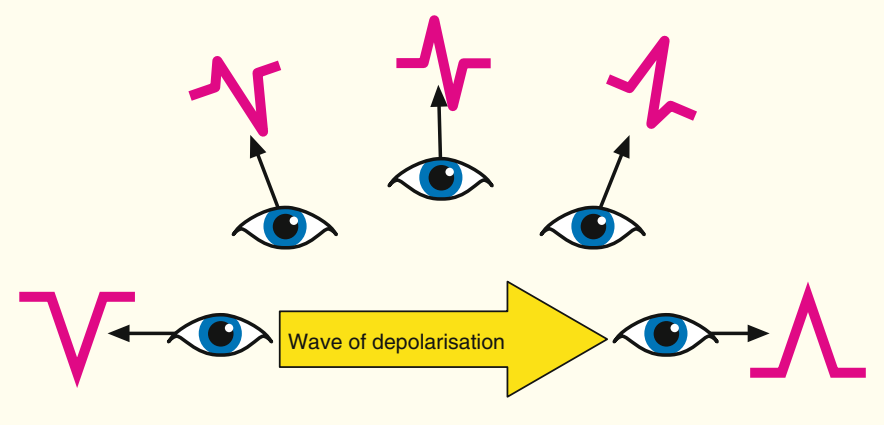


Fig. 2.19 Deflection

Summary of Key Points

- ECG waveforms are commonly made up of PQRS and T waves
- The P wave represents atrial depolarisation
- The QRS complex represents ventricular depolarisation
- The T wave represents ventricular repolarisation
- ECG waveforms are deflected in different directions depending on the position of the leads relative to the direction of the wave of depolarisation.
- The QRS complex can look different, and not all its waves are always visible.

Quiz

Q1. How many seconds does each 1 × 1 mm square represent?

(A) 0.04
(B) 0.08
(C) 0.20

Q2. Which of the following leads is commonly used as a rhythm strip at the bottom of a 12-lead ECG?

(A) aVR
(B) V_6
(C) II

Q3. Which of the following are bipolar leads?

(A) I, II and III
(B) V_1–V_6
(C) aVR, aVL and aVF

Q4. Where is the PR interval measured from?

(A) The end of the P wave to the start of the QRS complex
(B) The start of the P wave to the start of the QRS complex
(C) The start of the P wave to the end of the T wave

Q5. U waves are always present on a 12-lead ECG

(A) True
(B) False

Q6. If the wave of depolarisation is moving toward a lead then it is…

(A) Positively deflected
(B) Negatively deflected
(C) Equiphasically deflected

Answers: Q1 = A, Q2 = C, Q3 = A, Q4 = B, Q5 = B, Q6 = A

Chapter 3
Quality Issues Pertaining to ECG Recording

Abstract Recognition of quality issues pertaining to the recording of diagnostic quality ECGs is essential for all health care practitioners involved in the recording of ECGs. The skills required by the recorder of an ECG include the ability to recognise and reduce the various forms of interference that reduce the diagnostic quality of the ECG. It is important to understand that a lack of awareness concerning the quality issues related to ECG recording can lead to misdiagnosis and potential mismanagement of a patient. When reviewing the ECG prior to removing the cables and electrodes from the patient and interpreting the ECG; or referring the ECG to a member of staff qualified in ECG interpretation, the ECG recorder should briefly review the ECG to ensure it meets the criteria of an acceptable diagnostic recording. There are several key details that practitioners should check before proceeding to make a diagnosis.

Keywords Quality • Artefact • Calibration • Documentation • Interference

Background

Recognition of quality issues pertaining to the recording of diagnostic quality ECGs is essential for all health care practitioners involved in the recording of ECGs. The skills required by the recorder of an ECG include the ability to recognise and reduce the various forms of interference that reduce the diagnostic quality of the ECG. It is important to understand that a lack of awareness concerning the quality issues related to ECG recording can lead to misdiagnosis and potential mismanagement of a patient. When reviewing the ECG prior to removing the cables and electrodes

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DOI 10.1007/978-1-4471-4962-0_3, © Springer-Verlag London 2014

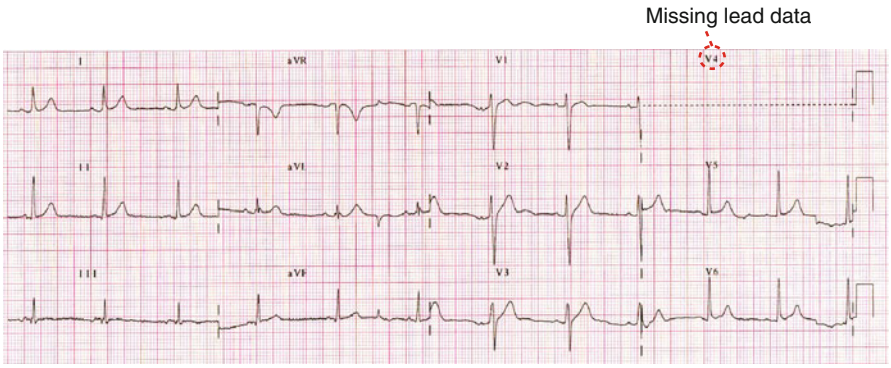


Fig. 3.1 Missing lead in V_4 position

from the patient and interpreting the ECG; or referring the ECG to a member of staff qualified in ECG interpretation, the ECG recorder should briefly review the ECG to ensure it meets the criteria of an acceptable diagnostic recording. There are several key details that practitioners should check before proceeding to make a diagnosis.

Leads

The first most obvious check that should be made is that all the leads are present in their correct positions on the ECG and that no areas are blank or missing data. Figure 3.1 shows an ECG where the cable/lead in the V_4 position had fallen off the patient due to poor attachment. Many ECG machines can detect missing signals; some will not let the user print the ECG until data for all lead positions is present.

The aVR Lead

The augmented vector lead aVR should be checked after a recording is made. PQRST waves in lead aVR are almost always negatively deflected (point downwards below the baseline). A positive deflection should alert the recorder to the possibility that the cables/leads attached to the limb electrodes could have been applied the wrong way round. This can mimic a condition called dextrocardia and is known as ‘technical dextrocardia’.

In both cases, in (technical or true) dextrocardia there are usually inverted P waves in lead I, and often a right axis deviation and prominent tall R waves in lead aVR.

Dextrocardia
A condition where the heart is located on the right hand side of the chest as oppose to the normal left sided position.

The main difference between technical dextrocardia and the true dextrocardia is seen in the R wave progression in the chest leads. In technical dextrocardia the R wave progression is normal (Fig. 3.3). Another good indicator is found by checking the deflection of lead aVR in the patient's previous ECGs. When confronted with a positively deflected aVR the practitioner should check that the limb leads are in the correct position and adjust accordingly if not. The two ECGs seen in Figs. 3.2 and 3.3 were taken from the same patient, the first with leads in normal positions (Fig. 3.2) and the second with the limb leads swapped over (Fig. 3.3).

R Wave Progression

The R waves in the chest leads ($V_1 - V_6$) are deflected differently, this is termed R wave progression (Figs. 3.4 and 3.5). In the early chest leads V_1 and V_2 the R waves are generally negatively deflected, moving through to predominantly positive in the late chest leads V_5 and V_6. Abnormalities in R wave progression are usually due to misplaced leads.

Figure 3.6 shows an ECG with leads V_2 and V_4 swapped over. The resulting abnormal R wave progression should alert the interpreter to the error made.

Calibration Markers

Are usually found at the beginning or end of the ECG recording (Fig. 3.7).

These markers visually display the speed and amplitude settings the ECG was recorded with. Markers should be 1 cm in height (2 large boxes). In certain

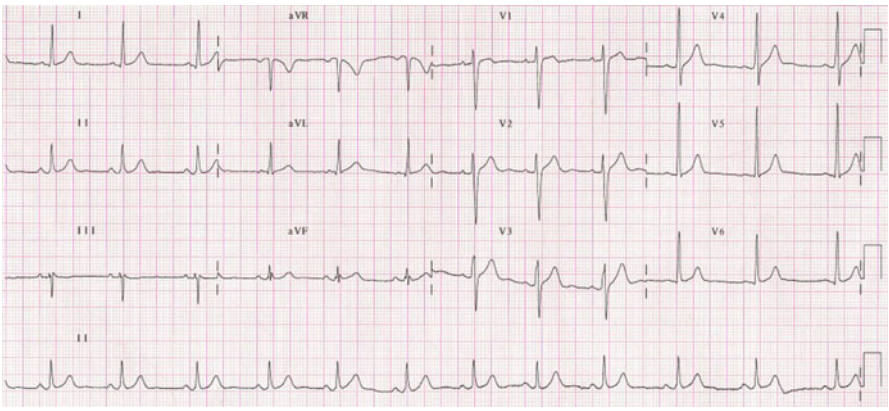


Fig. 3.2 Normal 12-lead ECG recorded with standard lead positioning

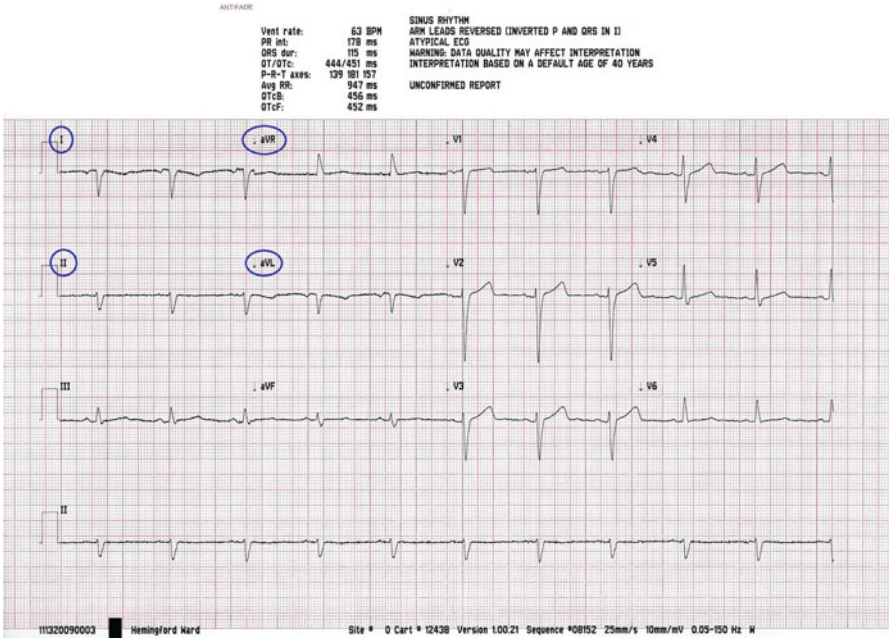


Fig. 3.3 Same ECG with limb leads swapped over. Note: positive aVR while lead I, II and aVL are now negatively deflected

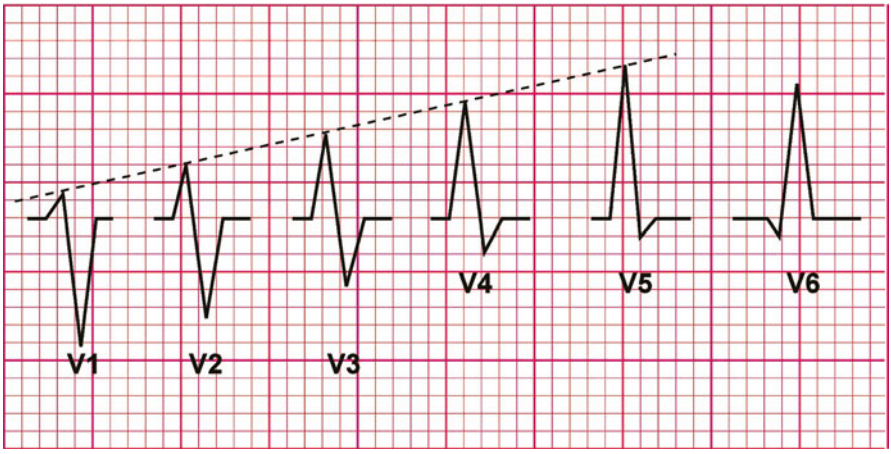


Fig. 3.4 Normal R wave progression from V_1 to V_6

situations, such as the presence of very large R waves (exceeding 35 mm in height). It may be necessary to reduce the amplitude to 0.5 cm to increase readability of the ECG. The calibration marker will change in appearance to indicate this (Fig. 3.8).

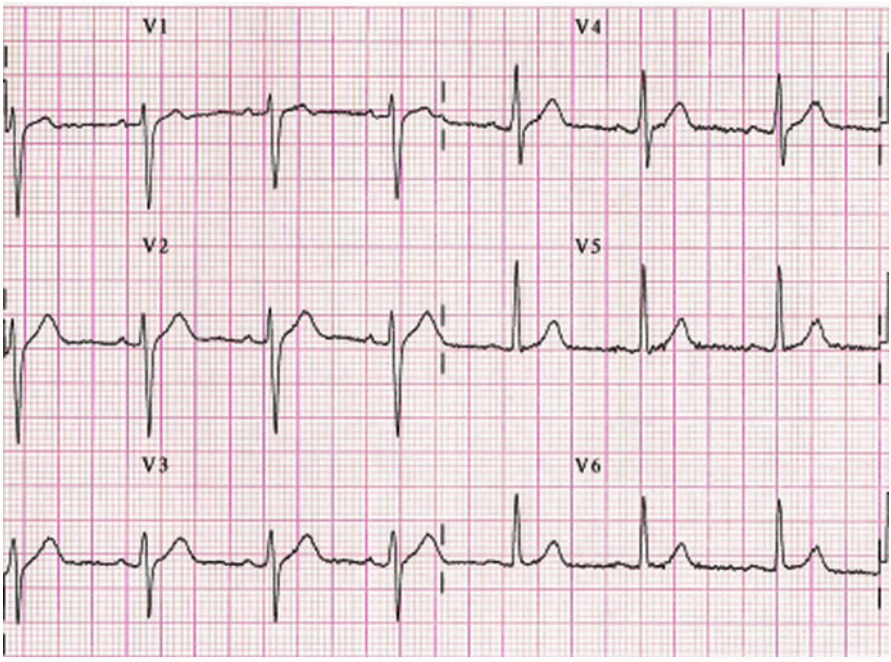


Fig. 3.5 normal R wave progression seen in the leads $V_1 - V_6$ on a 12-lead ECG

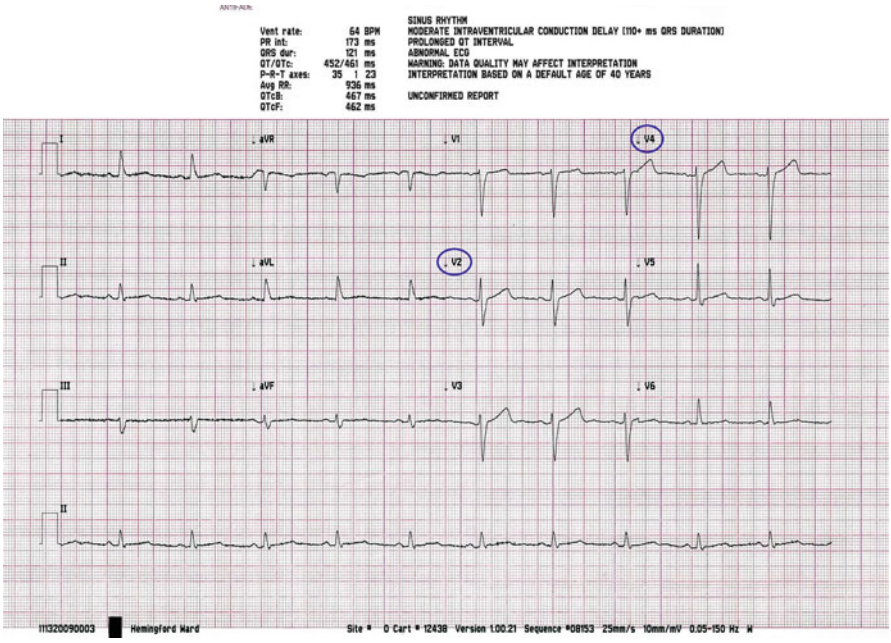


Fig. 3.6 Misplaced chest leads causing a change in R wave progression

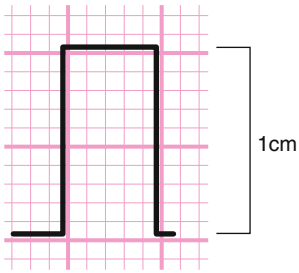


Fig. 3.7 Standard ECG calibration marker

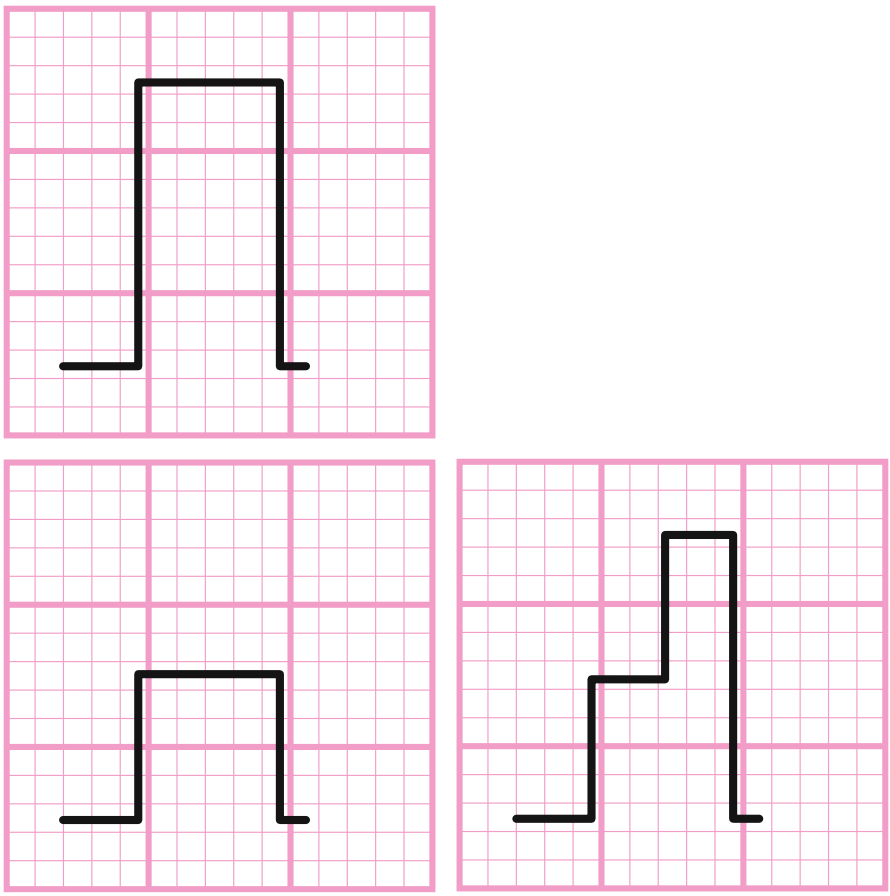


Fig. 3.8 *Upper left* image normal calibration marker. *Lower left* and *right* images at amplitude of 0.5 cm (set to half voltage)

If the settings are altered in any way they should be documented on the ECG to aid interpretation. When recording the ECG the calibration markers should be reviewed to ensure they are in text form at the bottom of the ECG printout.

The standard chart speed for ECG recording in the UK and USA is 25 mm/s. The speed can however be changed. This will in turn change the appearance of the

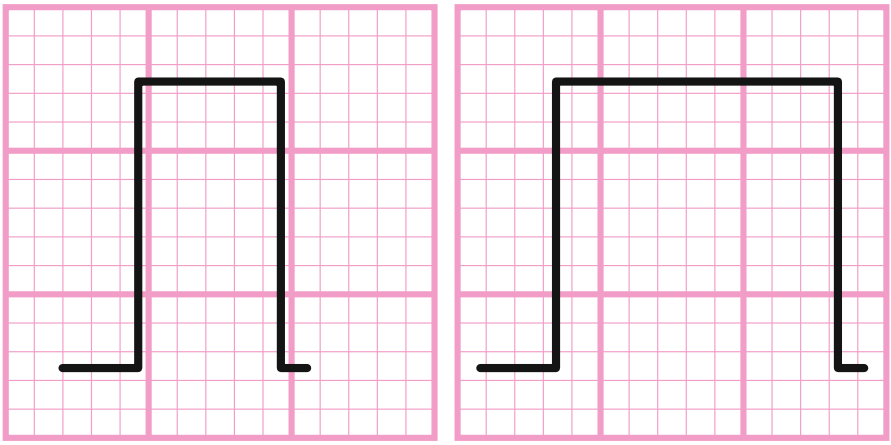


Fig. 3.9 *Left* image shows a normal calibration marker 25 mm/s. *Right* image shows calibration marker at 50 mm/s

calibration markers. For example changing the speed to 50 mm/s has the effect of doubling the width of the marker, as shown in Fig. 3.9.

Artifact

Is an artificial disturbance or interference which negatively effects the quality of the ECG. There are many different types of artefact which can present on an ECG recording. The three most commonly encountered types of artefact are:

- Muscle/somatic tremor.
- 60-cycle/AC mains interference.
- Baseline wander/sway.

Muscle/Somatic Tremor Artefact

This is probably the most commonly encountered form of artefact and is caused by muscular movement causing interference. The interference picked up by the ECG machine originates in muscles other than the heart (Fig. 3.10).

Primary Causes of Muscle/Somatic Tremor Artefact
- Pathological tremor (e.g. Parkinson's disease)
- Shivering
- Hiccups
- Movement of patient

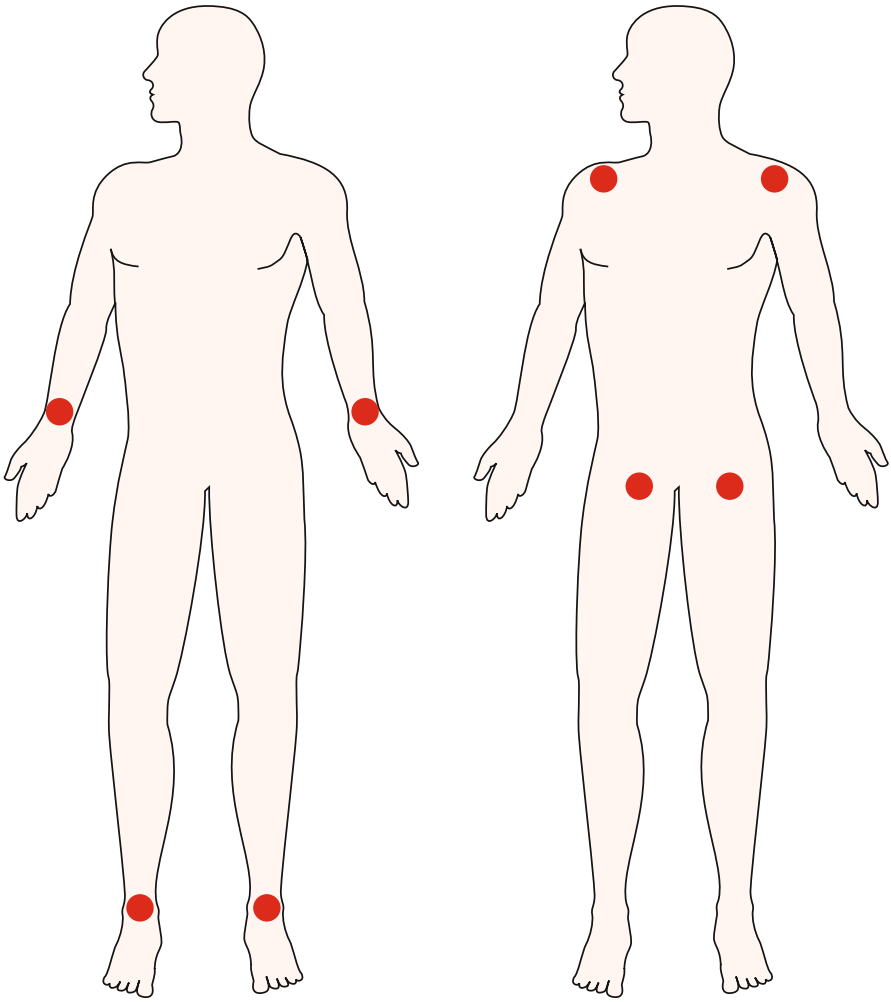


Fig. 3.10 *Left*: Standard limb lead position, *Right*: Modified limb lead positions

One of the potential problems caused by this form of artefact is that it can lead to misdiagnosis. This can arise due to loss of detail obscured by movement. This in turn makes it difficult to measure intervals and see waveforms, especially P waves and other smaller features such as the U wave.

Steps should be taken to remove or reduce this form of artefact prior to recording. When a patient is anxious, the patient should be reassured and fully informed of the reason for undertaking the 12-Lead ECG and their fears addressed. When a patient is cold the temperature should be increased and/or extra blankets applied. The patient should only be exposed for the minimum amount of time necessary to facilitate the recording of the ECG.

Where tremors are present due to pathology, such as myoclonic jerks or Parkinson's disease, the limb leads can be moved closer to the heart creating a modified ECG (Fig. 3.11). As a tremor is usually more pronounced toward the distal end of the

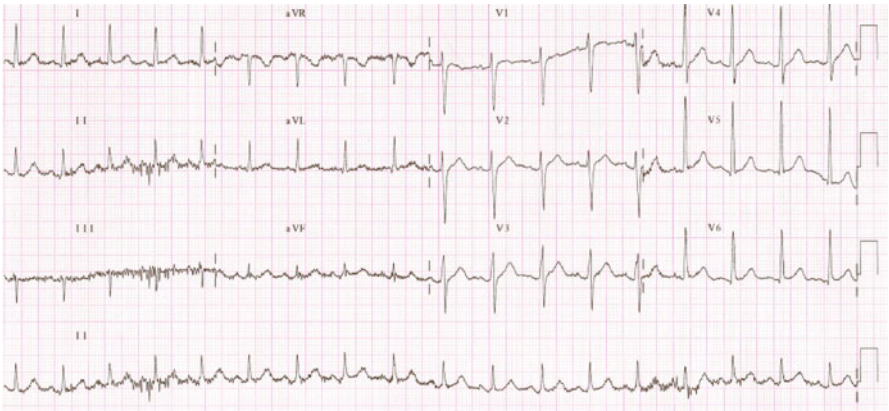


Fig. 3.11 Somatic muscle tremor in multiple leads, seen predominantly in leads II and III

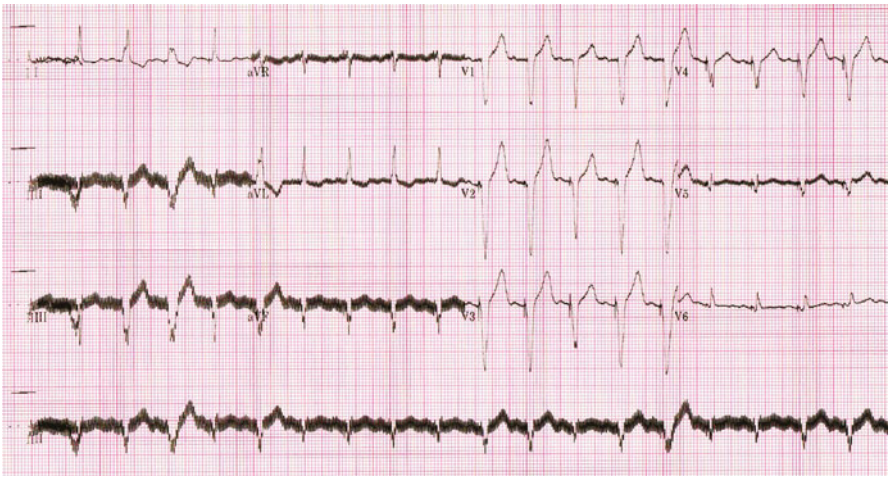


Fig. 3.12 60-cycle interference/AC mains interference

limbs, bringing the limb leads closer to the central body reduces some of this interference.

If a modified position is used, it should be subsequently documented on the ECG that the recording was carried out with limb leads in modified position, as this can affect the overall appearance of the ECG (i.e. the height of certain waveforms).

60-Cycle Interference/AC Mains Interference

This is caused by improper grounding of electrical equipment. It can be identified by the presence of 60 small spikes in a 1 s interval on the ECG, hence the name 60-cycle interference. The easiest way to spot this type of interference is by the presence of a thick dark black baseline (Fig. 3.12).

Primary Causes of 60-Cycle Interference
- Improperly grounded electrical equipment operating at a frequency of 50 Hz or greater.
- Close proximity of other electrical equipment.
- A fractured wire within a cable.

When presented with AC mains interference, attempts should be made to determine the source of the interference and remove it, or in the case of medical equipment, turn off any nonessential devices, such as infusion pumps that could potentially interfere with ECG quality. The bedside area may also contain patient's own electrical equipment such as radios or electric shavers; it is recommended that such equipment should be formally evaluated by the biomedical department to determine any possible cause of interference as well as general safety.

If there is no equipment within proximity that can explain the cause then another ECG machine can be used to determine if the problem lies with the machine itself. If machine error is suspected then the ECG machine should be inspected and repaired/replaced by appropriately trained personnel.

Baseline Wander

Literally a wander of the baseline (see Fig. 3.13), this form of artefact can increase the difficulty of ECG interpretation.

Primary Causes of Baseline Wander
- Anxiety
- Pain
- Perspiration
- Cable movement
- Pulmonary conditions

This form of artefact can be reduced or eliminated by ensuring cable movement is minimized, i.e. cable not dangling over the edge of the bed/trolley or twisted around each other. In the case of respiratory swing, which is found in patients with certain respiratory diseases such as COPD, the patient may have to be placed in a more upright position.

To reduce the possibility of base line wander being caused by perspiration, the patient's skin can be cleansed with an alcohol wipe. Alcohol wipes also remove other debris that can interfere with electrode contact. Pain and anxiety should also be considered and reduced or prevented wherever possible.

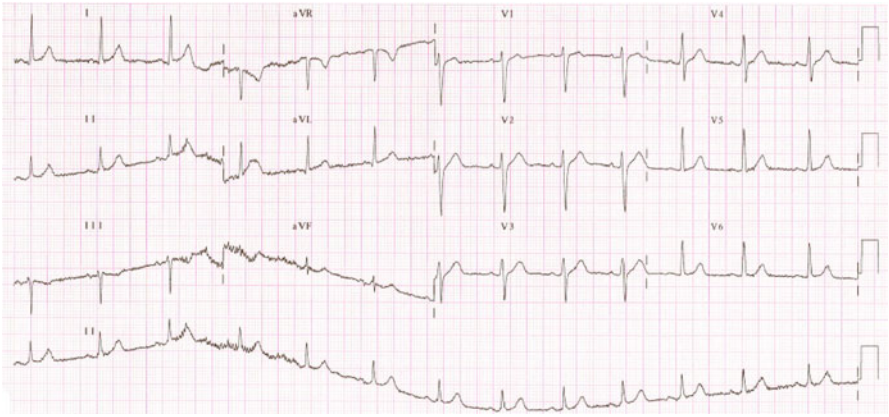


Fig. 3.13 Baseline wander

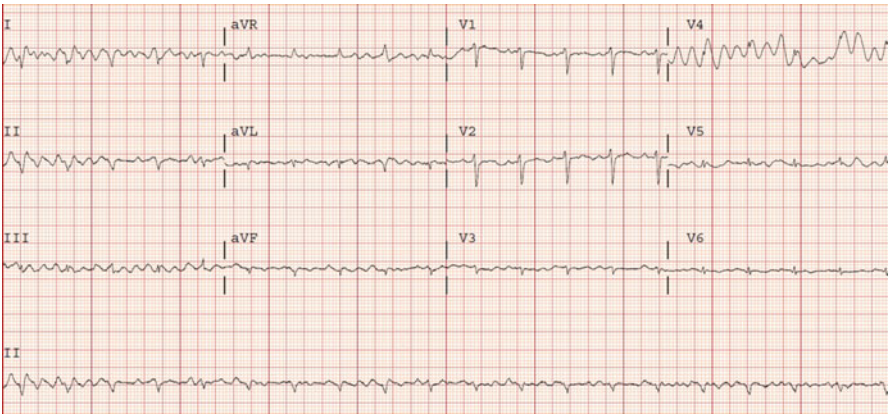


Fig. 3.14 Artefact mimicking atrial flutter

Other Forms of Artefact

There are also forms of artefact which occur less commonly than those stated above. Sometimes the cause is more difficult to ascertain. It is often the case in clinical settings that one member of staff may record an ECG, while another will interpret the results, i.e. a nurse will record an ECG and a doctor will comment on the ECG clinically. It is assumed that the person recording the ECG has done so in a standard and systematic way, and has attempted to remove all kinds of interference prior to recording. Figure 3.14 shows an ECG which at first glance looks like a condition known as atrial flutter.

The actual cause of the 'flutter waves' was interference caused by the patient being too tense and anxious. The patient in question was an elderly lady who had tensed her neck during recording, leading to the interference shown. After the patient was relaxed back properly the ECG was re-recorded showing no significant abnormalities. This case highlights the potential danger of misdiagnosis caused by interference or poor recording practices. Most forms of artefact can be reduced or removed completely following a few simple steps:

Reducing Artefact
- Ensure correct positioning of patient
- Ensure patient is relaxed and not too hot or cold
- Ensure equipment is in good working order and cables do not dangle over the edge of the bed/trolley or get twisted
- Shave excess body hair to ensure good electrode contact
- Use alcohol gel to dry skin prior to electrode attachment
- Turn off all unnecessary electrical equipment to reduce potential interference.

ECG Documentation

The health care practitioner who records an ECG should also ensure that the ECG has key information documented on it, as highlighted in Chap. 1. This information should include:

- Patient details: Name, hospital number, DOB and age.
- The date and time the recording was made.
- The name/title of the person making the recording.
- Any additional useful information, including any change in patient position or electrode position.
- Any other relevant medical data.

Summary of Key Points

- Knowledge of different types of ECG interference and methods of its reduction can increase the quality and diagnostic value of the 12-lead ECG
- Lead aVR is usually negatively deflected. If not, check the cables are attached to the correct limbs
- Check the chart speed and calibration markers on all ECGs recorded before attempting to interpret them
- Most of the common forms of artefact can be prevented or reduced prior to recording the ECG.

Quiz

Q1. Lead aVR is normally negatively deflected

(A) False
(B) True

Q2. Poor R wave progression can be an indicator

(A) That the chest leads have been misplaced
(B) That other medical equipment is interfering with the ECG recording
(C) The patient is not relaxed

Q3. The standard calibration markers on a 12-lead ECG are

(A) 4 cm in height (8 large boxes)
(B) 2 cm in height (4 large boxes)
(C) 1 cm in height (2 large boxes)

Q4. 60-cycle or AC mains interference is most commonly caused by

(A) A cold or anxious patient
(B) A fractured cable or improper grounding of electrical equipment
(C) Parkinson's disease

Q5. The standard ECG chart speed in the UK and the USA is said to be

(A) 45 mm/s
(B) 25 mm/s
(C) 15 mm/s

Q6. Checking the quality of an ECG is the job of

(A) A trained cardiac nurse
(B) A doctor
(C) Whoever records the ECG

Answers: Q1 = B, Q2 = A, Q3 = C, Q4 = B, Q5 = B, Q6 = C

Chapter 4
Principles of ECG Analysis

Abstract The purpose of this chapter is to look at the normal parameters that should be measured systematically. Later chapters discuss the meaning behind values that fall outside this 'normal' range.

Keywords Analysis • Interpretation • Rate • Rhythm • Axis

Background

The purpose of this chapter is to look at the normal parameters that should be measured systematically. Later chapters discuss the meaning behind values that fall outside this 'normal' range.

In order to interpret a 12-lead ECG correctly, a systematic method of examination is required. It is often easy in the clinical environment where time is at a premium to spot certain features on an ECG, and report on those features, possibly missing other important findings because a systematic method was not used. There are many different methods of systematic analysis to choose from, most cover the same basic principles, but may differ in order. The method proposed in this text consists of an examination of the following ECG features:

- Basic quality control checks
- Rate
- Rhythm
- P wave
- PR interval

The online version of this chapter (doi:10.1007/978-1-4471-4962-0_4) contains supplementary material, which is available to authorized users.

Please note some of the figures within this chapter have been reproduced in full size online at Extra Materials (extras.springer.com) for ease of viewing

- The QRS complex
- The ST segment
- The T wave
- The QT interval
- Additional features

Basic Quality Control Checks

Prior to interpreting the ECG it is helpful to perform some quick quality control checks (As discussed in Chap. 3). A brief check should ensure the ECG was recorded under normal parameters, and if not that you as the interpreter are aware of any changes made during the recording process. Checks should include:

- Calibration markers and recording speed
- Correct lead placement (R wave progression/lead aVR)
- That the quality of recording is sufficient to make an adequate diagnosis

The Rate

The next thing to determine is the heart rate. Many ECG machines will work this out automatically and display this information on the ECG. It is however important that as an interpreter you are able to work this out yourself. Machines can make mistakes, and some older machines do not display this information. There are many different methods that can be used to determine the rate. In this text we only demonstrate the use of one method, as this is easier to remember a single method when starting out. The method in question works for both regular and irregular heart rates, reducing the need to learn several different methods.

Step 1: Using the rhythm strip at the bottom of the ECG (commonly lead II or V_1), count any 30 large squares (Fig. 4.1).

Step 2: Count the number of QRS complexes within the 30 large squares chosen (Fig. 4.2).

Step 3: Multiply this number by 10 to get the heart rate. $7 \times 10 = 70$. Therefore the heart rate is approximately 70 BPM. This method works by determining an average from the chosen section. The ECG machine recorded a rate of 68 BPM for this patient, so although not exact, this method is fairly accurate.

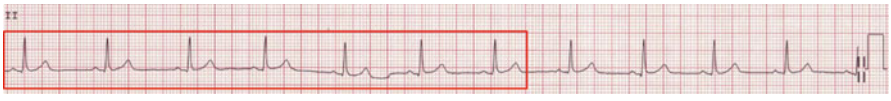


Fig. 4.1 30 large squares

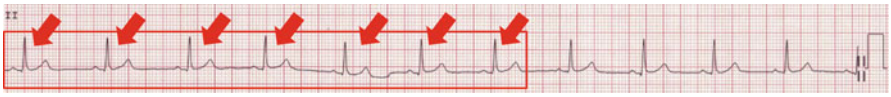


Fig. 4.2 In this example there are 7 QRS complexes in the 30 square area

The Maths
- Remember, each large box represents 0.2 s.
- So $0.2 \times 30 = 6$ s of time.
- $6 \times 10 = 60$ which is a minute.

Remember the 'normal' heart rate is said to be between 60 and 100 BPM (Beats Per Minute). A rate below 60 is a bradycardia, a rate above 100 is a tachycardia.

The Rhythm

There are two main ways of determining if the rhythm is regular or irregular. The first and arguably best method is to use is a set of calipers (like a compass, but with a needle on both ends). This works by placing the first point on the tip of an R wave along the rhythm strip section of the ECG. The caliper is then widened until the second needle touches the next R wave. The calipers can then be swung from R wave to R wave to determine if the rhythm is regular (Fig. 4.3).

Another way of achieving a similar result is to place a piece of paper just below the tips of the R waves and make a mark on the paper, just below two consecutive R waves. The paper can then be moved across the strip to determine if the rhythm is regular or not by seeing if the R waves remain in line with the paper marks (Fig. 4.4).

Questions that should be asked when looking at the rhythm include:

- Is the rhythm regular or irregular?
- If the rhythm is irregular, is it irregularly irregular or is there a pattern to the irregularity
- Are PQRST waves present?

The P Wave

The next step is to analyse the P wave (Fig. 4.5). The best leads to view the P wave are leads II and V_1. The principle features the interpreter should look for are:

- There are P waves present on the ECG
- That the morphology of the P waves is the same. (i.e. that they look the same as each other)

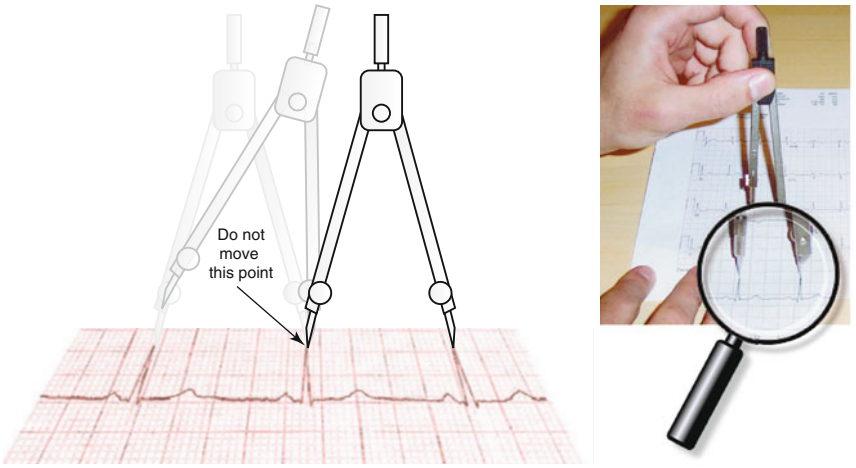
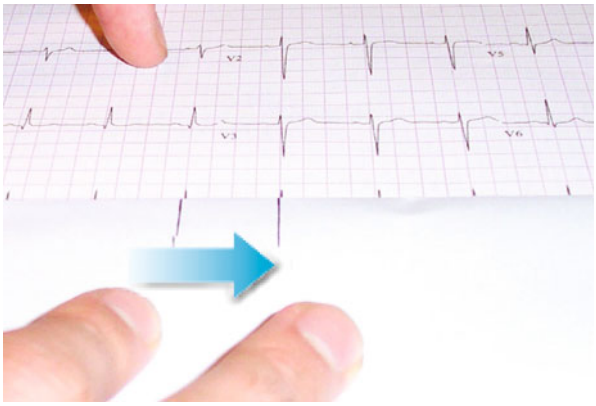


Fig. 4.3 The caliper method of determining rhythm regularity

Fig. 4.4 The paper method of determining rhythm regularity



- That each P wave is followed by a QRS complex
- The height of the P wave is no more than 2.5 mm (that's 2 and half small boxes)
- The width of the P wave is no greater than 2.5 mm
- That the P wave is positively deflected (points upward) in lead II
- That the P wave is smooth and uniform and not notched (bifid).

The PR Interval

The next stage is to measure the PR interval, measured from the start of the P wave to the beginning of the QRS complex (Fig. 4.6). The PR interval should be 0.12–0.20 s (3–5 small squares) in duration. The PR interval should be consistently the same length and not variable. In addition to this, the PR interval should be isoelectric and not dipped below the baseline.

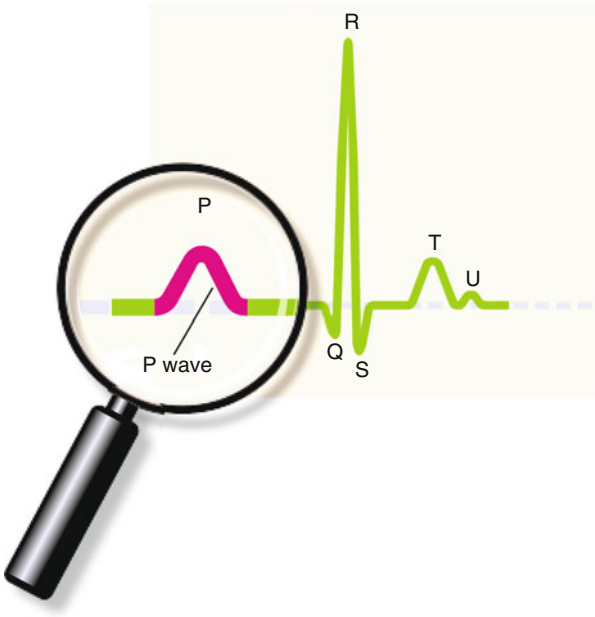


Fig. 4.5 The P wave

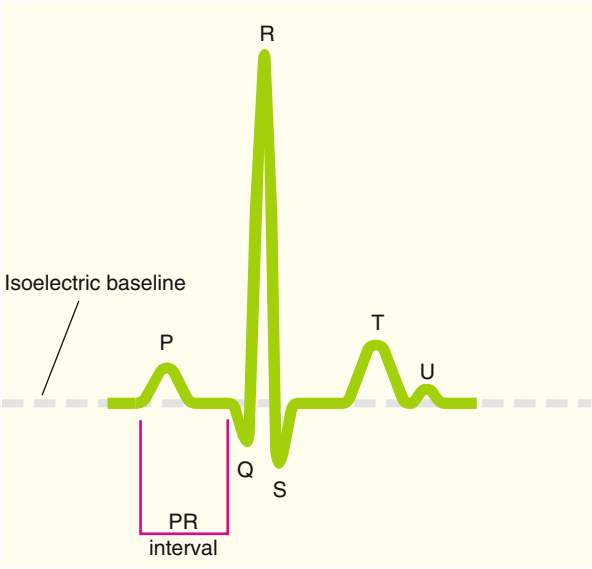


Fig. 4.6 The PR interval

The QRS Complex

The QRS complex is measured from the start of the Q wave to the end of the S wave (Fig. 4.7).

The main features to look for when examining the QRS complex are:

- The QRS complex is present
- The width of the QRS complex should be between 0.06 and 0.10 s (1.5–2.5 small squares)

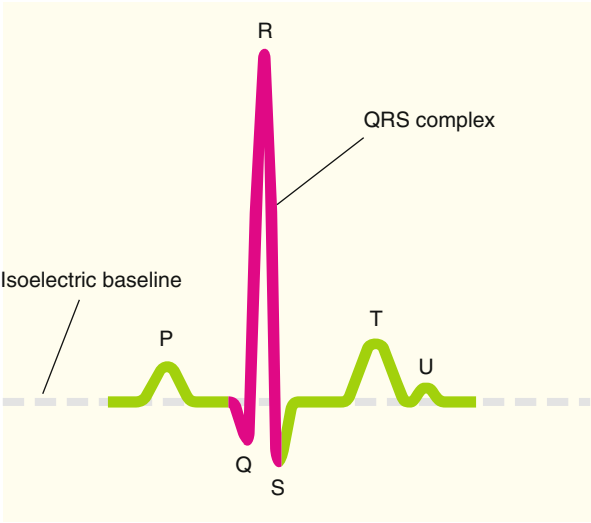


Fig. 4.7 The QRS complex

- The amplitude of the R and S wave should be less than 25 mm (5 large squares)
- The QRS complexes should share the same morphology
- A QRS complex follows each P wave
- The Q wave is less than 2 mm in depth and less than 1 mm in width.

The ST Segment

The ST segment is measured from the end of the S wave to the start of the T wave (Fig. 4.8).

The ST segment should be Isoelectric and not elevated above or depressed below the baseline. The ST segment should be examined in all leads.

The T Wave

The T wave follows the QRS complex and is usually asymmetric in appearance (Fig. 4.9). The T wave should be deflected in the same direction as the QRS complex and not be inverted. They should also be less than two thirds the height of the preceding R wave and not be too tall or peaked in appearance.

The QT Interval

The QT interval is measured from the start of the QRS complex to the end of the T wave (Fig. 4.10).

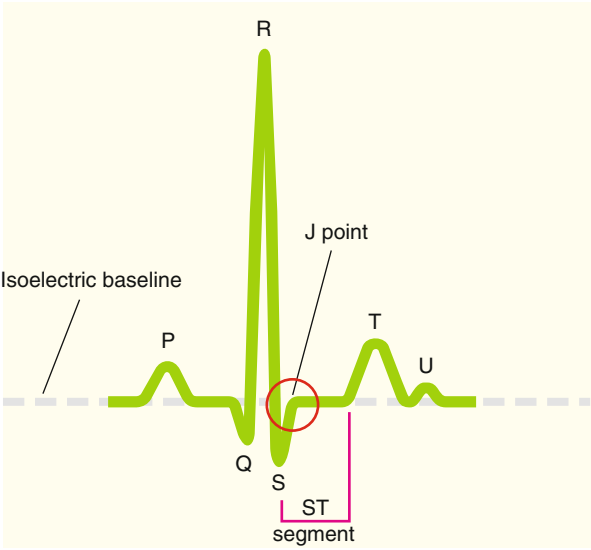


Fig. 4.8 The ST segment

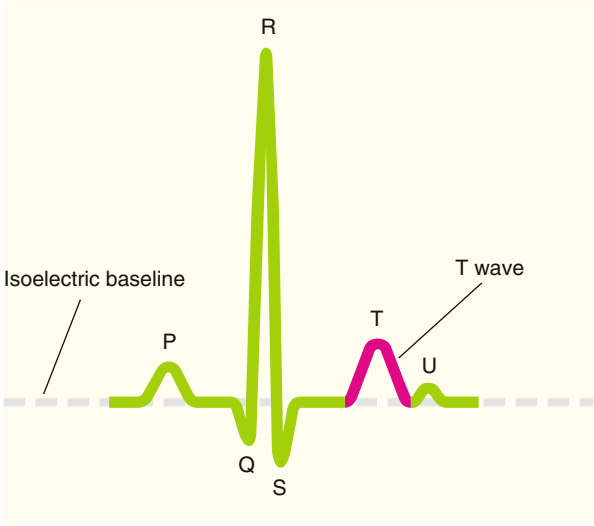


Fig. 4.9 The T wave

The QT interval should be around 0.36–0.44 s and not more than half the distance between two consecutive R waves. One of the problems with the QT interval is that as the heart rate increases the QT interval decreases. This occurs to ensure that the myocardium has reploarised sufficiently prior to the next depolarisation. In order to get a correct reading this effect needs to be compensated for. To this end a formula was created to calculate the compensated QT interval (QTc) (Fig. 4.11). The formula works by dividing the QT interval in seconds by the square root of the preceding R to R interval in seconds. This sounds complicated but fortunately most modern machines will work this out for you.

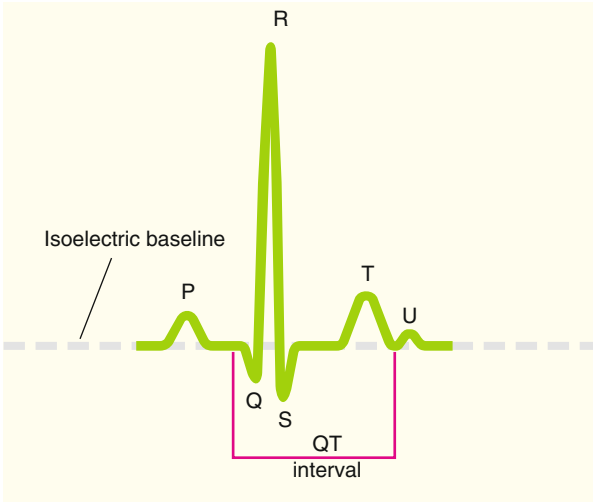


Fig. 4.10 The QT interval

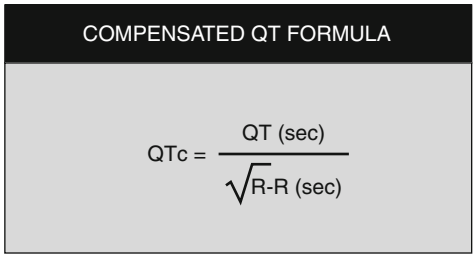


Fig. 4.11 Compensated QT formula (QTc)

Electrical Axis

Calculating the electrical axis is often considered one of the more complex elements of ECG interpretation. The electrical axis refers to the net or overall direction in which the electrical current travels in during ventricular depolarisation. This is achieved by adding together the sum of all of the individual vectors to determine the overall direction of the flow across the whole myocardium. Calculating the electrical axis is useful as it can provide additional evidence for the presence of various conditions.

As this book is intended to be a beginner's guide, the subject of cardiac axis is not discussed in depth. The reader should however be aware that there are several methods used to calculate cardiac axis available; such as the hexaxial reference system, and vector mathematics. The different methods available also vary in accuracy.

For the purpose of this text a simple (but less accurate) method of axis determination is provided. To work out if axis is normal or not look at the QRS complex's

Table 4.1 QRS axis quick reference guide

QRS AXIS		
Lead I (deflection)	Lead aVF (deflection)	Axis
		Normal Axis
		Left Axis
		Right Axis
		Extreme Right Axis

in leads I and aVF. If both the QRS complex in I and aVF point upwards (Are positively deflected) then the axis is normal. If the complexes are positive in lead I and negative in aVF then the axis is more leftward (left axis). If the opposite is true then the axis is more rightward (right axis). If the QRS complexes are negatively deflected in both leads then the axis has shifted to the extreme right (Table 4.1).

Some Conditions That Can Cause an Axis Shift
- Pregnancy & other mechanical shifts
- Aging process Inferior/lateral wall MI (Myocardial Infarction)
- Hemiblocks
- Bundle branch blocks
- Dextrocardia
- Emphysema

Additional Features

After performing all of the basic checks the ECG can be checked for any additional features, such as the presence of additional waves i.e. U waves, the notching of waves and/or any other notable features.

The Normal ECG and Normal Variants

To become proficient at reading ECGs there is no substitute for practice. Look at as many ECGs as possible (Fig. 4.12) and try to spot any abnormalities. Discuss your findings with a senior colleague who is trained in ECG interpretation. Slowly over time you will become more proficient. One of the problems many people face when new to ECG interpretation is being able to spot what is abnormal from what is normal. It takes seeing many, many normal ECGs to get a handle on this. There are

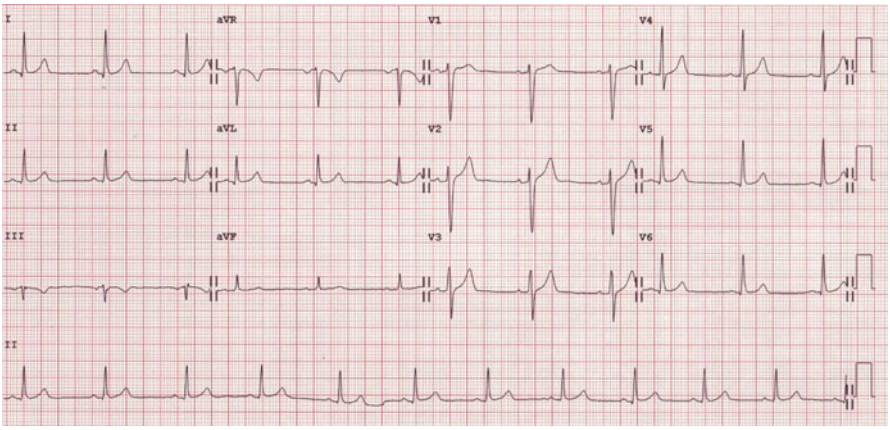


Fig. 4.12 A normal ECG

many 'normal variations' found on ECGs that interpreters should be aware of. Some of the main ones are listed below:

Bradycardia +/– Large R Waves

Tall thin individuals may have larger R waves. So can fit people, a condition called athletes heart is often found in the fit due an increase in heart size cause by exercise (discussed in Chap. 5). The same can be said for bradycardia, many fit people will have a lower resting heart rate.

Leads aVR and V1

Complexes look different in these leads. In aVR P, QRS and T waves are usually the opposite way up, this is normal for this lead. T waves may also be inverted in V1 as a normal variant.

Sinus Arrhythmia

Sometimes the rhythm will be irregular with all waves present (PQRST). A 10 % variance is considered quite normal. In the case of sinus arrhythmia the rhythm is irregular and corresponds to the respiratory cycle. This is caused by the heart rate increasing during inspiration. This is often found in the elderly, children or fit people, and causes no symptoms.

Q Waves

Q waves greater than 2 mm in depth can sometimes be seen. They are usually found in lead III, and without chest pain. If the patient is attached to a cardiac monitor and asked to take a deep breath the Q waves will normally diminish in depth indicating that they are a normal variant associated with a slight shift of the heart in the chest.

T Wave Changes

Sometimes ethnic origin can play a part in ECG findings. For example widespread T wave inversion (without pain) can be a normal variant for Afro-Caribbean males.

Remember
If in doubt about a finding being a normal variant or not, ask a senior colleague. It is far better to draw attention to a normal variant than to miss a possible serious condition. All ECGs should be checked by a trained member of staff until you as an interpreter are deemed competent and allowed to carry out this task independently by your employers.

When interpreting ECGs. ALWAYS look at the patient. The ECG is a useful guide, but without other observations and patient history, mistakes can be made. Always endeavour to see the patient yourself or a least get a detailed description of the patient if you are interpreting an ECG. Another useful aid is the patient's previous ECG(s). If available use an old ECG to look for changes and pre-existing conditions relevant to your patient.

Summary of Key Points

- A systematic approach should always be used when interpreting an ECG
- An awareness of normal variants is a useful aid to interpretation
- Always check the patient's yourself and obtain a detailed history
- There is no substitute for practice, the more ECGs you see the better and the more confident you will become at interpreting them.

Quiz

Q1. The 'normal' PR interval should be between…

(A) *3–5 small squares*
(B) *4–5 small squares*
(C) *3–6 small squares*

Q2. The ST segment should be…

(A) *Elevated above the base line*
(B) *Isoelectric*
(C) *Depressed below the baseline*

Q3. An inverted T wave in V_1 is always abnormal

(A) *False*
(B) *True*

Q4. Which two leads are used to determine the electrical axis?

(A) *I and III*
(B) *I and aVL*
(C) *I and aVf*

Q5. Q waves should be…

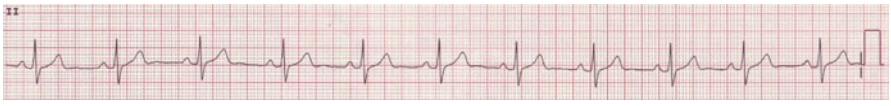
(A) *<2 mm in depth*
(B) *<5 mm in depth*
(C) *<1 mm in depth*

Q6. It is important to always see the patient if possible and get a detailed history as opposed to relying solely on the ECG

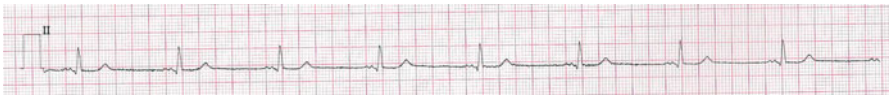
(A) *True*
(B) *False*

Work out the heart rate for the following ECG rhythm strips using the method demonstrated earlier.

Q7.



Q8.



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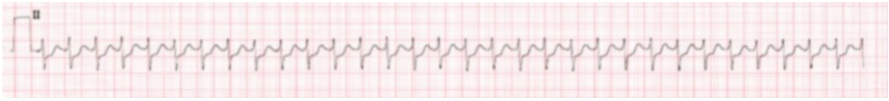

Answers: Q1 = A, Q2 = B, Q3 = A, Q4 = C, Q5 = A, Q6 = A, Q7 ≈ 70 BPM, Q8 ≈ 50 BPM, Q9 ≈ 190 BPM

Chapter 5
Chamber Abnormalities

Abstract As a result of physiological or pathological changes, the chambers of the heart may become enlarged. This can be due to an increase in the thickness of the wall of a heart chamber, caused by hypertrophy, or dilation (an increase in the radius of the chamber without wall thickening). The ECG alone cannot make a complete diagnosis of hypertrophic chamber abnormality, it can however help to provide supporting evidence for its potential presence. Echocardiographic studies remain the 'gold standard' for identifying hypertrophic changes. It is important to have an awareness of some of the principles of cellular growth and understand the difference between hyperplasia and hypertrophy.

Keywords Hypertrophy • Chamber enlargement • Voltage criteria • Chamber abnormality

Physiology

As a result of physiological or pathological changes, the chambers of the heart may become enlarged. This can be due to an increase in the thickness of the wall of a heart chamber, caused by hypertrophy, or dilation (an increase in the radius of the chamber without wall thickening). The ECG alone cannot make a complete diagnosis of hypertrophic chamber abnormality, it can however help to provide supporting evidence for its potential presence. Echocardiographic studies remain the 'gold standard' for identifying hypertrophic changes. It is important to have an awareness of some of the principles of cellular growth and understand the difference between hyperplasia and hypertrophy.

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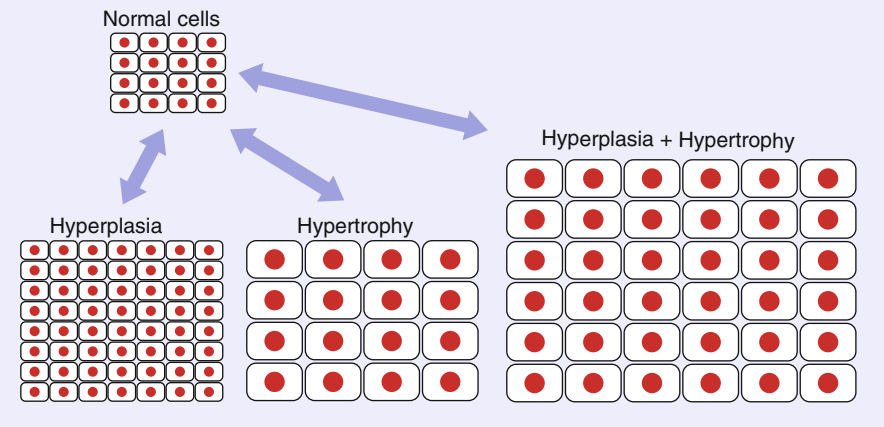


Fig. 5.1 Hyperplasia and hypertrophy

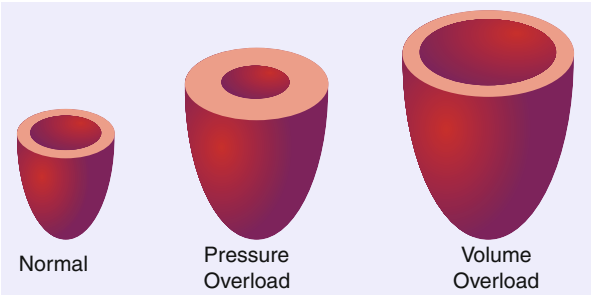


Fig. 5.2 Systemic and diastolic overload in relation to normal chamber size

Hypertrophy is an increase in cellular size. In contrast hyperplasia is an increase in the number of cells. Sometimes the two can be found together.

This is summarized in Fig. 5.1.

There are two distinct causes of chamber enlargement in the heart. The first is due to an overload of pressure, usually as a result of increased resistance. This can for example be caused by conditions such as systemic hypertension. This pressure overload usually results in an increase in chamber wall thickness. The second main cause of chamber enlargement is caused by volume overload. This can be due to heart failure, valvular regurgitation or stenosis, and often results in dilation of the chamber. This can be seen in Fig. 5.2, compared with a normal chamber, for example the ventricles.

Atrial Abnormality

As discussed previously, the P wave represents the depolarisation of both the left and right atria. The initial half of the P wave represents depolarisation of the right atria as the electrical impulse travels from the Sinoatrial node (SAN) to the right atrium and then the left. The impulse reaches the Atrioventricular node (AV) at the

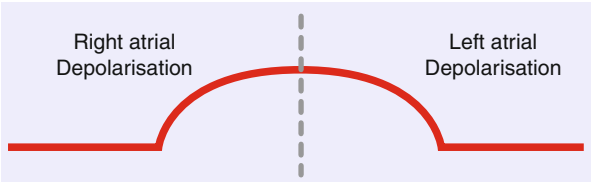


Fig. 5.3 Atrial activation

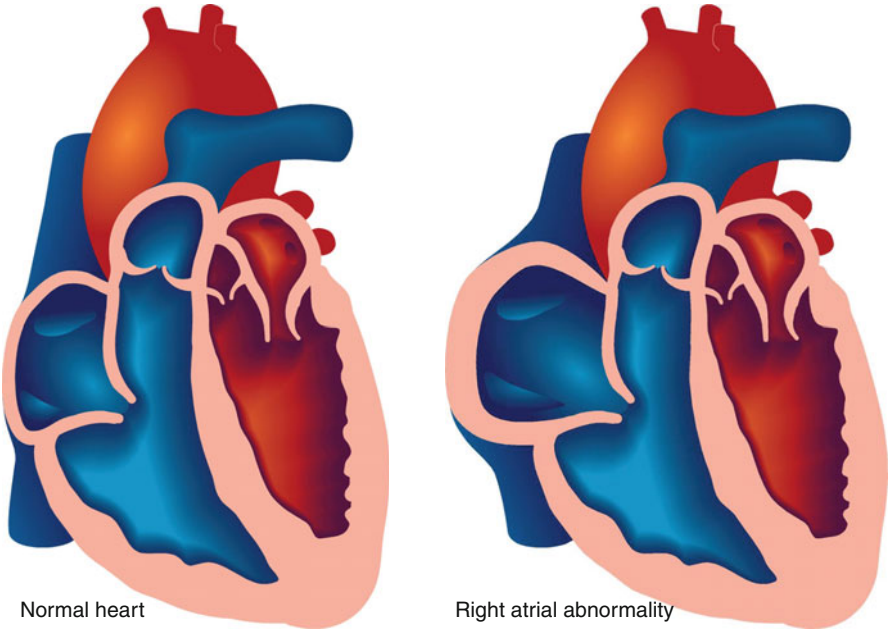


Fig. 5.4 A normal heart (*left*) and a heart with a right atrial enlargement (*right*)

midpoint of the P wave. The second half of the P wave denotes depolarisation of the left atrium (Fig. 5.3).

Changes in the size and shape of the P wave can point towards an atrial abnormality, which may be caused by right or left atrial enlargement (Figs. 5.4 and 5.6 respectively) or more rarely a conduction delay. Additional evidence for the presence of an atrial abnormality, enlargement, dilation or hypertrophy is the coexistence of a ventricular hypertrophy.

Right Atrial Abnormality

Right atrial abnormality, also known as P. pulmonale (Fig. 5.4). This is seen on the ECG as peaked P waves which are greater than 2.5 mm in amplitude (height). Remember each small square of ECG paper is 1 mm^2, so it's taller than two and half small squares (Fig. 5.5).

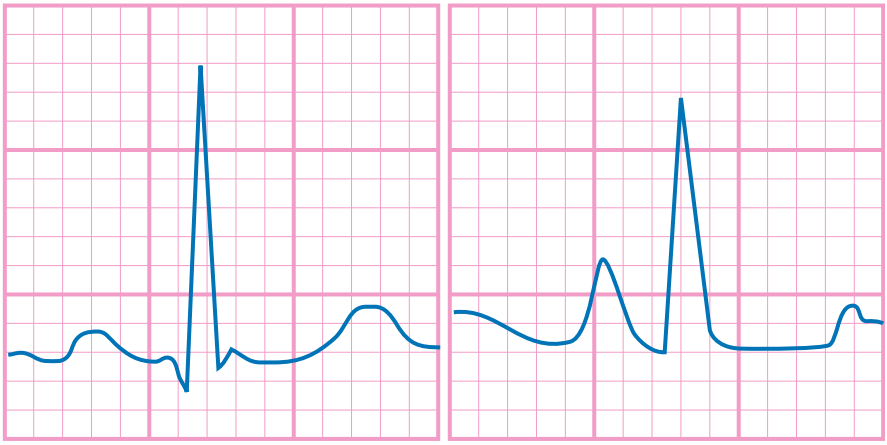


Fig. 5.5 Normal P wave (*left*), peaked P wave with amplitude >2.5 mm (*right*)

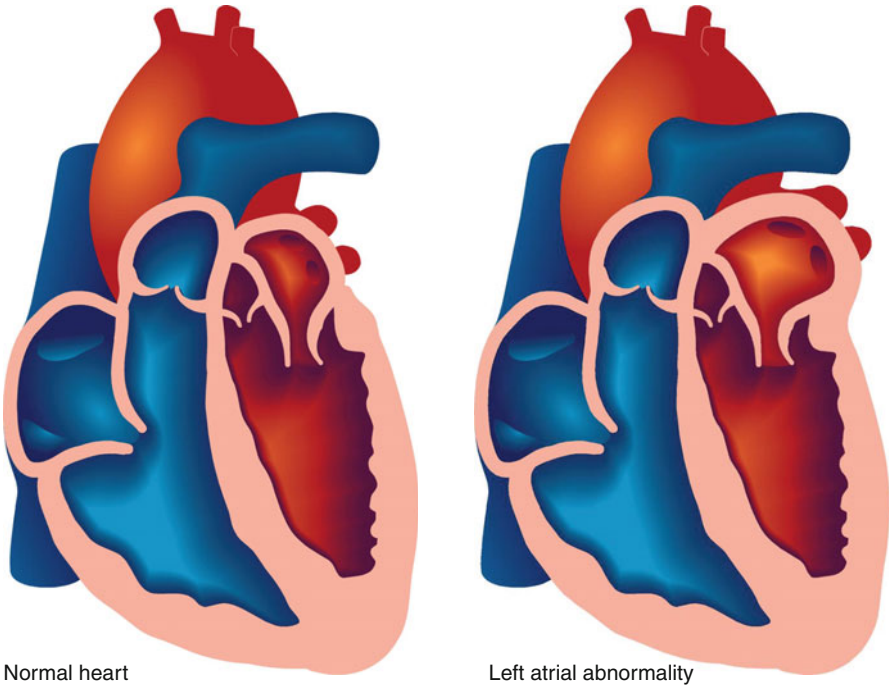


Fig. 5.6 A normal heart (*left*) and a heart with a left atrial enlargement (*right*)

The reason this condition is also known as P. pulmonale, is that it is associated predominantly with pulmonary causes. This condition will often be seen in patients with

- Pulmonary hypertension
- Pulmonary stenosis
- Pulmonary embolus

Look for these P wave changes in leads II, III and aVF. P waves may also be peaked. The presence of large and/or peaked P waves >2.5 mm in height should alert you to the possibility of right atrial abnormality.

Right Atrial Abnormality

Key Points

- P waves >2.5 mm in height and peaked
- Look in leads II, III and avF
- Associated with pulmonary causes

Left Atrial Abnormality

Left atrial abnormality, also known as P. mitrale, as it is commonly associated with mitral valve disease (Fig. 5.6). Left atrial abnormality is seen on an ECG as broad, notched/bifid 'M' shaped P waves. These P waves are often greater than 2.5 mm in width (see Fig. 5.7). This is best seen in leads II and V_1. Another indicator is seen in lead V_1 where there is a negative deflection below the base line of more than 1 mm and a width greater than 1 m (Fig. 5.8).

Left Atrial Abnormality

Key Points

- P waves >2.5 mm in width
- Notching of P wave 'M' shaped
- Negatively deflected >1 mm in V_1
- Look in leads II and V_1
- Associated with mitral valve disease

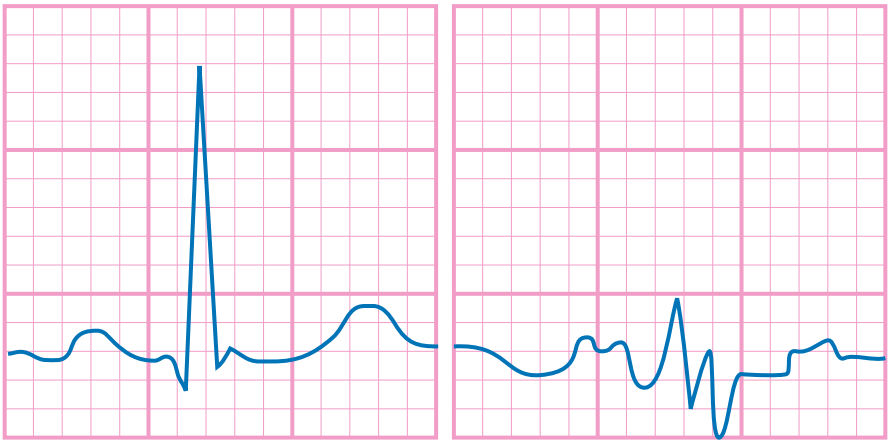


Fig. 5.7 Normal P wave (*left*), notched P wave (*right*)

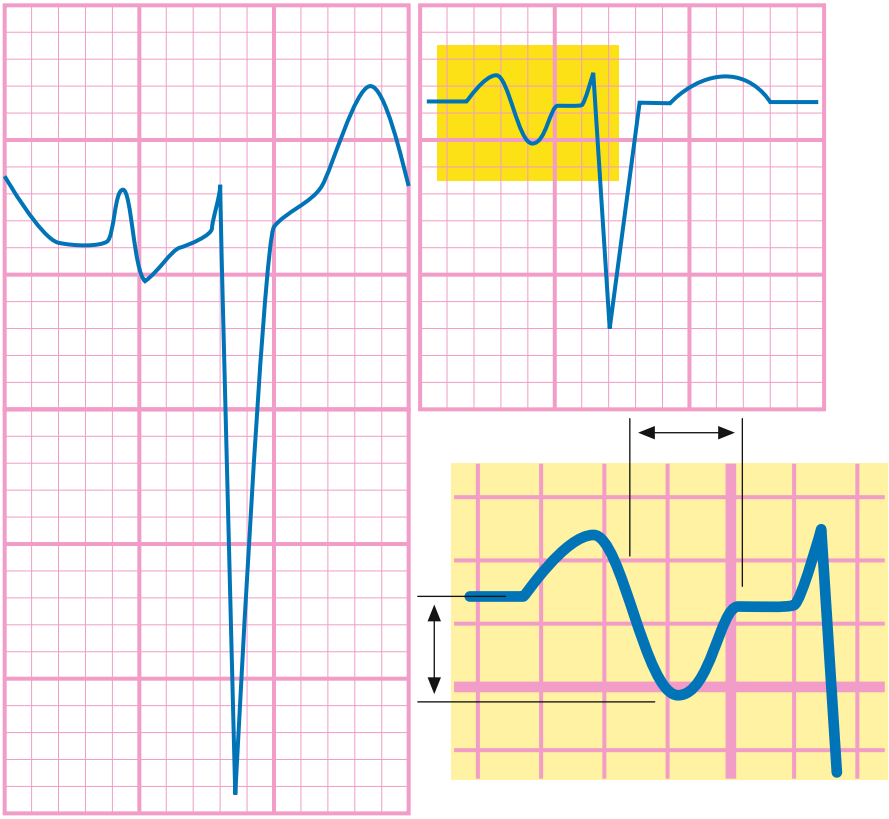


Fig. 5.8 P wave in lead V1 >1 mm in length and depth

Bilateral Atrial Abnormality

Bilateral atrial abnormality refers to an abnormality that affects both atria (Fig. 5.9). This can be detected by the combination of indicators for both right and left atrial abnormality, present on the same ECG. Table 5.1 summarises some of the key P wave changes present in right, left and bilateral atrial abnormality.

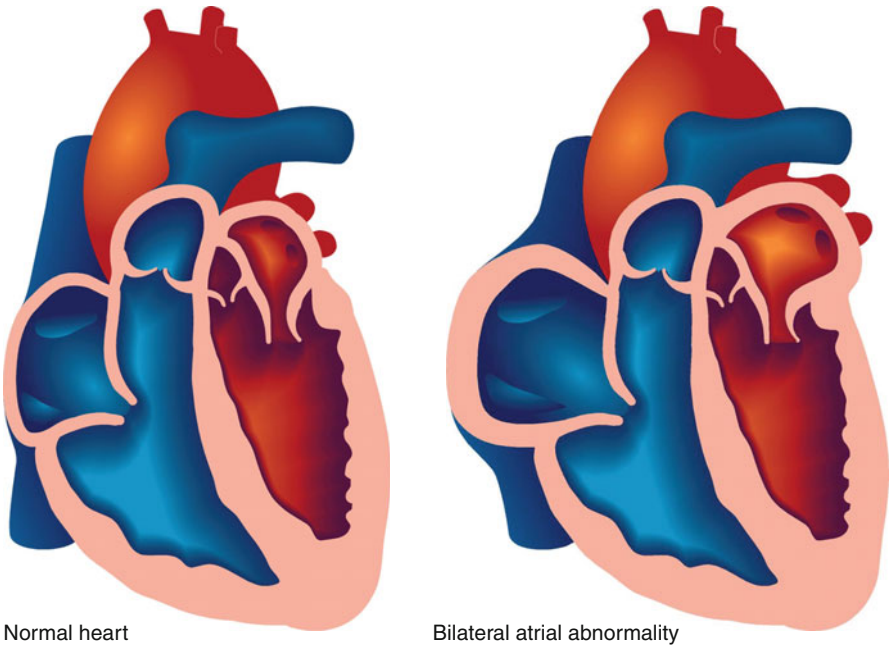


Fig. 5.9 A normal heart (*left*) and a heart with both left and right atrial enlargement (*right*)

Table 5.1 P wave changes in leads II and V1, occurring with atrial abnormalities

Abnormality	Lead II	Lead V_1
Right atrial abnormality		
Left atrial abnormality		
Bilateral atrial abnormality		

Ventricular Abnormality

Left Ventricular Hypertrophy (LVH)

The potential presence of LVH is identified by an increase in the QRS voltage (Fig. 5.10), this is due to the increased muscle mass of the hypertrophied left ventricle (Fig. 5.11). There are many different scoring systems and criteria identified in the texts for detecting LVH (e.g. Romhilt-Estes scoring system, Sokolow-Lyon criteria, Cornell criteria, etc.). For the purpose of this introductory text, only two methods are shown, one using the limb leads and the other using the chest leads.

The S wave in lead V_1 is added to the R wave in V_6. If the sum is greater than or equal to 35 mm then the voltage criteria for LVH is met. The other method that can be used involves the limb leads I and aVL. If a QRS complex exceeds 20 mm (4 large boxes) then the voltage criteria for LVH can be seen using just the limb leads.

Causes of LVH
- Hypertension
- Hypertrophic cardiomyopathy
- Aortic regurgitation/stenosis

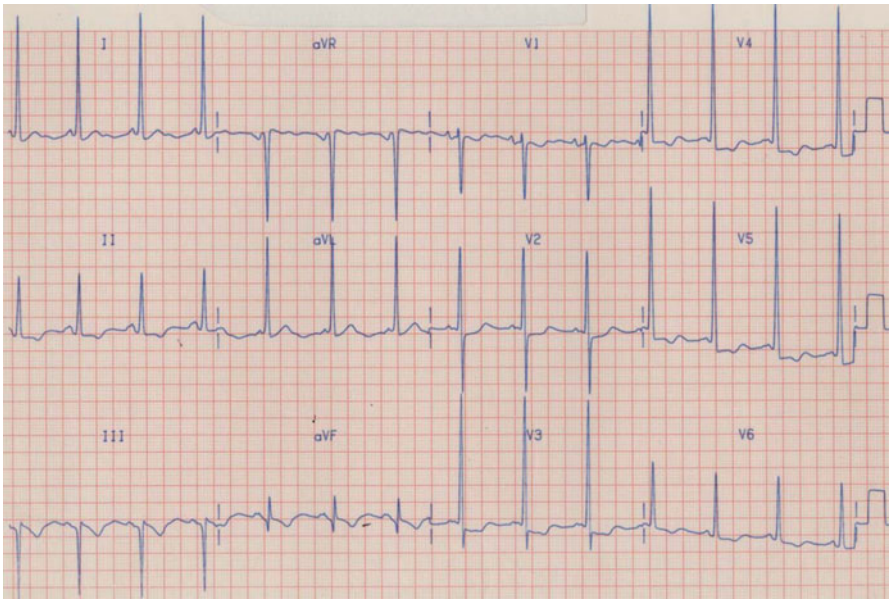


Fig. 5.10 Increased QRS voltage

LVH Key Points
(not all of these may be present)

- Left axis deviation
- S wave in V_1 + R wave in V_6 ≥35 mm in height
- Accompanied left atrial abnormality
- In severe cases associated ST segment depression and T wave inversion, 'strain pattern'.

General Tip
When recording findings on the ECG it is good practice to write something like 'voltage criteria for LVH met' instead of LVH because only a cardiac echo can truly diagnose LVH.

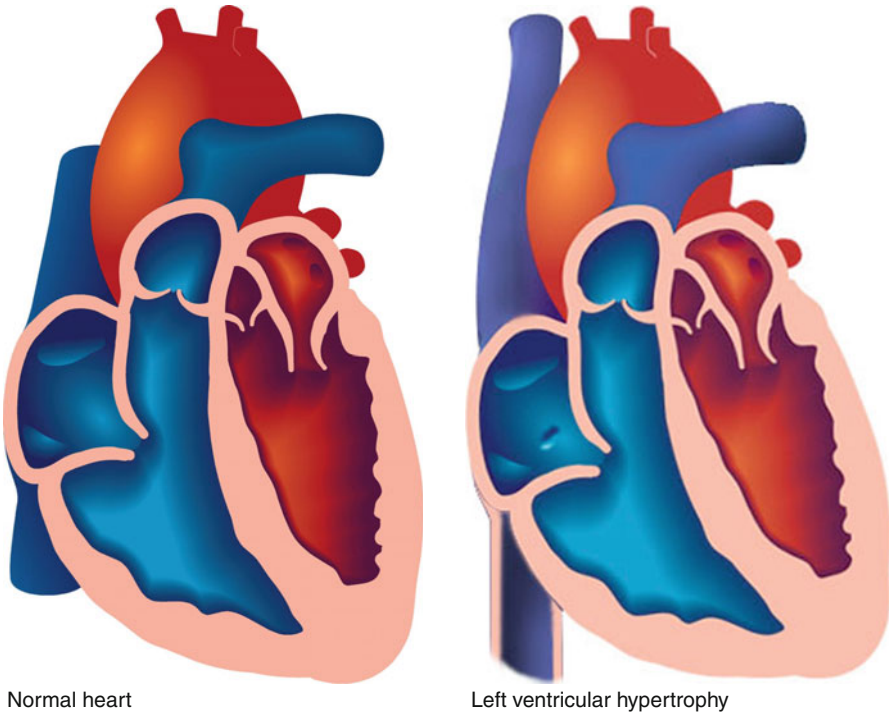


Fig. 5.11 A normal heart (*left*) and a heart with LVH (*right*)

Right Ventricular Hypertrophy (RVH)

Right ventricular hypertrophy (Fig. 5.12) is less common than left ventricular hypertrophy. RVH is often accompanied by right atrial abnormality and tall R waves in lead V_1 (height of R wave > depth of S wave), and right axis deviation. In more severe cases ST segment depression and T wave inversion, indicating 'strain' pattern can sometimes be seen (Fig. 5.13).

To determine the voltage criteria for RVH the R wave in lead V_1 is added to the S wave in lead V_6. If the sum is 10 mm or more then the voltage criteria for RVH is present. This can be seen in Fig. 5.11.

Causes of RVH
- Chronic lung disease
- Pulmonary hypertension
- Congenital defects
- Atrial septal defect
- Pulmonary valve stenosis

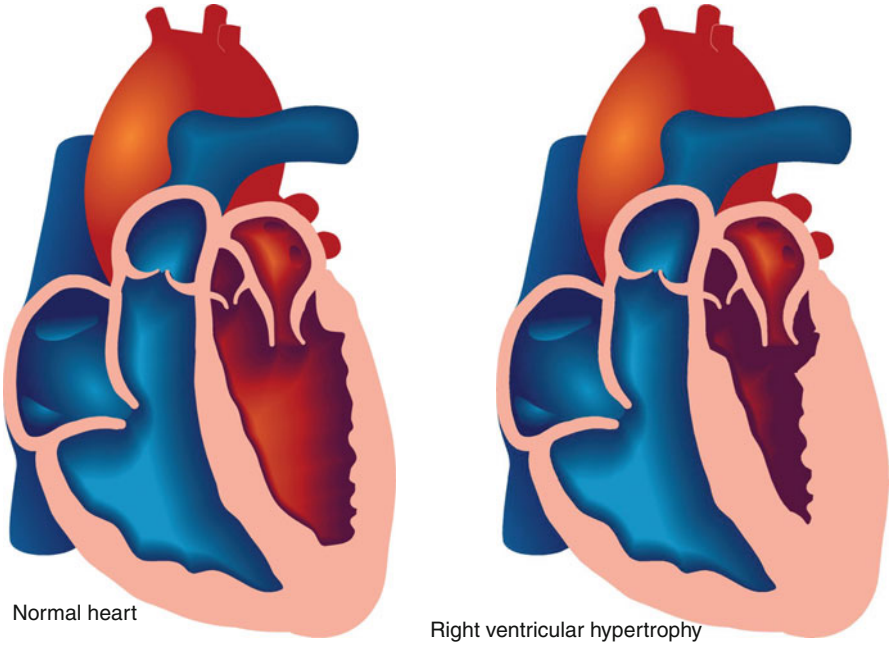


Fig. 5.12 A normal heart (*left*) and a heart with RVH (*right*)

RVH Key Points
(not all of these may be present)

- Right axis deviation
- Tall R waves in lead V_1
- R wave in V_1 + S wave in $V_6 \geq 10$ mm in height
- Accompanied right atrial abnormality
- In severe cases associated ST segment depression and T wave inversion due to 'strain'.

Biventricular Hypertrophy

It is possible to have enlargement of both ventricles, termed biventricular hypertrophy. This can be difficult to spot as the two may 'cancel' each other out to some degree, and the ECG may appear normal. Biventricular hypertrophy may be present if the voltage criteria for LVH is present in limb leads, with tall R waves in lead V_1 (Fig. 5.14).

Normal Variants

When the voltage criterion for LVH exists in adults under the age of 40, this may be a normal variant and not a pathological change. Tall R waves may be found as a normal variant in:

- The young
- Athletes
- Tall and thin individuals

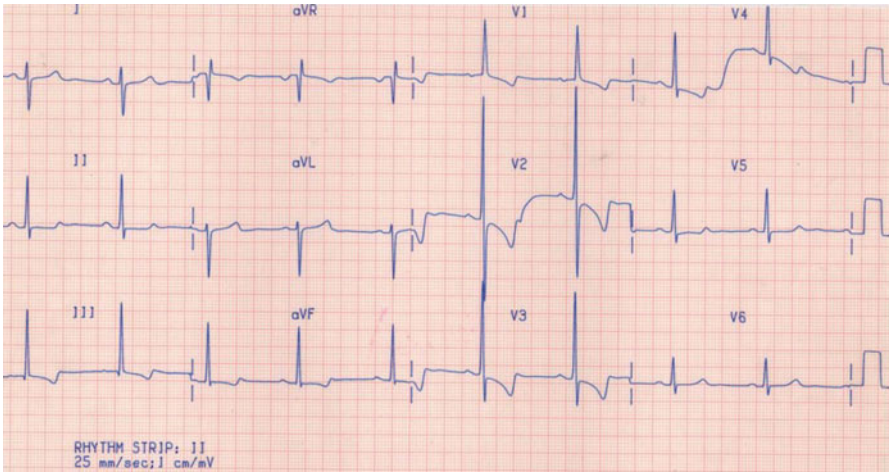


Fig. 5.13 RVH with associated ST-T wave abnormalities (strain), and right axis deviation. ECG taken from a 32 year old female with congenital pulmonary stenosis

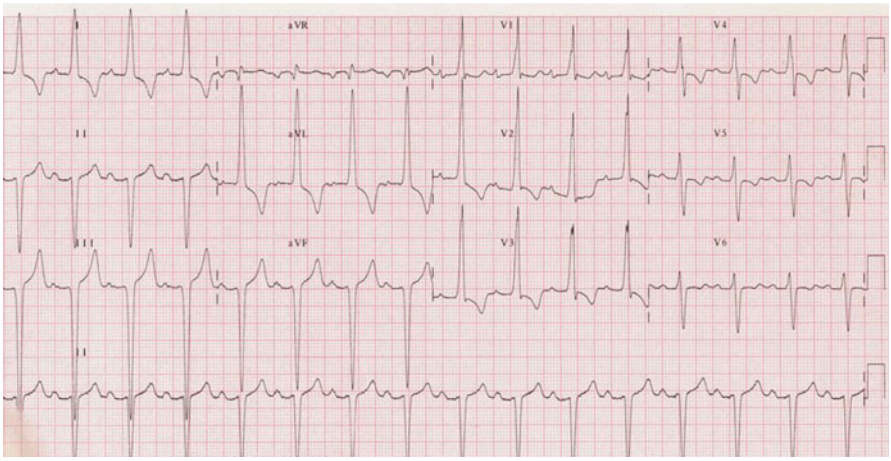


Fig. 5.14 Biventricular hypertrophy. Voltage criteria for LVH found in frontal plane with tall R waves in lead V1

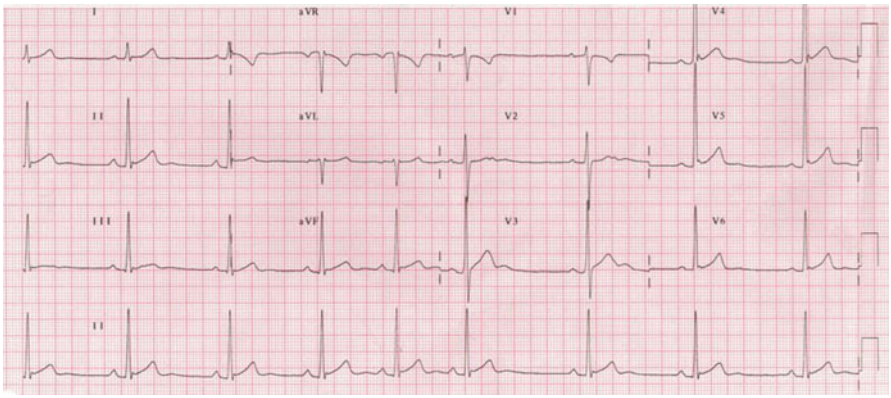


Fig. 5.15 'Athletes heart', physiological LVH

Other Causes of Tall R Waves
- Incorrect ECG machine calibration
- Normal variant
- Right bundle branch block (RBBB)
- Ventricular rhythms originating in the left ventricle
- Posterior Myocardial Infarction (MI)
- Wolff-Parkinson-White syndrome (WPW)
- Dextrocardia

Figure 5.15 demonstrates the appearance of physiological LVH or 'Athletes heart' as it is sometimes known. Voltage criteria for LVH exists, there are no ST segment or T wave changes or coexistent pathology present.

Summary of Key Points

- Echocardiography remains the gold standard for proving the presence of a chamber enlargement.
- Of the chamber abnormalities discussed, LVH is the most commonly encountered in clinical practice.
- Left and right atrial enlargement is often found in the presence of an associated ventricular enlargement or hypertrophy.
- Left Ventricular Hypertrophy may be due to a physiological cause in young, fit or tall and thin individuals.
- Other causes for tall R waves should always be considered.
- Always rule out the obvious first and check the machine is calibrated correctly to avoid an incorrect diagnosis.

Quiz

Q1. Left atrial abnormality is also sometimes known as?

(A) *P. pulmonale*
(B) *P. mitrale*
(C) *P. pulmanade*

Q2. Any enlargement of the atria is always caused by hypertrophy

(A) *True*
(B) *False*

Q3. Voltage criteria for Left Ventricular Hypertrophy is found by…

(A) *Adding S wave in V1 to the R wave in V6*
(B) *Adding R wave in V1 to the S wave in V6*
(C) *Adding Q wave in V1 to the R wave in V6*

Q4. The voltage criteria for Left Ventricular Hypertrophy in the precordial leads is said to be…

(A) *≥20 mm*
(B) *≥35 mm*
(C) *<30 mm*

Q5. Large R waves may be a normal variant in…

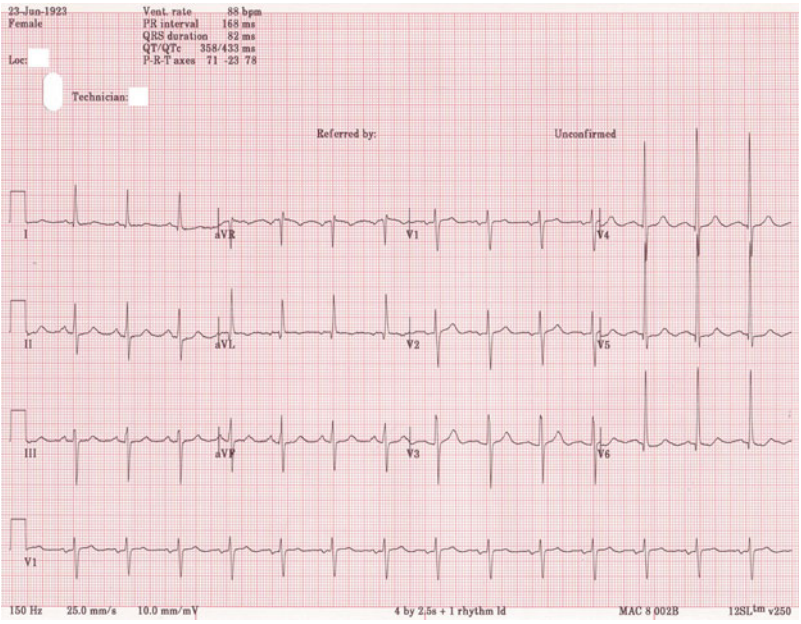
(A) *People of an Afro-Caribbean origin*
(B) *Babies*
(C) *Tall and thin individuals*

Q6. Right Atrial Enlargement often presents as…

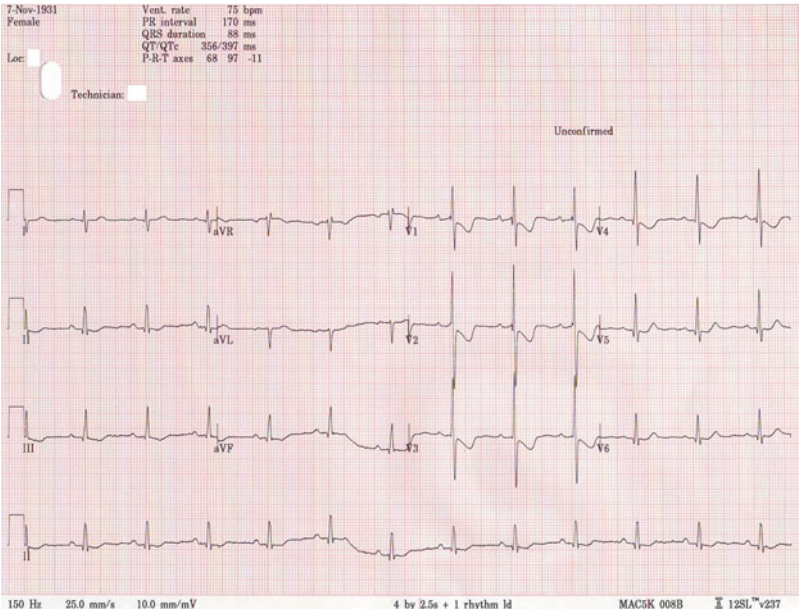
(A) *Tall, peaked P waves >1 mm in amplitude*
(B) *Tall, peaked P waves >2.5 mm in amplitude*
(C) *Tall, tented T waves >2.5 mm in amplitude*

Try to interpret the following ECGs.

Q7.



Q8.



Answers: Q1 = B, Q2 = B, Q3 = A, Q4 = B, Q5 = C, Q6 = B, Q7 = LVH, Q8 = RVH with strain pattern

Chapter 6
Arrhythmias

Abstract An arrhythmia occurs as a result of abnormal electrical conduction and genesis of an abnormal electrical current. There are various types of arrhythmias that can be classified in terms of their origin as, atrial, junctional or ventricular arrhythmias. Many arrhythmias are initiated by ectopic foci.

Keywords Arrhythmias • Flutter • Fibrillation • Arrest rhythms • Reentry • Ectopic's

Background

An arrhythmia occurs as a result of abnormal electrical conduction and genesis of an abnormal electrical current. There are various types of arrhythmias that can be classified in terms of their origin as, atrial, junctional or ventricular arrhythmias. Many arrhythmias are initiated by ectopic foci.

Ectopic Beats

Otherwise known as extrasystoles or premature beats, ectopics are caused by an intracellular build-up of positive ions, (usually calcium) that can trigger an impulse causing depolarisation that does not originate from the SAN. The other principle mechanism is altered automaticity. This includes increased/enhanced automaticity, where another part of the conduction system below the SAN takes over as the dominant pacemaker temporarily (as discussed in Chap. 1). In contrast abnormal

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automaticity may also cause an ectopic beat, which involves the abnormal spontaneous depolarisation of myocardial cells. The latter is often caused by an electrolyte imbalance (see Chap. 9). Ectopics can better be described as premature beats and can occur as, atrial, junctional or ventricular premature beats, depending on where in the heart they originate from.

Ectopic Foci
An electrical trigger initiating a premature beat.

Compensatory and Non-compensatory Pauses

When premature beats occur they are usually immediately followed by a pause. These pauses are compensatory pauses and allow the tissues to recover before the next beat. Pauses can be complete compensatory pauses (Fig. 6.1) as with premature ventricular beats or incomplete pauses, as seen in premature atrial beats.

Atrial Premature Beats

Can arise from either single, or multiple ectopic foci. On the ECG they can be recognised as they occur earlier than the next expected P wave and differ in morphology to the normal P waves (Fig. 6.2). Not all of these premature beats are always conducted resulting in ventricular activation and a subsequent QRS complex. This is due to the tissue being refractory and insufficiently recovered to conduct another impulse.

Junctional Premature Beats

Originate from the His bundle or the AV junction. The impulse may activate the ventricles via the normal pathway. Alternatively the atria may also be activated by retrograde conduction. This is where the impulse travels back up towards the atria instead of down towards the ventricles activating the atria. Junctional premature beats can be seen on the ECG as they occur before the next normal sinus beat. Often there is no P wave present if the atria and ventricles are depolarised simultaneously, as this is masked by the QRS complex (Fig. 6.3). Alternatively the P wave may be inverted (Fig. 6.4) and can occur either before, or after the QRS complex (Fig. 6.5).

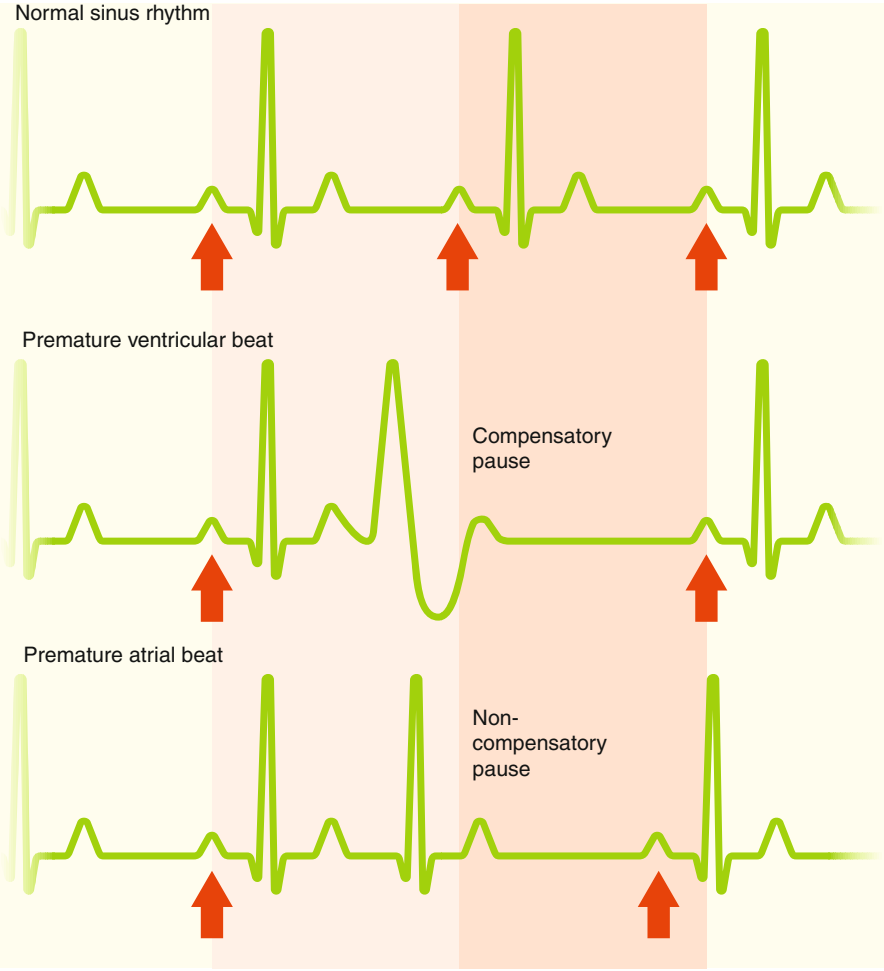


Fig. 6.1 Compensatory vs. non-compensatory pauses

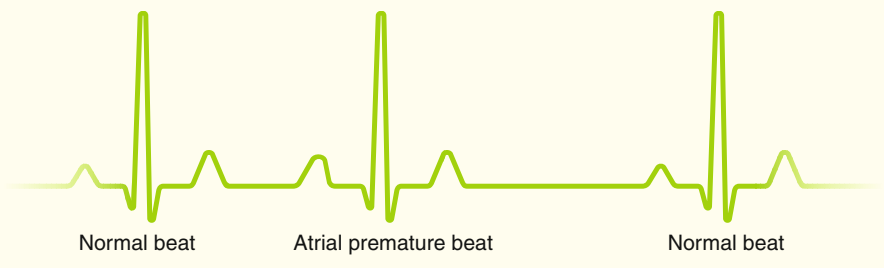


Fig. 6.2 An atrial premature beat

Fig. 6.3 Hidden P wave masked by QRS complex

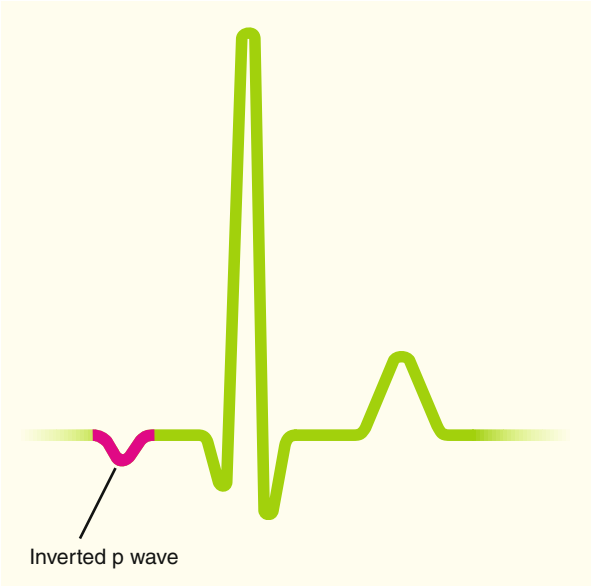


Fig. 6.4 An inverted P wave

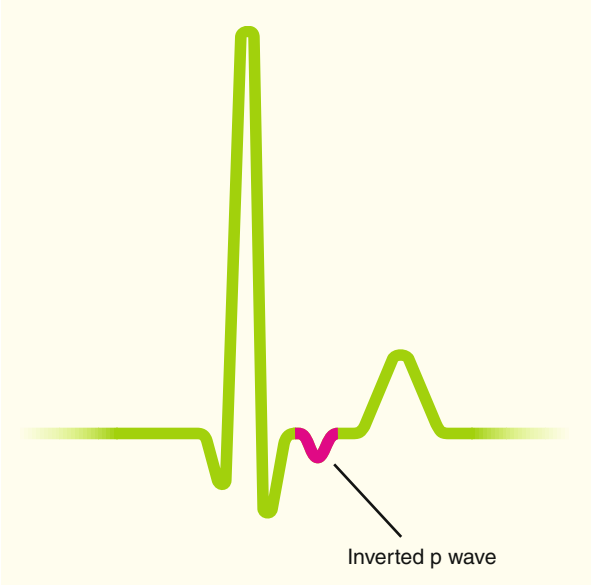


Fig. 6.5 Inverted P wave after QRS complex

Ventricular Premature Beats

Premature beats can also originate from the ventricles. Ventricular premature beats look different to the normal QRS complex (have a different morphology). The QRS of the premature beat is usually much wider than the intrinsic rhythm; it also usually has no preceding P wave. Figure 6.6 shows a normal beat compared with the ventricular premature beat in Fig. 6.7, which demonstrates the difference in morphology.

The atria can also be activated retrogradely and sometimes P waves can appear inverted (upside down) merged with the ectopic T wave. The T waves of the ventricular premature beats are usually defected in the opposite direction to the intrinsic rhythm, and also appear odd in appearance.

Pathological Ventricular Premature Beats

Most ventricular premature beats are benign and require no intervention. There are however a small minority of potentially dangerous patterns of premature beat that

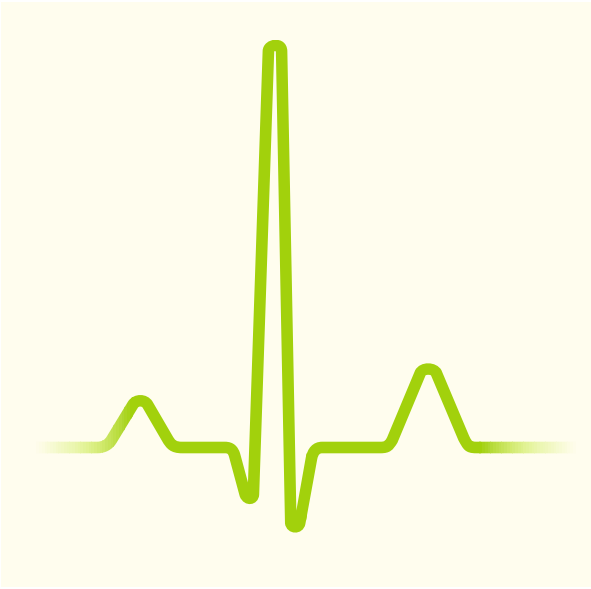


Fig. 6.6 A normal beat

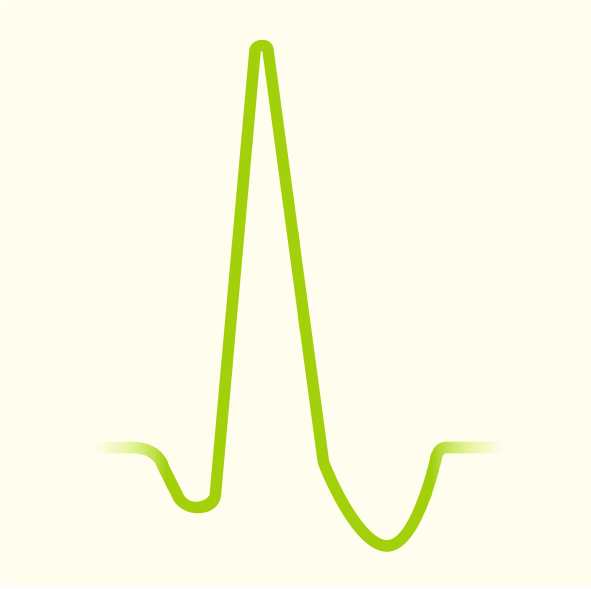


Fig. 6.7 A premature ventricular beat

can lead to problems. Premature beats reduce cardiac output, so multiple premature beats can lead to haemodynamic problems. Some of the more dangerous patterns of ventricular premature beat can predispose people to dangerous arrhythmias such as ventricular fibrillation or ventricular tachycardia. These more dangerous patterns include:

1. Couplets (Fig. 6.8)
2. **Triplets** (Fig. 6.9)
3. **Salvos** (Fig. 6.10)

Multiple Focus Ventricular Premature Beats

These are ventricular premature beats that differ in morphology from each other (Fig. 6.11). This is because they often originate from different areas of the ventricle. The presence of these multiform ventricular premature beats can point toward the presence of serious heart disease.

Fig. 6.8 Two ventricular premature beats occurring together

Fig. 6.9 Three ventricular premature beats occurring together

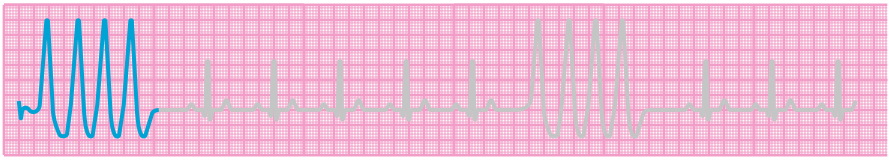


Fig. 6.10 Four ventricular premature beats occurring together

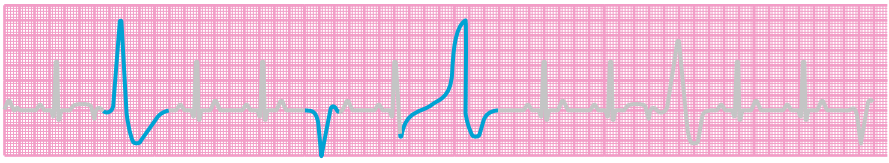


Fig. 6.11 Multiform ventricular premature beats

R on T Phenomenon

Represents a serious threat of subsequent ventricular fibrillation (VF) or tachycardia (VT). This is because the premature beat occurs during the T wave of the previous beat. As the cells are still recovering from depolarisation and not yet fully repolarised. Another potential complication of R on T phenomenon is 'torsade de pointes' (twisting about a point), a form of polymorphic VT so named due its spindle like appearance (Fig. 6.12).

Bigeminy and Trigemany

These conditions consist of regular patterns of premature beats. And can occur in either the atria or ventricles. Atrial bigeminy consists of an atrial premature beat after each sinus beat (Fig. 6.13). Atrial trigeminy involves an atrial premature beat after every second sinus beat.

Ventricular bigeminy in contrast consists of alternating normal (intrinsic) QRS complexes followed by a regular repeating ventricular premature beat (Fig. 6.14). Ventricular trigeminy like atrial trigeminy occurs after every second QRS complex

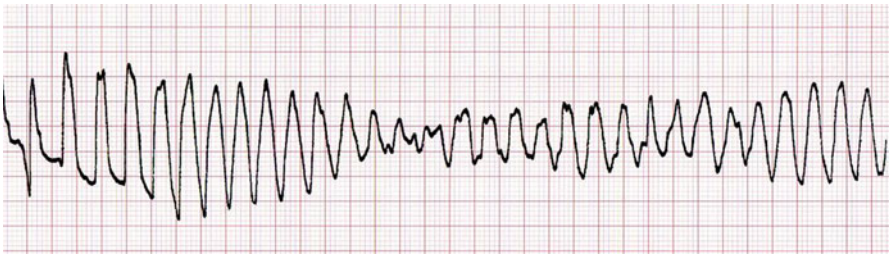


Fig. 6.12 Torsade de pointes

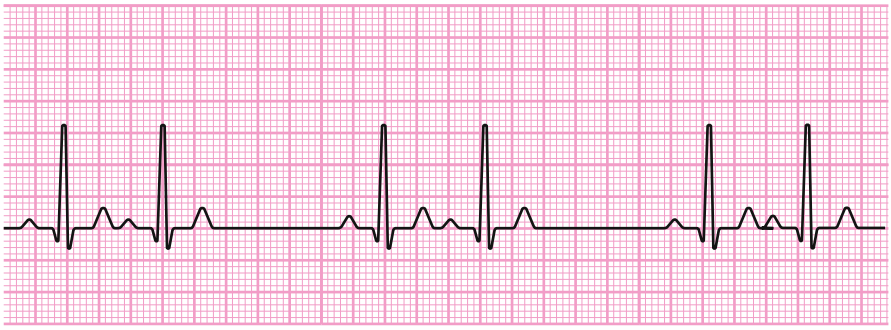


Fig. 6.13 Atrial bigeminy

Fig. 6.14 Ventricular bigeminy

Fig. 6.15 Ventricular trigeminy

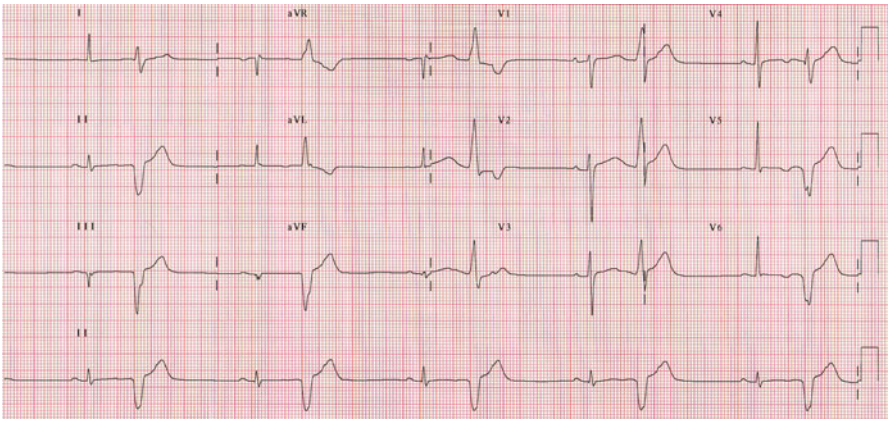


Fig. 6.16 Ventricular bigeminy

(Fig. 6.15). Ventricular bigeminy/trigeminy can predispose individuals to more dangerous arrhythmias such as ventricular fibrillation/tachycardia (VF/VT) due to increased ventricular irritability (Fig. 6.16).

Escape Beats

When there is a failure of the SAN to produce an impulse, another part of the conduction system will usually take over as the pacemaker, and trigger an 'escape beat'. This is the hearts safeguard system. Most of these escape beats originate in the AV junction, although they can also be generated even lower down in the ventricles if the AV junction fails to produce an impulse. Escape beats can look at first glance similar to an ectopic beat, as they too can look different in morphology, and have no preceding P wave. There is an easy way to tell them apart however: Premature beats occur early (Fig. 6.17), whereas escape beats occur late (Fig. 6.18).

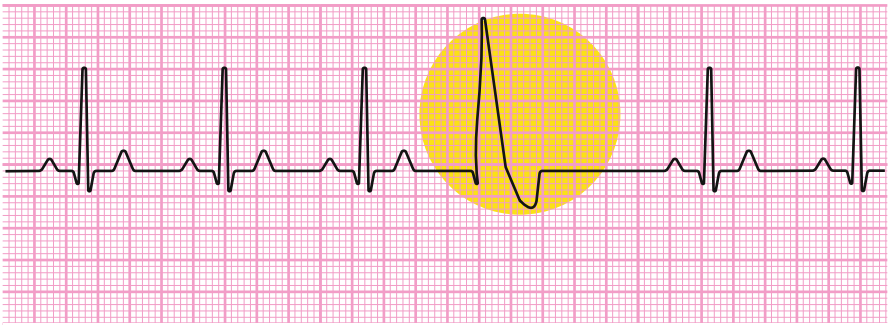


Fig. 6.17 Premature beat

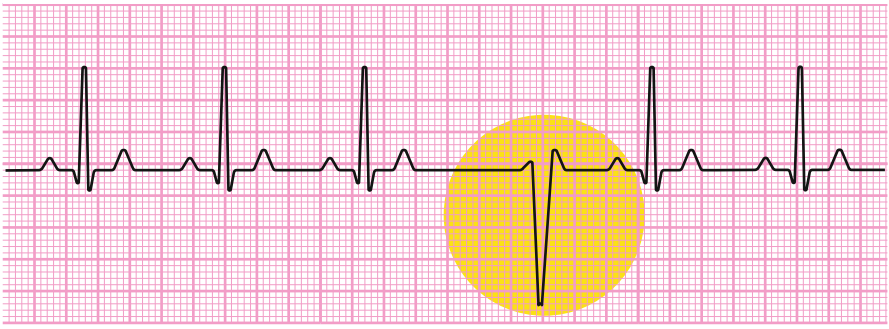


Fig. 6.18 Escape beat

Fig. 6.19 Atrial tachycardia as seen in lead V1

Atrial Arrhythmias

Atrial Tachycardia

Is characterized by the presence of three or more atrial premature beats, and an atrial rate of between 140 and 250 BPM (Fig. 6.19). P waves, best seen in leads II or V_1 are sometime present and usually hidden in the preceding T waves. The rhythm is regular. Atrial Tachycardia can also cause loss of 'atrial kick' and a reduced cardiac output if sustained.

Causes of Atrial Tachycardia
- Caffeine and other stimulants
- Physical/emotional stress
- Hypoxia
- Electrolyte imbalance
- Cardiomyopathy
- MI

Atrial Kick
The contribution of atrial contraction before ventricular systole that increases ventricular ejection efficiency.

Atrial Fibrillation

Is the most commonly encountered arrhythmia. Atrial Fibrillation (AF) increases in incidence with age. It is caused by multiple electrical triggers (multiple ectopic foci) occurring in the atria (Fig. 6.20), most commonly occurring around the region of the pulmonary veins.

As a result of these multiple triggers the atrial rate becomes very high, up to 600 BPM. Fortunately, not all of these impulses are conducted to the ventricles as this would be haemodynamically catastrophic. A ventricular rate below 100 BPM is termed 'controlled AF', whereas a ventricular response of more than 100 BPM is termed 'uncontrolled' or 'fast' AF, which may cause damaging haemodynamic changes.

The high atrial rate causes the atria to fibrillate rapidly; this in turn means the atria cannot fill adequately before emptying. This subsequently results in a loss of 'atrial kick' and a reduction in cardiac output. There is also the risk of clot formation as the atria do not empty adequately. Potential clots could migrate to other parts of the body with serious consequences, such as stroke.

The ECG characteristics of AF are best seen in the rhythm strip on the ECG (usually lead II or V_1) (Figs. 6.21 and 6.22). The rhythm is irregularly irregular; this is because there is no repeated pattern to the irregularity.

The other primary feature of AF is the complete absence of P waves and the presence of a chaotic baseline consisting of fibrillatory or 'f' waves (Fig. 6.21). There are three main types of Atrial Fibrillation, comprised of:

- Paroxysmal Atrial Fibrillation
  Terminates spontaneously, usually in less than 7 days
- Persistent Atrial Fibrillation
  Does not terminate spontaneously and lasts longer than 7 days
- Permanent Atrial Fibrillation
  Not terminated or reverted.

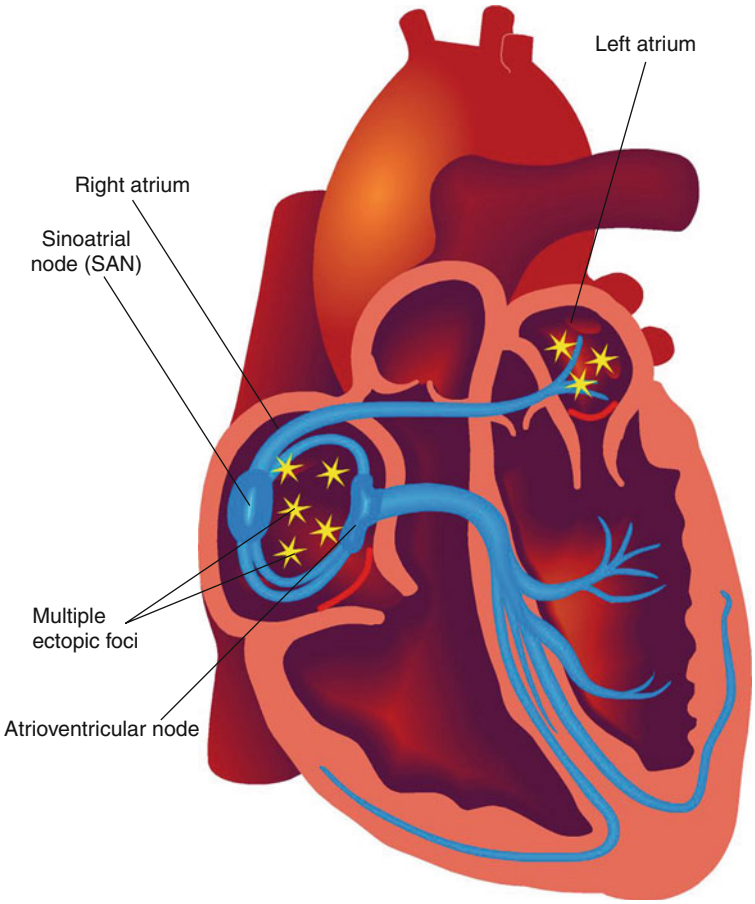


Fig. 6.20 Multiple atrial ectopic foci

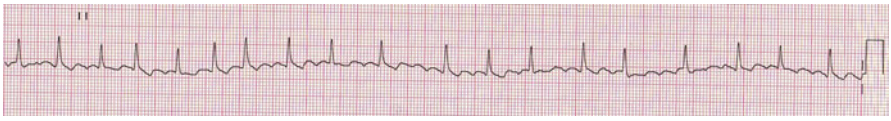


Fig. 6.21 Atrial Fibrillation as seen in lead II

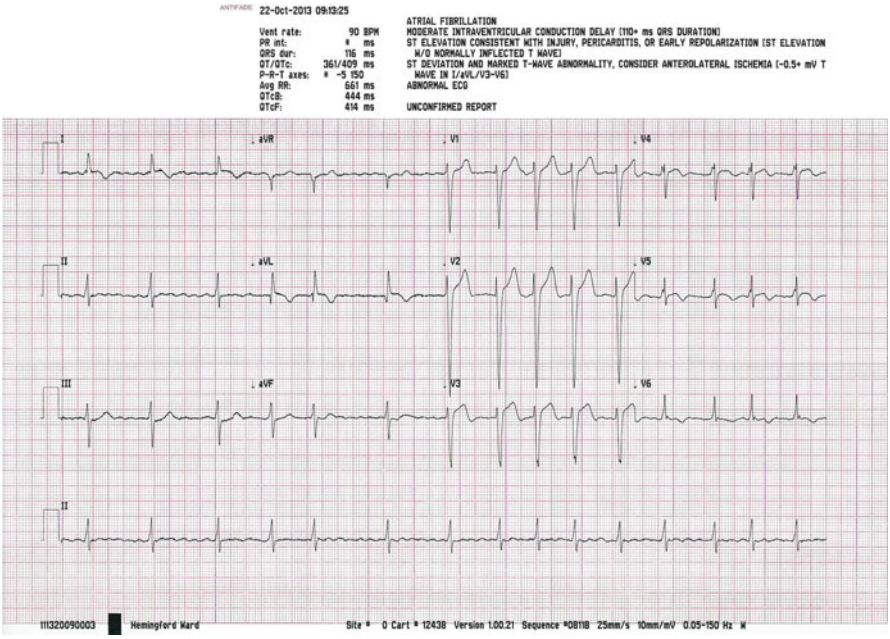


Fig. 6.22 12 lead ECG showing Atrial Fibrillation

If the arrhythmia persists, the initial electrical remodelling of the atria is followed by structural remodelling, which helps to maintain the arrhythmia.

Atrial Fibrillation

Symptoms

- Palpitations
- Chest pain
- Fatigue
- Dyspnoea
- Dizziness
- Loss of consciousness

Causes

- Cardiac surgery
- Hypertension
- Sick Sinus Syndrome (SSS)
- Hyperthyroidism
- Rheumatic valve disease
- Alcohol
- Ischemic heart disease
- Pre-excitation syndromes
- Acute infection
- Pulmonary Embolism (PE)
- Pleural effusion
- Atrial Septal Defect (ASD)
- Cardiomyopathy

Atrial Flutter

Atrial Flutter is rhythm disturbance associated with structural heart disease. Atrial Flutter is a form of atrial tachycardia that usually originates in the right atria (Fig. 6.23) and is usually paroxysmal, lasting several hours or days. Longer lasting or chronic flutter is rare. Atrial Flutter and Atrial Fibrillation are closely linked and can alternate in individuals. A prolonged Atrial Flutter often converts itself into Atrial Fibrillation. Flutter can be identified on the ECG by its classic 'sawtooth' like appearance, seen best in lead II, III and aVF. As with Atrial Fibrillation there are no P waves, but instead prominent Flutter 'F' waves (Fig. 6.24).

Multifocal Atrial Tachycardia (MAT)

Caused by multiple pacemakers outside the sinoatrial node. MAT can be identified on an ECG due to an irregular rhythm that can at first glance appear similar to AF. The principle difference is that there are P waves in MAT. Due to the different origin of the pacemaker sites the P waves differ in morphology and there are usually three or more distinct different morphologies of P wave present. The ventricular rate is also above 100 BPM. MAT is a rare arrhythmia, occurring normally in very ill individuals with severe infection. It can also be caused by hypoxia, severe pulmonary disease, atrial dysfunction or ischemic heart disease.

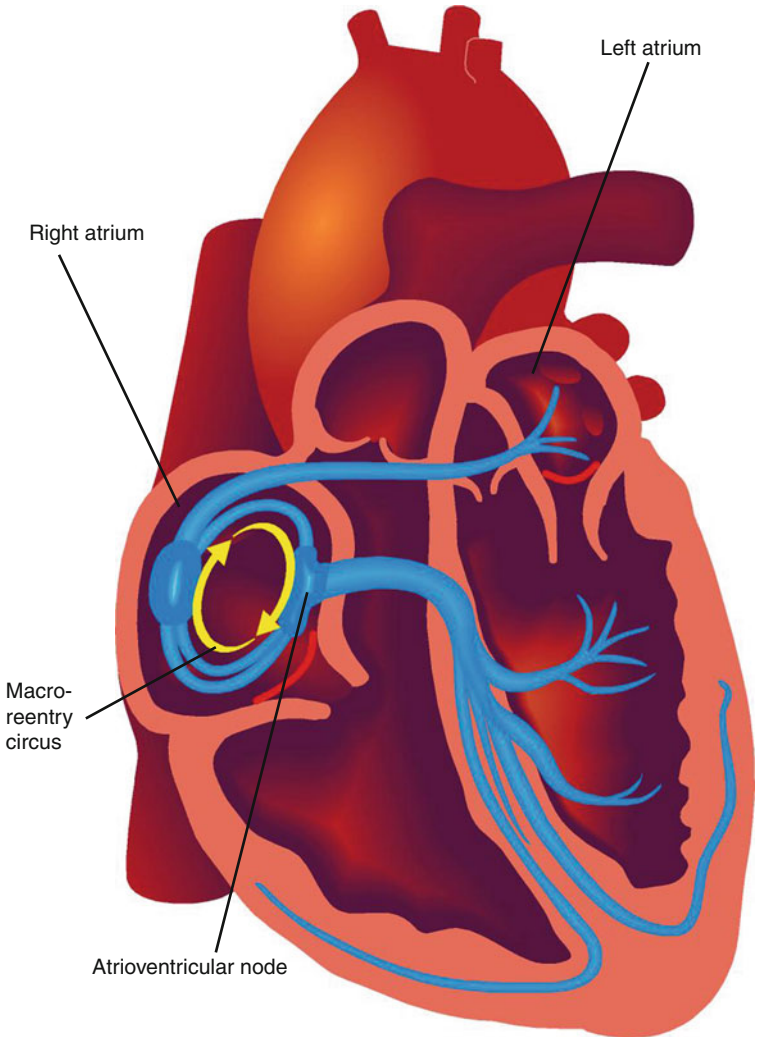


Fig. 6.23 The macro-reentry circus of atrial flutter

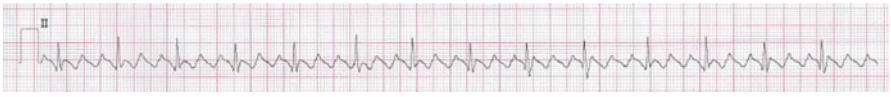


Fig. 6.24 Atrial Flutter, as seen in lead II

Wandering Atrial Pacemaker (WAP)

This is essentially the same as Multifocal Atrial Tachycardia but with a rate below 100 BPM.

Junctional Arrhythmias

Junctional Escape Rhythm

If the sinoatrial node fails to generate an impulse, the AV junction may take over and initiate an 'escape' beat, as mentioned earlier. These escape beats are the hearts back up system to keep the heart functioning (Fig. 6.25).

Junctional Tachycardia

A tachycardia made up of three or more Premature Junctional Beats. Junctional Tachycardia is usually caused by the AV node taking over as the dominant pacemaker due to enhanced automaticity. As in junctional escape beats the atria are retrogradely depolarized, resulting in inverted P waves that can occur after the QRS complex.

AV Nodal Re-entry Tachycardia

This is caused by the presence of an extra pathway of connection between the atria and the ventricles. This means that impulses generated in the SAN have the potential to travel back up through the extra pathway and depolarise the SAN. This is known as retrograde conduction. This can create a loop, sustaining the tachycardia.

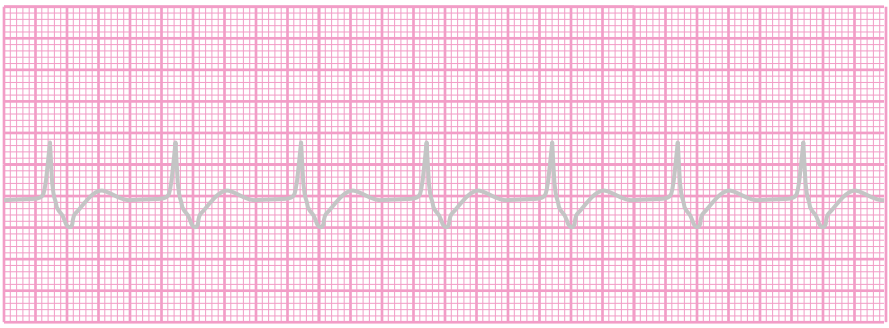


Fig. 6.25 Junctional rhythm

Wolff-Parkinson-White Syndrome WPW

Named after the discoverers of the syndrome, WPW is caused by a foetal abnormality resulting in an additional pathway between the atria and the ventricles. As discussed earlier the atria and ventricles are electrically isolated by the atrioventricular ring. Electrical impulses pass from atria to ventricle by the AV node and the His bundle into the ventricles. In WPW an extra 'accessory' pathway exists between the atria and ventricles (Fig. 6.26). This pathway is called the 'Bundle of Kent'. The electrical impulse can now travel through the AV node in the normal way and through the accessory pathway.

Because there is no AV node to slow the impulse in the accessory pathway the PR interval is short. The impulse is then slower to activate the ventricles as it is not passing through the normal conduction system. This leads to the formation of a 'delta wave' (Fig. 6.27) on the ECG. The short PR interval and delta wave are the key ECG features of this syndrome (Fig. 6.28).

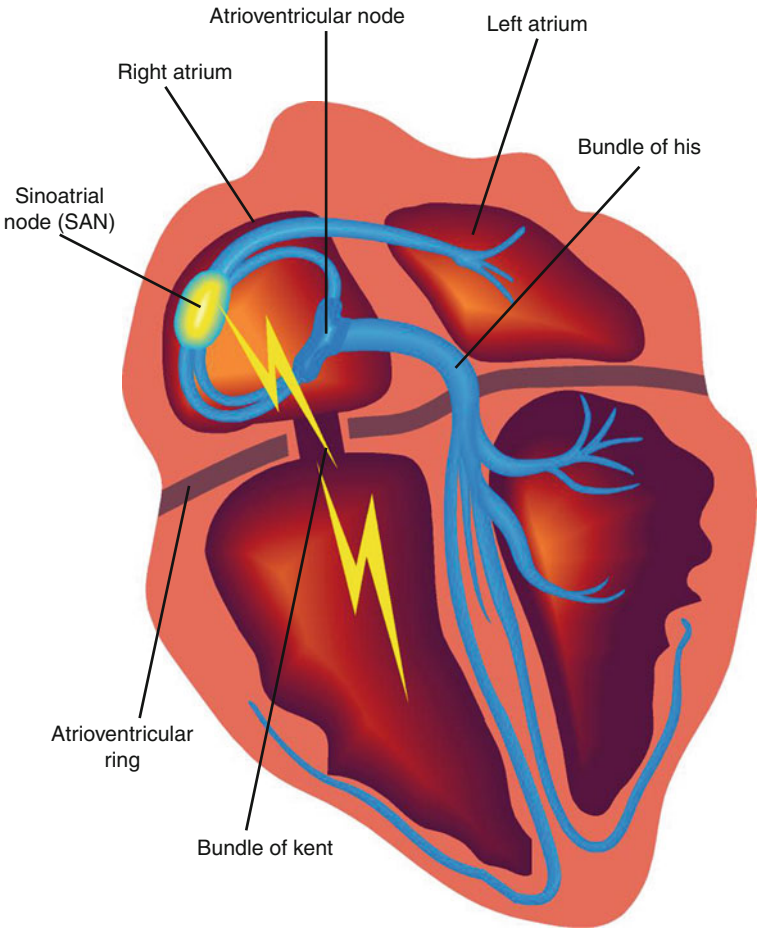


Fig. 6.26 WPW accessory pathway

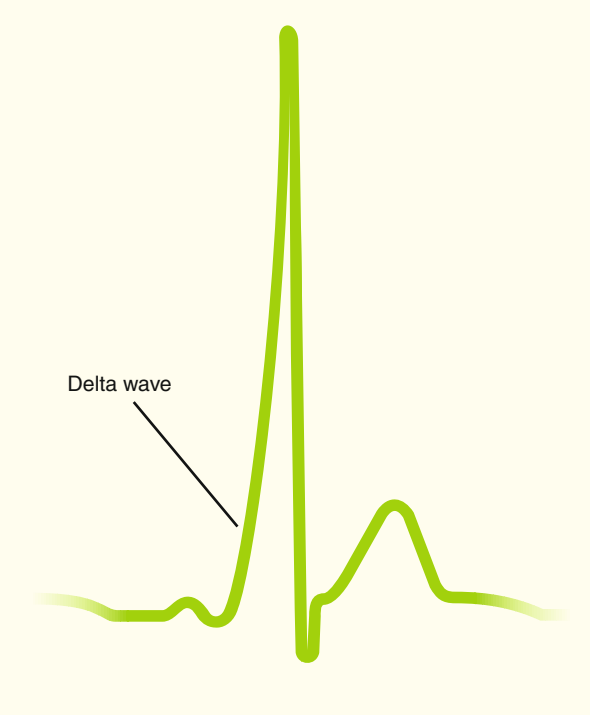


Fig. 6.27 Delta wave and short PR interval

The impulse can also travel back up to the atria via the accessory pathway and create a sustained tachycardia. WPW normally occurs in the young and adults aged 20–35 years of age. Most people with this condition seek medical attention due to the frequent occurrence of Supra Ventricular tachyarrhythmias.

WPW Type A and B
- There are two types of WPW, type A and B.
- Type A – QRS complexes are positively deflected in V1
- Type B – QRS complexes are negatively deflected in V1

Lown-Ganong-Levine Syndrome (LGL)

Also named after the people who discovered the syndrome. LGL differs from WPW in that the atria are connected to the ventricles below the AV node by 'James fibres'. The impulse bypasses the AV node and is therefore not delayed causing a short PR interval like WPW (Fig. 6.29). There is however no delta wave in LGL. This is because the ventricles are depolarized normally. The prominent R waves and short PR interval are the main ECG characteristics of this syndrome. The syndrome is also more common in women than men.

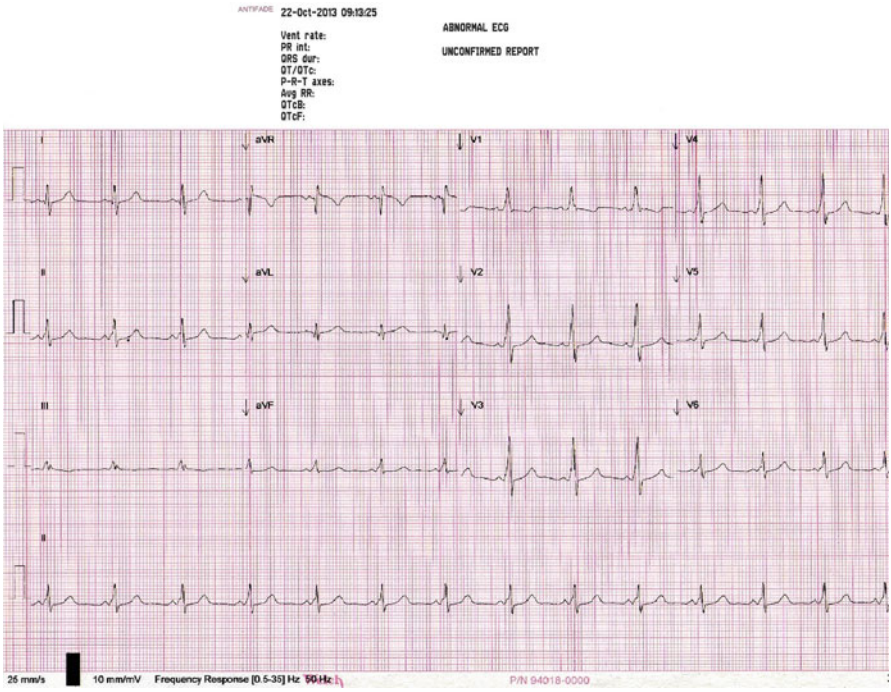


Fig. 6.28 WPW syndrome type A

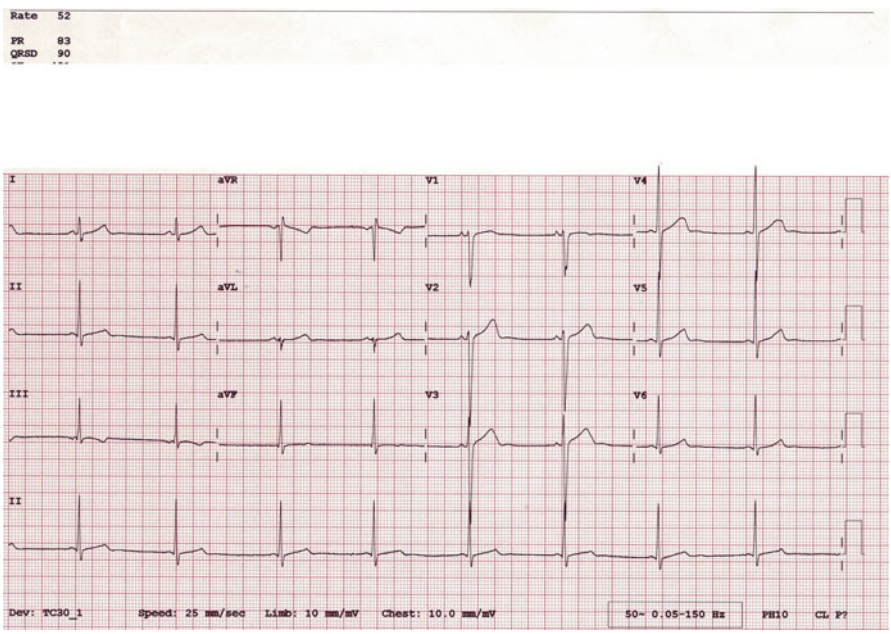


Fig. 6.29 LGL syndrome

Re-entry

Is caused by an electrical impulse returning to depolarise previously depolarised tissue a second time. There are different mechanisms that make this possible.

For re-entry to occur there must be a pathway for the impulse to take that allows the impulse to 'double back' on itself allowing it the depolarise tissue a second time. Re-entry is often triggered by a premature beat. The impulse would normally travel through tissue equally, passing through both limbs of the pathway at the same time. In some cases where there is ischemic damage to the heart one of the limbs can take longer to recover than the other. This recovery phase is known as refractoriness. Whilst tissue is refractory an impulse cannot travel through the tissue. By the time the impulse travels down the fast limb of the pathway the slow limb then recovers in time for the impulse to travel back up the slow pathway and depolarise the previously depolarised tissue. This can create a sustained loop, leading to a tachycardia.

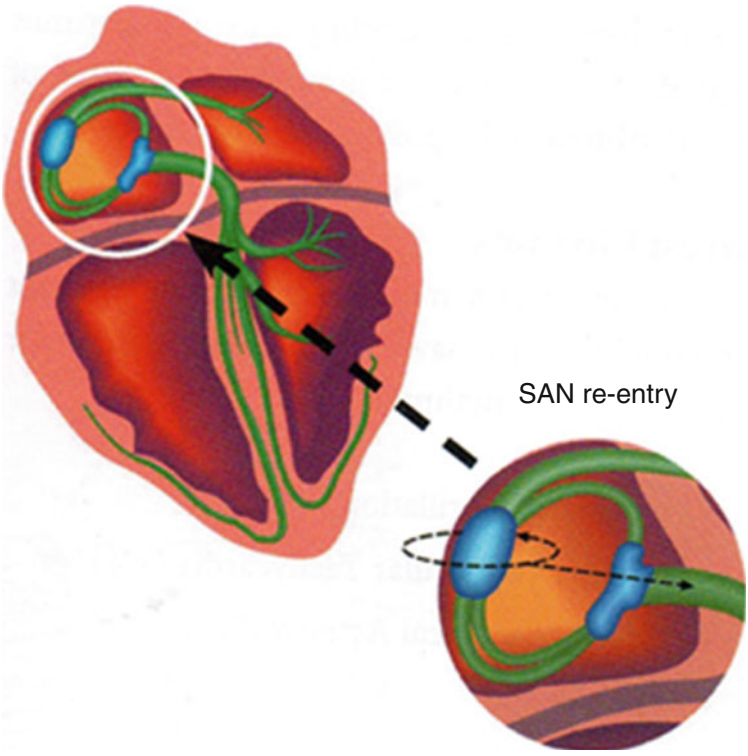


Fig. 6.30 SAN re-entry system

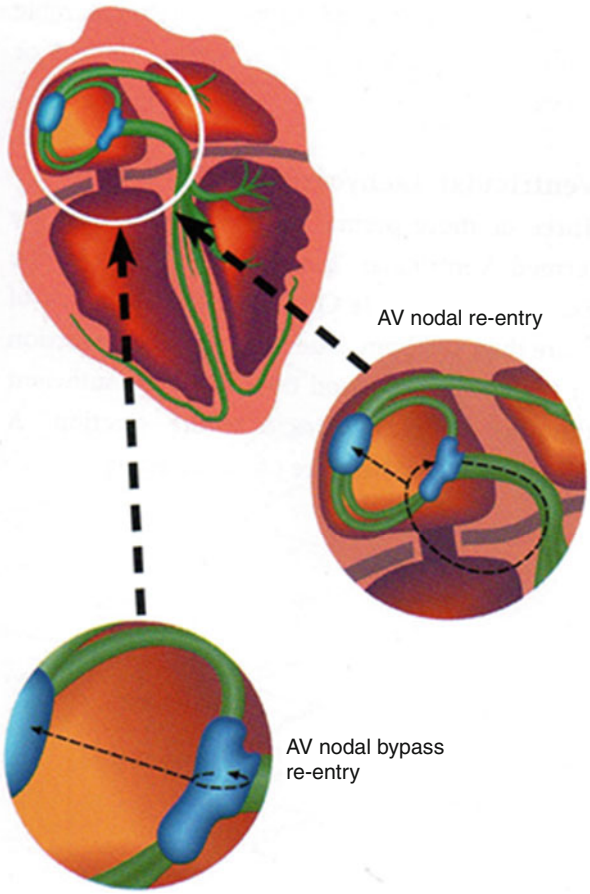
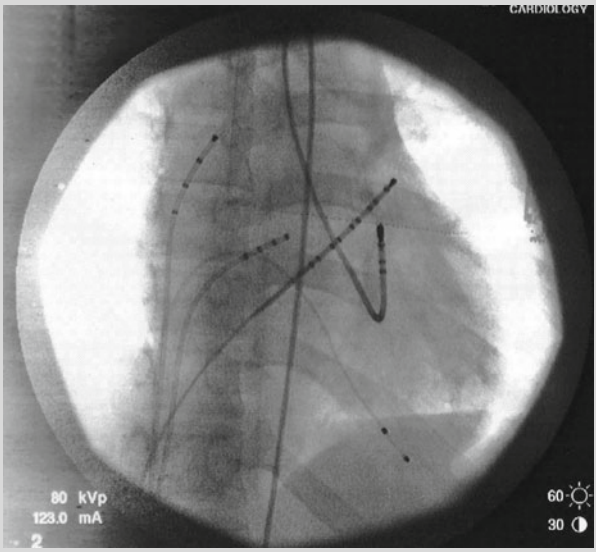


Fig. 6.31 AV nodal re-entry and AV nodal bypass re-entry systems

These slow and fast pathways can exist in different places, such as the SAN, this leads to what is termed SAN re-entry tachycardia (Fig. 6.30). When the pathway is in the AV node, this is termed AV nodal re-entry tachycardia (AVNRT). This form of re-entry tachycardia is the most common (Fig. 6.31) and presents on the ECG with a regular rhythm, fast rate usually above 130 BPM. There may also be retrograde P waves present as the SAN is triggered from below.

The other mechanism for re-entry is an extra pathway that bypasses the AV node entirely allowing impulses to go back up through this pathway creating a sustained loop (Fig. 6.31). People are sometimes born with these extra pathways, in conditions such as WPW (Wolfe Parkinson White syndrome).



Electrophysiology Studies
Catheters are passed into the heart via the veins or arteries in the groin. These catheters have sensors on them allowing them to pick up a more detailed electrical trace than the surface ECG. This can be used to determine the origin of an Arrhythmia. Often the arrhythmia causing cells can be destroyed by ablation. This is the heating or cooling of cells to destroy them.

Ventricular Arrhythmias

Electrophysiological studies are sometimes carried out to investigate the cause and origin of an arrhythmia, and if possible to treat the cause.

Arrest Rhythms

Cardiac arrest is a medical emergency. Prompt intervention can save lives. There are four principle arrest rhythms:

- Ventricular Fibrillation (VF)
- Pulseless Ventricular Tachycardia (VT)
- Pulseless Electrical Activity (PEA)
- Asystole

Of the four only VF and VT are rhythms that can be treated by defibrillation (passing an electrical current through the heart to reinitialise it ↯). Defibrillation should only be attempted by a trained professional. The survival outcomes of Cardiac arrest tend to be more favourable if the rhythm is VT or VF as oppose to PEA or asystole.

Ventricular Tachycardia (VT)

Three or more premature ventricular beats are termed Ventricular Tachycardia (VT) (Fig. 6.32). On the ECG there is a wide QRS complex and a rate of more than 100 BPM. Due to the rapid contraction of the ventricles, blood does not have sufficient time to fill the ventricle before ejection. A sustained VT is therefore life threatening.

It can be difficult to tell the difference between a Supra-Ventricular Tachycardia (SVT) and a Ventricular Tachycardia (VT). The wider the QRS complexes the more likely it is to be VT. Another good indicator is the presence of P waves that have no relationship with the QRS complex. VT that turns into VF is considered one of the main causes of sudden cardiac death. Treatment can include implantation of an Automated Internal.

Cardiac Defibrillator (AICD) that sits in the chest and monitors the heart rhythm. The device is capable of delivering a shock to the patient if it detects a dangerous rhythm that will respond to defibrillation.

Ventricular Flutter

Is a form of VT with a rate greater than 300 BPM and a 'sine wave' like QRS appearance (Fig. 6.33).

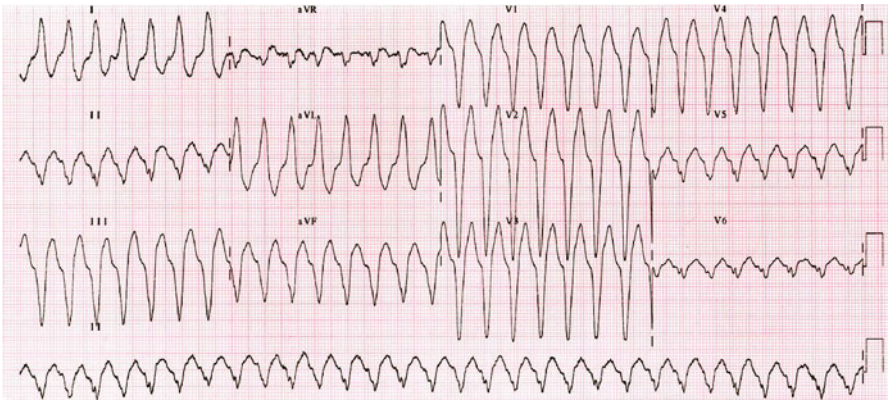


Fig. 6.32 VT

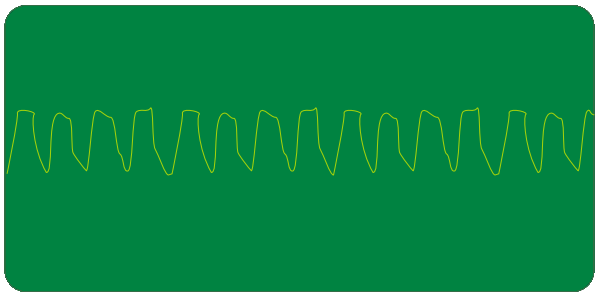


Fig. 6.33 Ventricular Flutter

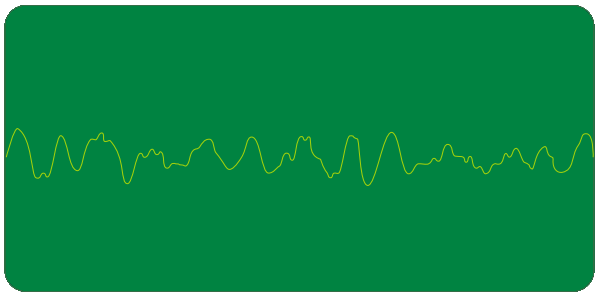


Fig. 6.34 Fine Ventricular Fibrillation

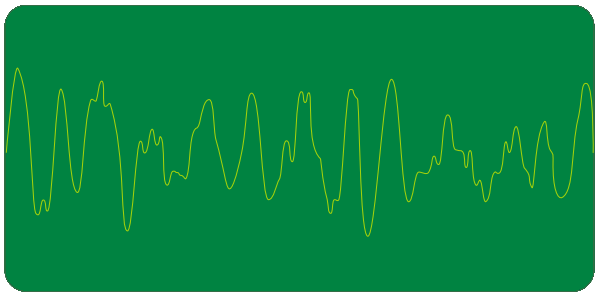


Fig. 6.35 Coarse Ventricular Fibrillation

Ventricular Fibrillation (VF)

VF is the most common initial rhythm during a cardiac arrest. The ventricles contract randomly giving rise to the chaotic irregular appearance on the ECG (Figs. 6.34 and 6.35).

VF is a shockable rhythm, early defibrillation saves lives. Sometimes movement artefact can resemble VF, if for example a patient on a cardiac monitor was to brush their teeth. It may sound obvious but it is important to always treat the patient and not the rhythm.

Defibrillation
An electrical current is passed through the myocardium to spontaneously depolarise the heart, allowing the natural pacemaker to take over control. There are three main types of external defibrillator:

- Monophasic: The older and less efficient type of defibrillator still in use in clinical areas. Requires a higher energy output setting of 360 J
- Biphasic: The newer and more efficient form of defibrillator. Requires an energy setting of 150–360 J
- AED's (Automated External Defibrillators): Interpret the rhythm for the user and advise with audio instructions. Often seen in public places and in non-cardiac specific clinical settings

Defibrillation should only be carried out by appropriately trained and assessed health care professionals.

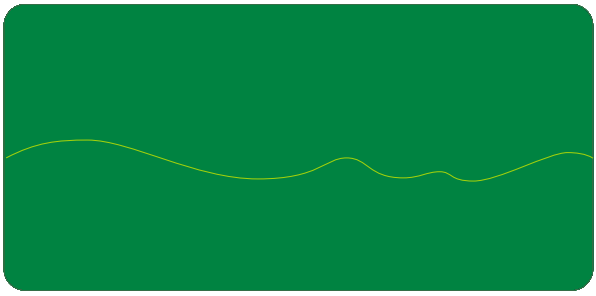


Fig. 6.36 Asystole

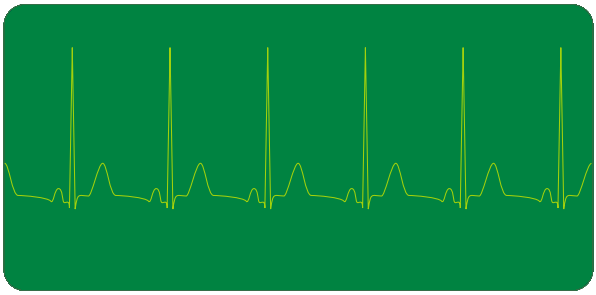
Asystole

Absence of any ventricular or atrial activity (Fig. 6.36). There is no cardiac output in this rhythm and unless reversible causes are present the outlook is extremely poor. Sometimes P waves may be present (termed P wave asystole), these patients may be suitable for pacing. It is also important to check the gain on the monitor as fine VF can sometimes be missed as it can look like asystole if not set properly (1 mV per cm). A complete flat line on a monitor is an indication that one of the monitoring leads is not connected properly.

Pulseless Electrical Activity

A rhythm that can look normal electrically but there is no cardiac output and the patient is in cardiac arrest (Fig. 6.37). In this instance the electrical activity in the heart is working but there is no mechanical action taking place. As with asystole there is a poor outcome. Treatment consists of correcting reversible causes and CPR.

Fig. 6.37 PEA



Reversible Causes
(The 4 h's and the 4 t's)
- Hypoxia
- Hypovolaemia
- Hypo/hyperkalaemia and other metabolic
- Hypothermia
- Tension pneumothorax
- Tamponade
- Toxins
- Thrombosis (either cardiac or pulmonary)

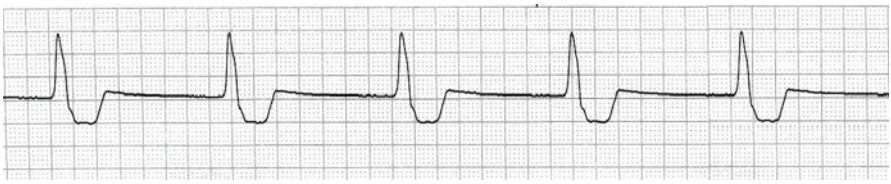


Fig. 6.38 Idioventricular rhythm

Idioventricular Rhythm

Is a reperfusion arrhythmia commonly seen after a Myocardial Infarction (MI). Idioventricular rhythm acts as a ventricular escape rhythm, with the dominant pacemaker originating from the ventricles. This rhythm protects the heart from asystole. It can be identified on the ECG by the wide strangely shaped QRS complexes with the absence of P waves (Fig. 6.38). The rate in Idioventricular rhythm is usually between 20 and 40 BPM. Idioventricular rhythm with a rate above this is termed accelerated Idioventricular rhythm.

Summary of Key Points

- It is important that health care professionals are able to identify and respond to arrhythmias appropriately. Including knowing which rhythms respond to defibrillation and which do not.
- Arrhythmias can be classified according to their point of origin within the heart.
- Premature ventricular beats are normally benign and very common. There are however a small subset of more dangerous patterns of premature beat that can lead to more serious arrhythmias.
- Atrial Fibrillation is the most commonly encountered arrhythmia
- Prompt CPR and defibrillation saves lives.

Quiz

Q1. There are no P waves in AF

(A) *True*
(B) *False*

Q2. Two Premature Ventricular Beats together are called…

(A) *Triplets*
(B) *VT*
(C) *Couplets*

Q3. Paroxysmal AF usually terminates in…

(A) *<1 day*
(B) *<7 days*
(C) *<12 days*

Q4. WPW is identified on the 12-lead ECG by…

(A) *A long PR interval and a delta wave*
(B) *A short PR interval and no delta wave*
(C) *A short PR interval and a delta wave*

Q5. The two 'shockable' rhythms are…

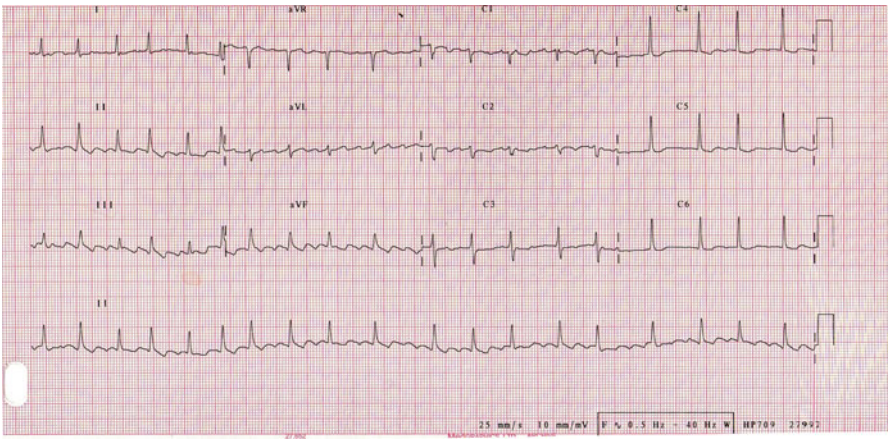
(A) *VF and VT*
(B) *VF and Asystole*
(C) *PEA and VT*

Q6. How many reversible causes are there for cardiac arrest?

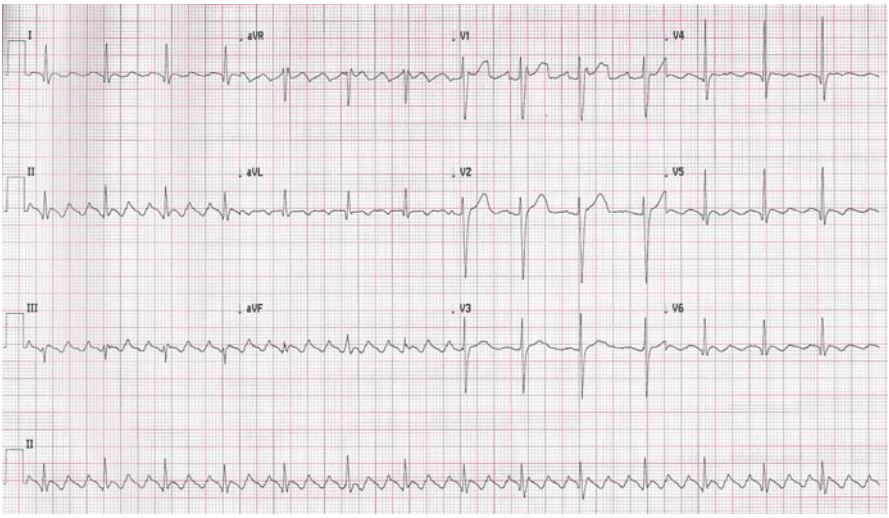
(A) *4*
(B) *10*
(C) *8*

Interpret the following ECGs:

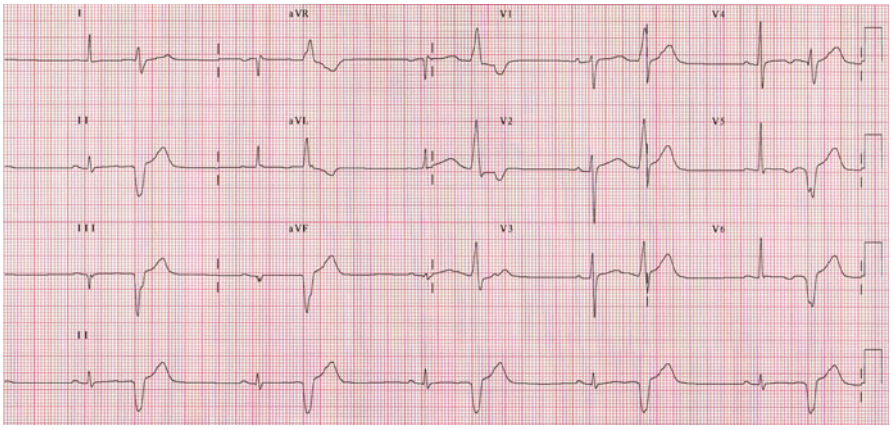
Q7.



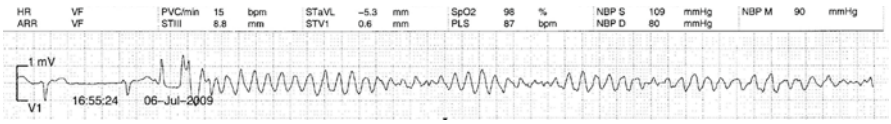
Q8.



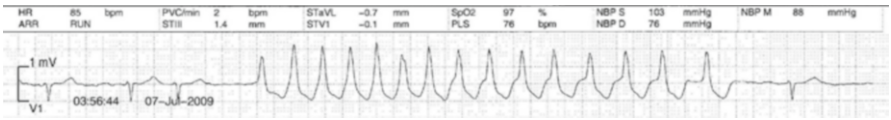
Q9.



Q10.



Q11.



Answers: Q1 = A, Q2 = C, Q3 = B, Q4 = C, Q5 = A, Q6 = C, Q7 = Atrial fibrillation, Q8 = Atrial Flutter, Q9 = Ventricular bigeminy, Q10 = Ventricular fibrillation, Q11 = Ventricular tachycardia.

Chapter 7
Conduction Blocks

Abstract There are various types of conduction block, all of which affect different sections of the conduction system. The 12-lead ECG helps to identify the location of the blocked impulse, and can give clinical staff an indication of the severity of the block.

Keywords Blocks • Bundle branches • Atrioventricular • Sinoatrial • Conduction

Background

There are various types of conduction block, all of which affect different sections of the conduction system. The 12-lead ECG helps to identify the location of the blocked impulse, and can give clinical staff an indication of the severity of the block.

Bundle Branch Blocks

Refers to a block in the electrical impulse down one of the bundle branches, known either as a left or right Bundle Branch Block (LBBB/RBBB). The principle characteristic of a Bundle Branch Block is a widened QRS complex. This occurs because the impulse cannot travel down the fast conduction pathway due to the blockage (Fig. 7.1). The impulse takes an alternative route through the myocardium resulting in delayed ventricular depolarisation and the characteristic widened QRS complex (Fig. 7.2). A normal QRS complex has a width of 0.06–0.10 s (1.5–2.5 small

The online version of this chapter (doi:10.1007/978-1-4471-4962-0_7) contains supplementary material, which is available to authorized users.

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A. Davies, A. Scott, *Starting to Read ECGs*,
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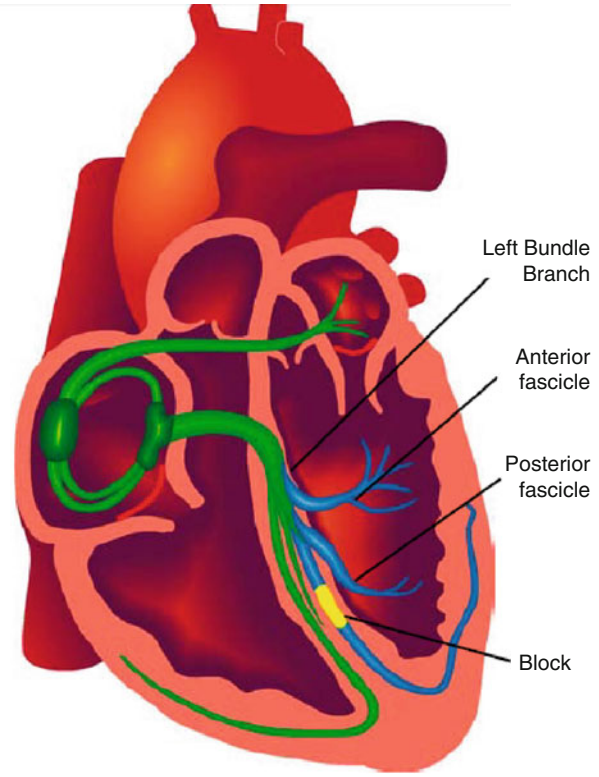


Fig. 7.1 Left Bundle Branch Block

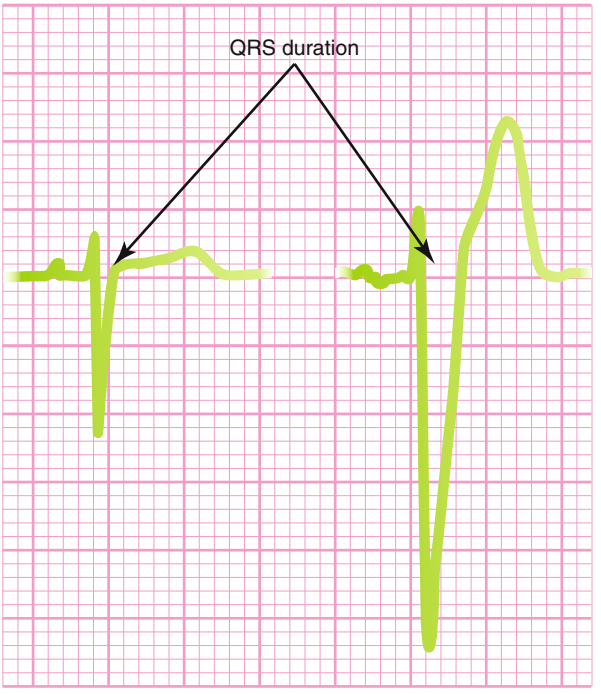


Fig. 7.2 *Left*: Normal QRS duration, *Right*: Widened QRS duration of a LBBB

squares). Anything larger than this should alert the practitioner to the possible presence of a Bundle Branch Block. It is however important to rule out any other causes of a widened QRS.

Other Causes of Widened QRS Complex
- Ventricular paced rhythm
- Ideoventricular rhythm/ventricular escape
- Broad complex tachycardia
- Ventricular premature beats

The easiest method of determining the difference between a LBBB and a RBBB is to look in lead V_1.

Left Bundle Branch Block (LBBB)

If the QRS complex is negatively deflected in lead V_1, wider than 2.5 small squares and cannot be explained by any of the other causes of a widened.

QRS then it is a LBBB (Fig. 7.3).

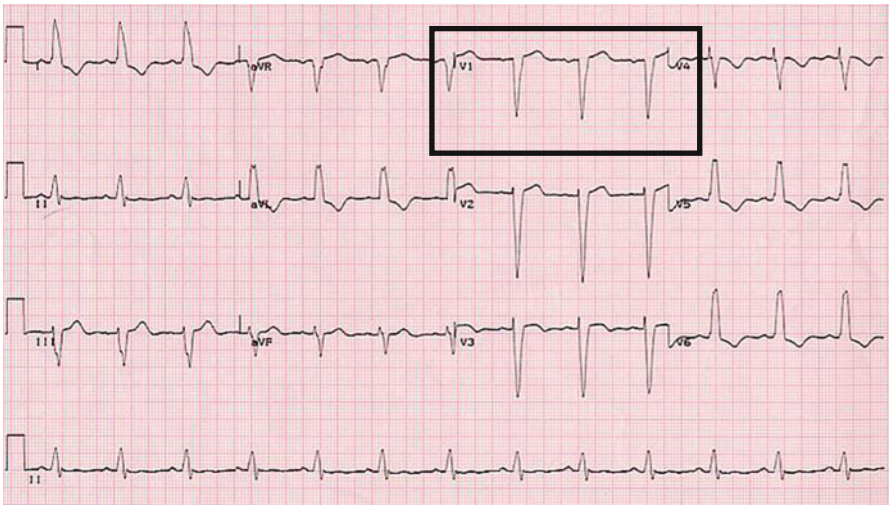


Fig. 7.3 LBBB

Recognising LBBB
- Widened QRS complexes in all leads
- QS pattern in V_1
- T-wave inversion in V_6
- ST-segment elevation sometimes seen in $V_1 - V_4$

Warning: Chest Pain and LBBB

If a patient is experiencing chest pain and has a newly diagnosed LBBB, this should be treated as medical emergency. The patient could be having a Myocardial Infarction and should be treated as such (see Chap. 10 for more details on the management of chest pain and MI).

Right Bundle Branch Block (RBBB)

If the QRS complex is positively deflected in lead V_1, wider than 2.5 small squares and cannot be explained by any of the other causes of widened.

QRS then it is a RBBB (Figs. 7.4 and 7.5). The incidence of RBBB increases with age and does not normally require treatment.

Recognising RBBB
- Widened QRS complexes in all leads
- Widened QRS complexes
- rsR pattern in V_1
- T-wave inversion in V_6

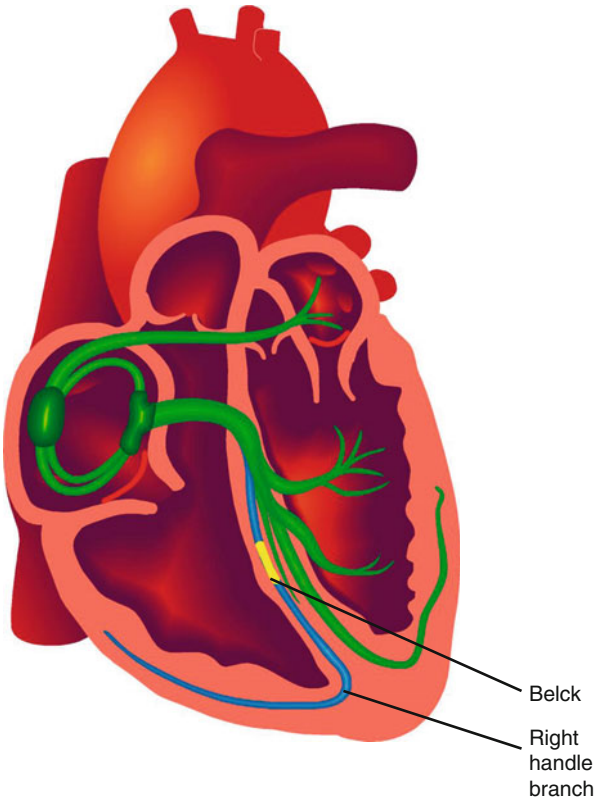


Fig. 7.4 Right Bundle Branch Block

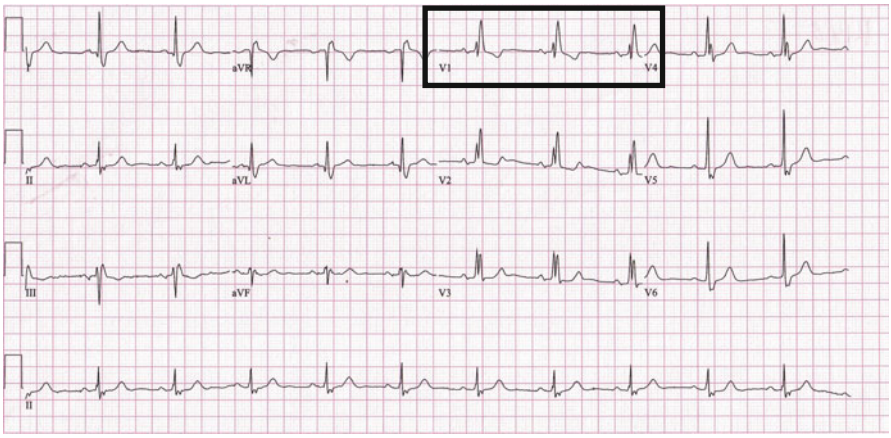


Fig. 7.5 RBBB

Causes of RBBB

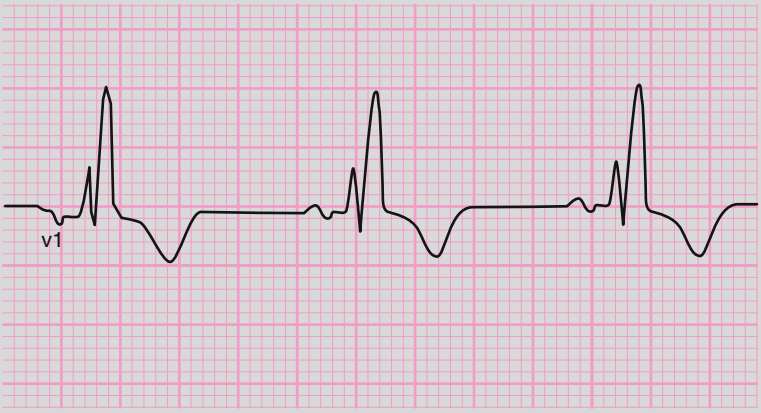
- Ischemic Heart Disease (IHD)
- Myocardial Infarction (MI)
- Normal variant
- Cardiomyopathy
- Fibrosis of conduction system
- Hypertension
- Pulmonary Embolism
- Atrial Septal Defect (ASD)
- Congenital heart disease

Remember

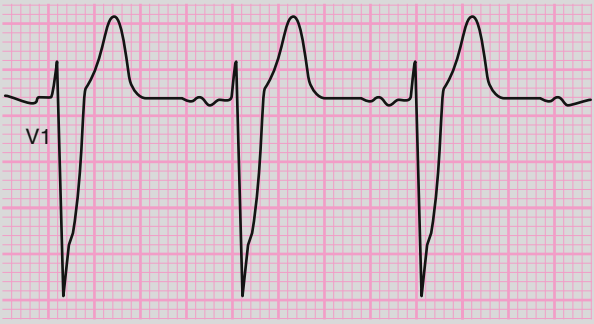
Quick way to tell right and left Bundle Branch Block apart. Widened QRS, look in lead $V_{1.}$

Causes

Positively deflected = RBBB



Negatively deflected = LBBB



Incomplete Bundle Branch Blocks

An incomplete Bundle Branch Block has the same appearance as either a LBBB or a RBBB with the exception of the QRS duration. The QRS width does not fit the requirement for a true Bundle Branch Block but the other features are present.

This is termed an incomplete Bundle Branch Block.

Atrioventricular Blocks (AV Blocks)

This is the failure of the Atrioventricular node to conduct an impulse. AV blocks can be classified as 1st, 2nd and 3rd degree AV blocks. These blocks can be temporary or permanent.

1st Degree AV Block

A 1st degree AV block is a delay between depolarisation of the atria and ventricular depolarisation occurring in the AV node (Figs. 7.6, 7.7 and 7.8). This is seen on the

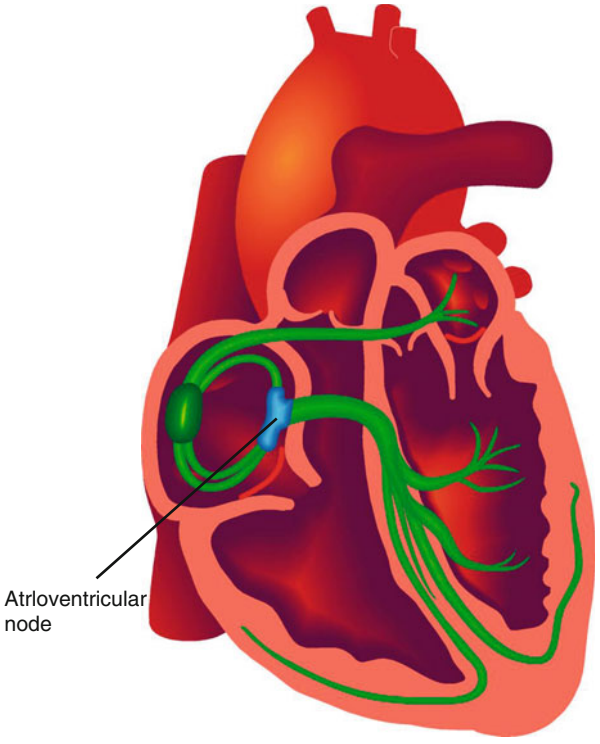


Fig. 7.6 Site of 1st degree AV block

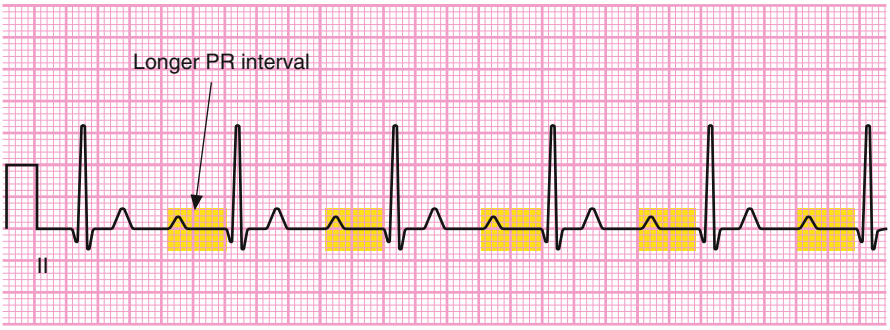


Fig. 7.7 1st degree AV block

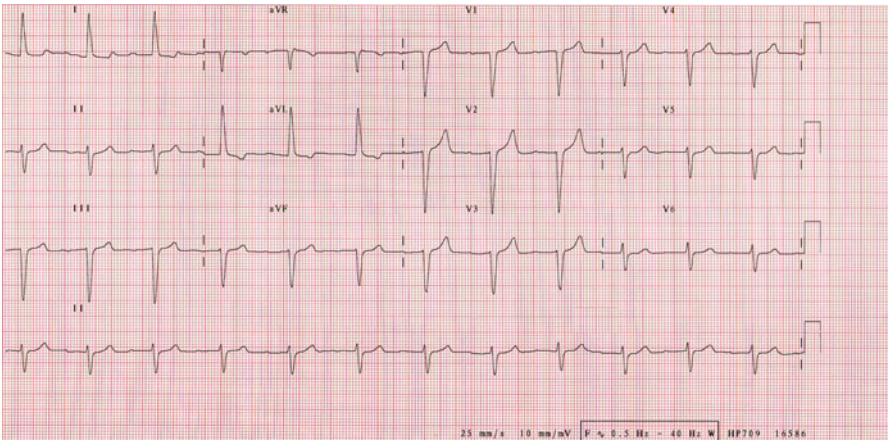


Fig. 7.8 1st degree AV block

ECG as an increase in the PR interval of more than 0.20 s (5 small squares). Each P wave is followed by a QRS complex. The best leads to see this type of block in are lead II and V_1 as the P waves are usually more easily visualised in these leads, especially when used as a rhythm strip at the bottom of the 12-lead ECG. 1st Degree AV block does not normally require treatment.

Causes

- Normal variant
- Ischemic Heart Disease (IHD)
- Increased vagal tone
- Drugs prolonging AV conduction time, such as Beta Blockers
- Congenital heart disease
- Fibrosis of the conduction system
- Acute MI

2nd Degree AV Block

Second degree AV block is split into two further subtypes, known as Type I and Type II 2nd Degree AV blocks.

Type I

This is identified on the ECG by a PR interval which increases in length during progressive cycles, ending in a P wave without a subsequent QRS complex (Fig. 7.9). The cycle then repeats leading to the appearance of grouped QRS complexes on the ECG. The rhythm will be irregular, but there will be a pattern to the irregularity with the groups of QRS complexes. The increasing PR interval ending in the absence of a QRS complex is referred to as the 'Wenckebach phenomenon'. This form of block does not normally require intervention unless the patient is symptomatic. Many people experience this form of block while sleeping. If however the patient has this form of block while awake and is symptomatic, consideration should be given to the insertion of a Permanent Pacemaker (PPM). There is a possibility that this block may progress to a worse form of block requiring treatment.

Causes
- Normal variant
- Ischemic Heart Disease (IHD)
- Drugs prolonging AV conduction time, such as Beta Blockers
- Atrial Septal Defect (ASD)
- Acute MI
- Mitral valve prolapse
- Aortic valve disease

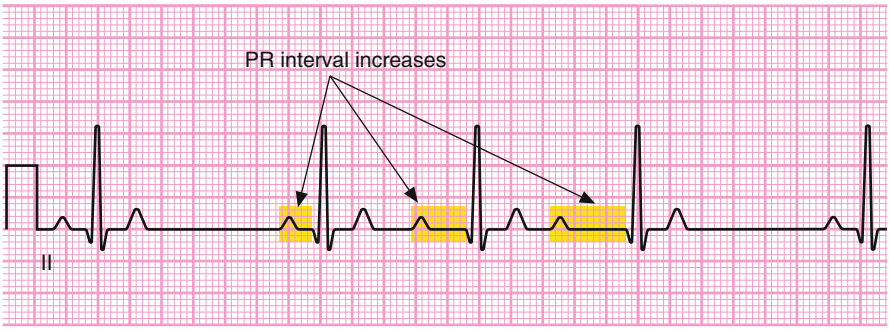


Fig. 7.9 2nd degree, type I AV block

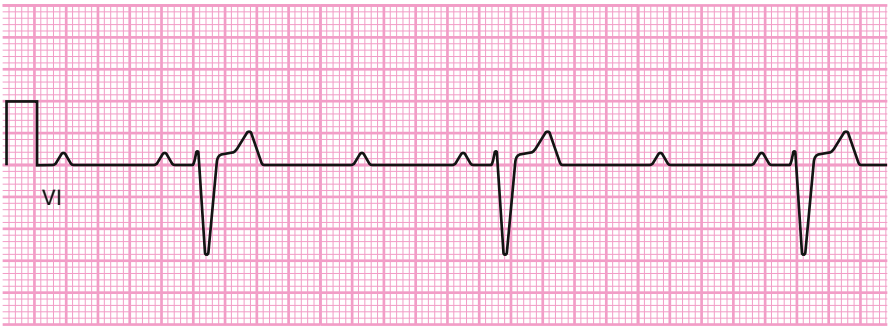


Fig. 7.10 2nd degree, type II AV block

Type II

The second type of 2nd degree block occurs in the bundle branches or the His bundle and is a form of intermittent bilateral Bundle Branch Block (both the left and right Bundle Branches) and is often found in the presence of an existing Bundle Branch Block. The ECG characteristics consist of occasional dropped QRS complex and a constant (same length) PR interval (Fig. 7.10). This form of block carries a worse prognosis. Cardiac output may be reduced if there is a slow ventricular rate. The patient may also progress from a 2nd degree type II block to Complete Heart Block (CHB). If the block is intermittent the rhythm will be irregular, if however the block is consistent the rhythm may be regular.

Causes
- Normal variant
- Cardiomyopathy
- Coronary Artery Disease (CAD)
- Fibrosis of the conduction system
- MI (usually anterior wall)

3rd Degree AV Block

Otherwise known as Complete Heart Block (CHB). This is due to the inability of any atrial impulse to pass to the ventricles. The presence of escape beats prevents ventricular standstill (P wave Asystole) occurring. The principle ECG characteristics consist of complete atrial/ventricular disassociation. There is no connection between the P waves and QRS complexes. P waves occur without preceding QRS

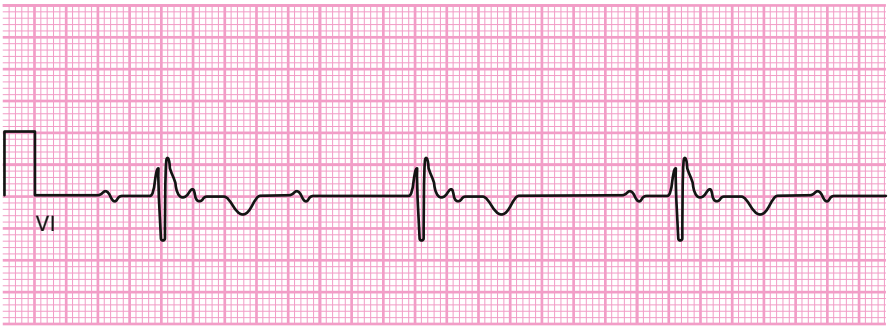


Fig. 7.11 3rd degree AV block

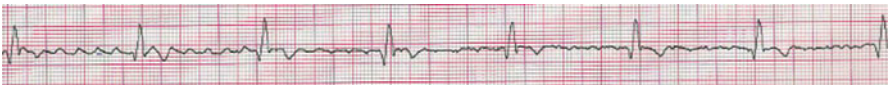


Fig. 7.12 3rd degree AV block and atrial fibrillation

complexes, both P waves and QRS complexes are regular but not associated with one another (Fig. 7.11). This can lead to serious haemodynamic effects causing patients a variety of symptoms, including: chest pain, altered mental status and loss of consciousness.

Causes
- Normal variant
- Ischemic Heart Disease (IHD)
- Increased vagal tone
- Drugs prolonging AV conduction time, such as Beta Blockers
- Congenital heart disease
- Fibrosis of the conduction system
- Acute MI

3rd Degree AV Block and Atrial Fibrillation

There is one incidence where AF may appear regular rather than irregularly irregular and this is in the presence of 3rd degree AV block. No P waves are visible and the characteristic undulating chaotic baseline of AF can be seen but with a regular ventricular rhythm (Fig. 7.12).

Pacing
Patients may require temporary pacing. If however the block is permanent a Permanent Pacemaker (PPM) system may be required.

Sinoatrial Blocks (SA Blocks)

Caused by the inability of the SAN to activate the atria, leading to an absence of P waves on the ECG. These blocks can be classified as either complete or incomplete SA blocks. Incomplete blocks cause the occasional loss of beats. Complete SA block occurs when no impulses leave the SAN leading to 'Sinus Arrest', a complete lack of heart beats (no PQRS or T waves present).

Incomplete SA Blocks

The intermittent absence of atrial activation causes a gap or 'pause' on the ECG. This pause is either caused by a gradual reduction in the P to P interval (the distance between one P wave and the next). This results in a pause, the cycle then continues. This is also known as type I sinus exit block (Fig. 7.13).

The other form of incomplete SA block, known as type II sinus exit block is determined due to its mathematical relationship with the conduction cycle. In this type of SA block there is no shortening of the P to P interval but instead an unexpected absence of a P wave and subsequent QRS complex. The pause is multiple of the P to P interval (Fig. 7.14).

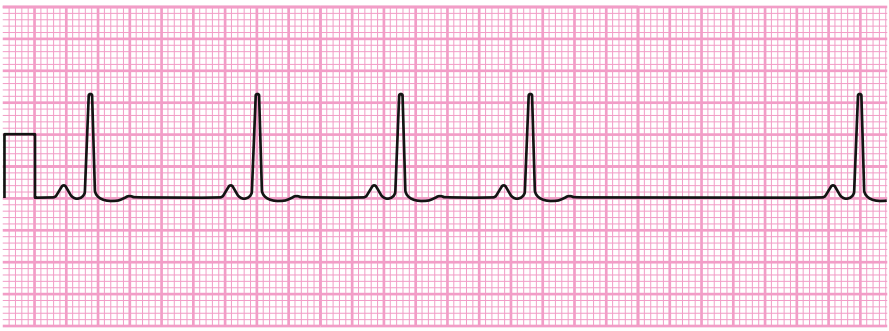


Fig. 7.13 Type I sinus exit block

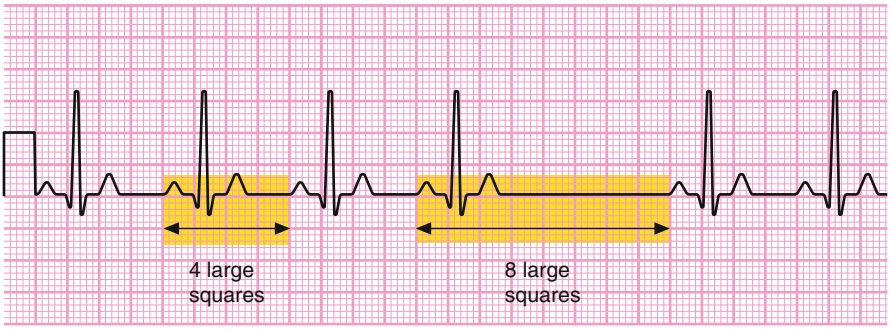


Fig. 7.14 Type II sinus exit block

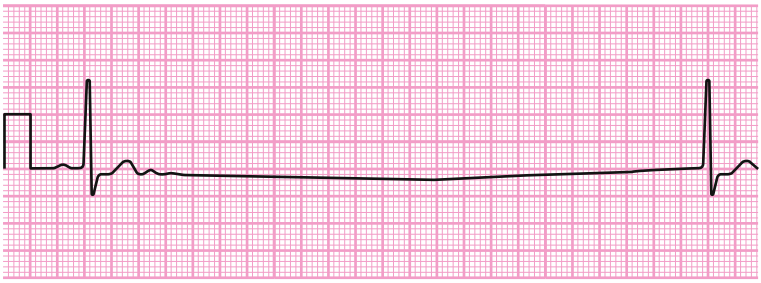


Fig. 7.15 Complete SA block

Complete SA Block

Complete SA block otherwise known as 'sinus arrest' has no such mathematical relationship. The pauses can last several seconds, and may cause patients to collapse (Fig. 7.15). The pause is normally terminated by an escape beat. As discussed earlier these escape beats originate further down the conduction system and are known as junctional or ventricular escape beats, depending upon their origin. These beats act as a safety net preventing Asystole. Treatment may require the use of drugs such as atropine or the insertion of an artificial pacemaker.

Common Causes of Absent P Waves
- Impulse does not leave SAN
- SAN fails to generate an impulse
- Impulse is inadequate and fails to activate the atria
- Paralysis of the atria preventing activation.

Sick Sinus Syndrome (SSS)

Otherwise know as Brady-tachy syndrome features alternating periods of tachycardia and bradycardia on the same ECG (Fig. 7.16). Atrial fibrillation or flutter may also be seen on the ECG causing the tachycardia. The bradycardia is caused by sinus pauses or periods of sinus arrest.

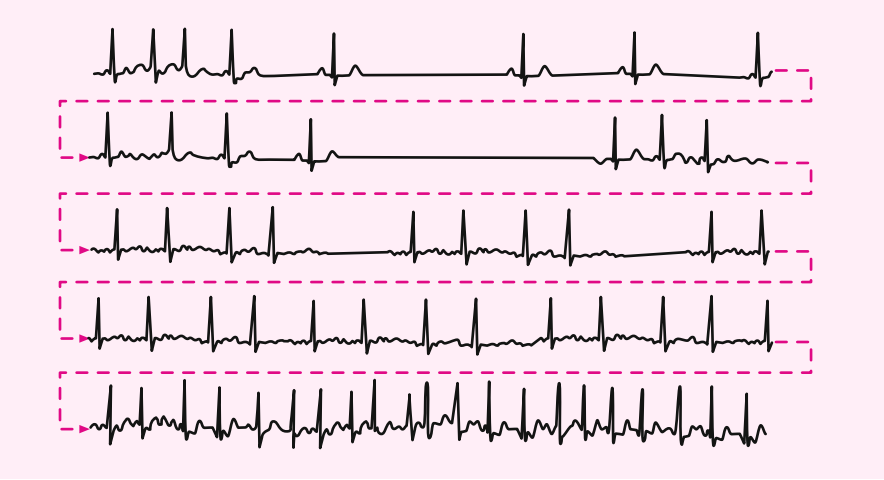


Fig. 7.16 SSS taken from a continuous rhythm strip

Causes
- Fibrosis of the SAN
- Cardiomyopathy
- Ischemic Heart Disease (IHD)
- Drugs (such as Digoxin, Beta Blockers, Calcium Channel Blockers)
- Surgical damage to the SAN
- Inflammatory cardiac disease

Summary of Key Points

- The type of block depends on its location within the conduction system.
- AV blocks are split into three types, 1st, 2nd and 3rd degree, with 2nd degree AV blocks further subdivided into type I and II 2nd degree AV blocks.
- SA blocks can be classified as either complete or incomplete.
- LBBB and RBBB can be discerned by looking in lead V_1.
- Chest pain and a new LBBB should be considered a medical emergency.

Quiz

Q1. LBBB is identified by…

(A) A widened QRS and a negative deflection in lead V1
(B) A widened QRS and a positive deflection in lead V1
(C) A narrow QRS and a negative deflection in lead V1

Q2. RBBB normally requires treatment

(A) True
(B) False

Q3. Wenckeback phenomenon refers to…

(A) Short PR interval
(B) Gradually increasing PR interval ending in a dropped beat
(C) Alternating dropped beats

Q4. 1st Degree AV block is identified by…

(A) A PR interval >0.20 s, each P wave is followed by a QRS complex
(B) A PR interval <0.20 s, each P wave is followed by a QRS complex
(C) A PR interval >0.20 s, every other P wave is followed by a QRS complex

Q5. The purpose of an escape beat is…

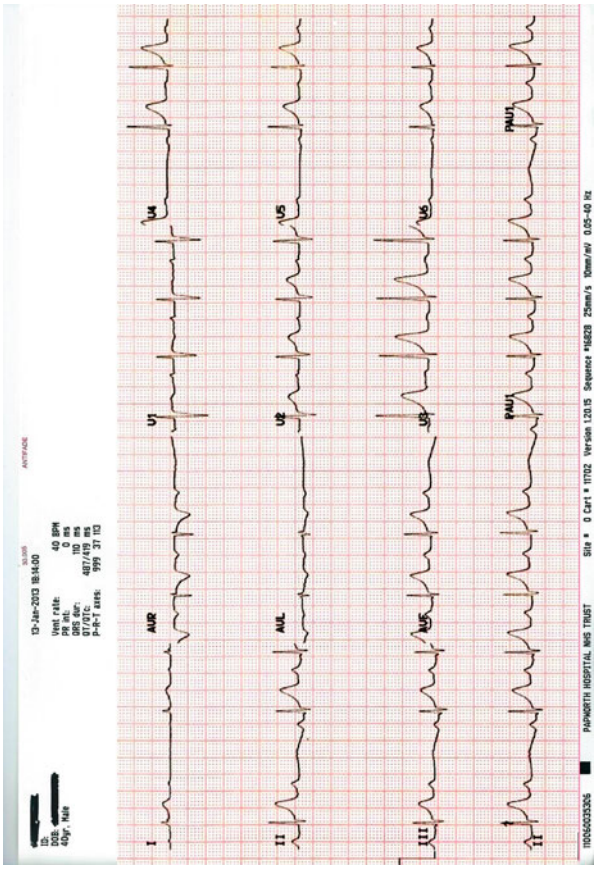
(A) To repolarize the atria
(B) Acts a buffer between the atria and ventricles preventing Ventricular Fibrillation (VF) occurring
(C) Act as a safety net to prevent ventricular standstill (Asystole)

Q6. SA block can either be complete or incomplete

(A) True
(B) False

What does the following ECGs show?

Q7.



Answers: Q1 = A, Q2 = B, Q3 = B, Q4 = A, Q5 = C, Q6 = A, Q7 = 2nd Degree AV block type I

Chapter 8
Miscellaneous Cardiac Conditions

Abstract There are various miscellaneous cardiac conditions that are easily identifiable on an ECG. The aim of this chapter is to look at some of these conditions as they don't readily fit into the classifications discussed in previous chapters. We aim to present a basic overview of the most commonly encountered conditions that present on a regular basis in the clinical setting.

Keywords Pacing • Pacemaker • Pericarditis • Long QT syndromes

Background

There are various miscellaneous cardiac conditions that are easily identifiable on an ECG. The aim of this chapter is to look at some of these conditions as they don't readily fit into the classifications discussed in previous chapters. We aim to present a basic overview of the most commonly encountered conditions that present on a regular basis in the clinical setting.

Paced Rhythms

Sometimes due to damage to the conduction system, or to manage certain types of heart block, it becomes necessary to insert a Permanent Pacemaker (PPM) system. The PPM system consists of one or more electrodes (leads) and a battery (pulse generator). The pulse generator can detect the patient's rhythm and emit an

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Table 8.1 Common pacemaker codes

Chamber(s) paced	Chamber(s) sensed	Sensing response	Rate modulation	Multisite pacing
0 = None	0 = None	0 = None	0 = None	0 = None
A = Atrium	A = Atrium	T = Triggered	R = Rate modulation	A = Atrium
V = Ventricle	V = Ventricle	I = Inhibited		V = Ventricle
D = Dual (A + V)	D = Dual (A + V)	D = Dual (T + I)		D = Dual

electrical impulse via the electrode(s) to the myocardium, leading to subsequent activation of one or more of the hearts chambers as required.

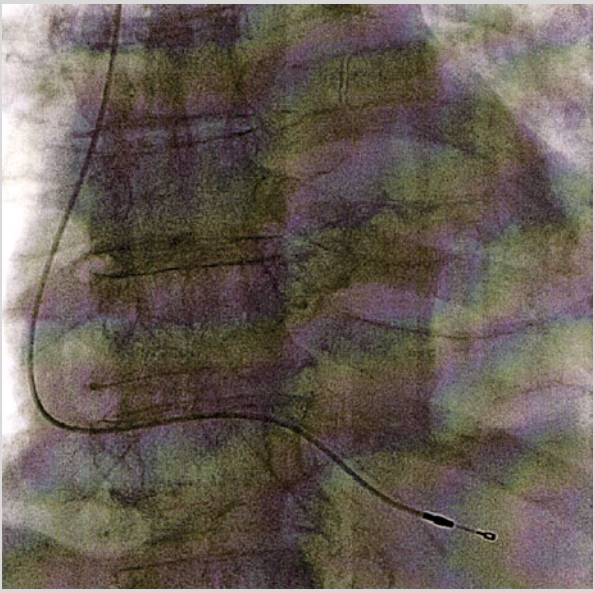
Types of Permanent Pacemaker (PPM)

There are various types of PPM that pace different chambers of the heart. The choice of pacemaker system used depends very much on the patient's condition and the underlying rhythm of the patient. Most systems are inserted in theatre or a cardiac catheterization lab using fluoroscopic imaging to position the leads, which are inserted through a vein (i.e. the cephalic or Subclavian vein). There are several different modes a pacemaker can be set to. To distinguish between these different settings pacemakers are given a code to define their type and function. Table 8.1 summarises these pacemaker codes.

This may seem at first glance quite complicated but as with ECG recognition it gets easier the more you practice and the more you see in clinical settings in patients notes. Two examples of commonly encountered pacemaker system are: A VVI pacemaker describes a PPM system that paces the ventricle, senses the ventricle and inhibits pacing if it detects the patients intrinsic rhythm. A DDDR

Types of Permanent Pacemaker (PPM)
- Atrial pacing
- Ventricular pacing
- Dual chamber/Sequential A-V pacing, (paces atrium and ventricle)
- Bi-ventricular pacing, (paces both ventricles)

Fluoroscopic Imaging
Low level X-ray imaging is used to position the pacemaker leads in the correct position in the relevant heart chamber(s).



This image shows an electrode positioned in the patients right ventricle.

pacemaker, on the other hand, refers to a system that paces both the atria and ventricle in which both can be triggered/inhibited and can respond to rate demands, i.e. increases the heart rate if the patient needs a higher output for a physical task.

The majority of PPM systems are placed in the right side of the heart to pace the right atria and/or the right ventricle. The exception to this is the Bi-v pacemaker, also known as Cardiac Resynchronization Therapy (CRT), which is often used to treat heart failure by synchronizing the contraction of the ventricles to improve the quality of contractions. The Bi-v pacemaker paces the left ventricle in addition to the right. The pacing lead cannot be passed directly into the left ventricle as the arterial blood would clot around the electrode increasing the risk of thromboembolism; there is also a risk of damage to the aortic valve. To get around this problem the left ventricle lead is placed in a vein outside the heart, behind the left ventricle, reached via the coronary sinus (Fig. 8.1) to pace the left ventricle from behind. The different pacemaker systems are highlighted in Fig. 8.2.

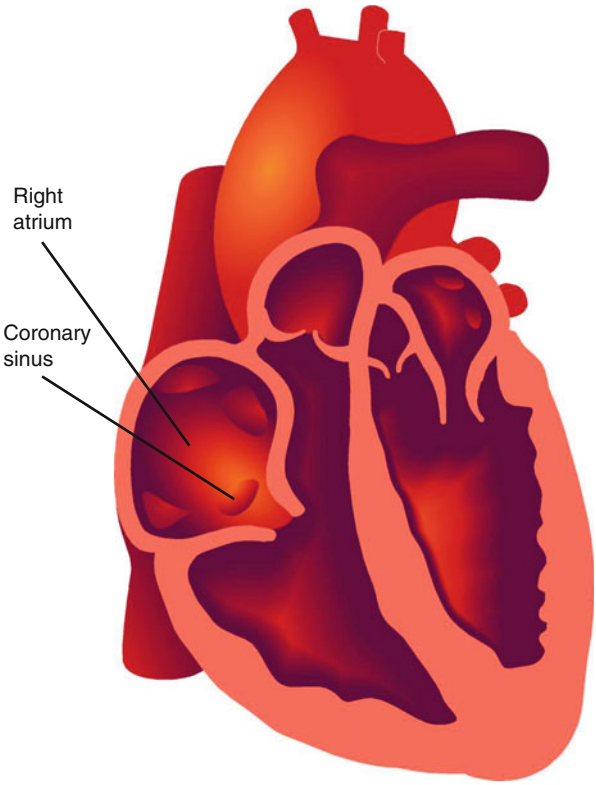


Fig. 8.1 Image of the heart showing the location of the coronary sinus

ECG Identification of Pacemakers

Pacemakers are identified on the ECG by a wide QRS (>0.11) and the presence of 'pacing spikes'. Pacing spikes (Fig. 8.3) are small vertical lines seen just before either the P wave, the QRS complex or both.

The location of the spikes helps to identify the type of pacemaker system in situ. A spike before a P wave denotes atrial pacing, whereas one before the QRS complex identifies ventricular pacing. Spikes before both P waves and QRS complexes can be either dual chamber (atria and ventricle) or Bi-v pacing. As both chambers are synchronized in the Bi-v system only one spike is usually visible. Figures 8.4 and 8.5 show single and dual chamber pacemaker systems.

Problems with Pacemakers

As with all manmade devices there is the possibility that the device can be faulty, either due to a fault in the manufacturing process or in the programming of the device. Pacemaker malfunctions can often be seen on the 12-lead ECG. The most commonly encountered problems associated with pacemaker systems include: failure to sense, failure to capture, failure to pace and over sensing.

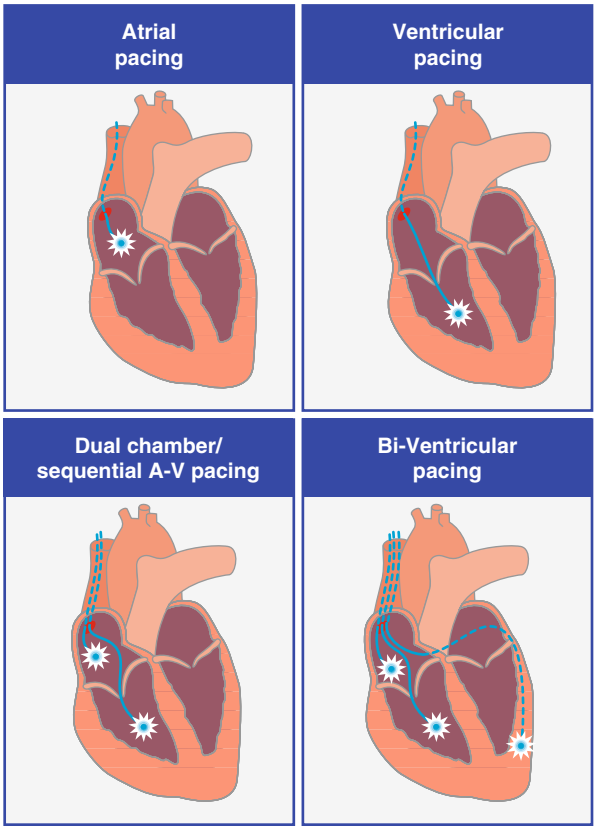


Fig. 8.2 The different systems used for permanent pacing

Fig. 8.3 A pacing spike

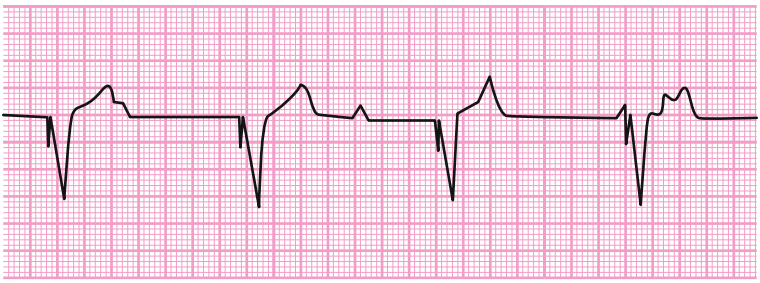


Fig. 8.4 Single chamber ventricular pacing (spikes before QRS complex)

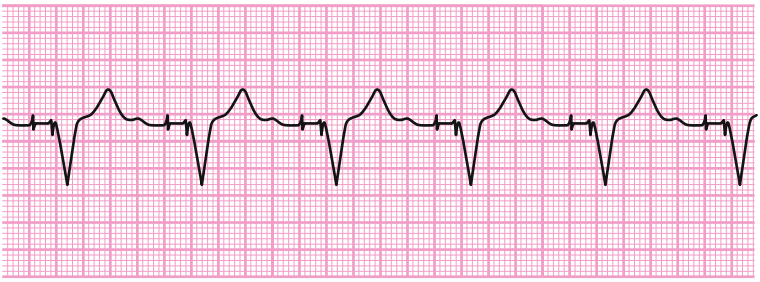


Fig. 8.5 Dual chamber pacemaker (spikes before both P and QRS)

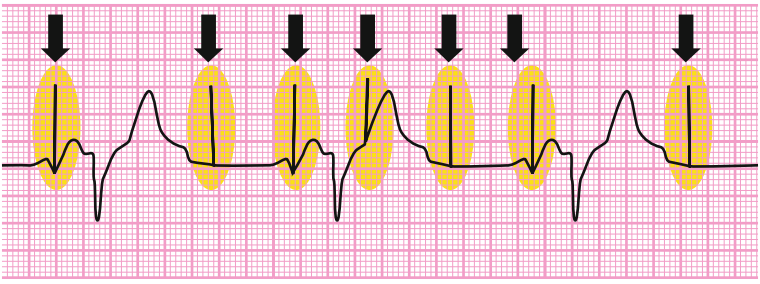


Fig. 8.6 Failure to Sense. *Arrows* show presence of pacing spikes on the ECG appearing at various points in the intrinsic cardiac cycle

Failure to Sense

The pacemaker continues to 'fire' despite the presence of existing spontaneous depolarisation. This is to say the pacemaker fails to detect the presence of the patient's natural heart beat and activates anyway. This can be dangerous for the patient if the pacing spike falls on the T wave as this can trigger VT or VF requiring emergency treatment. This is identified on the ECG by the presence of pacing spikes appearing at various points in the intrinsic cardiac cycle. The patient may be aware of palpitations or the sensation of missing beats (Fig. 8.6).

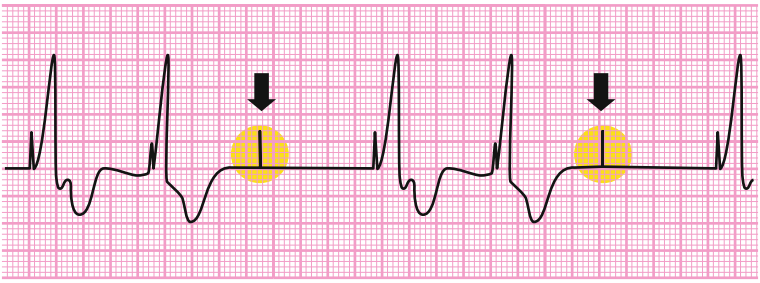


Fig. 8.7 Failure to Capture. *Arrows* show presence of pacing spikes on the ECG without a subsequently paced QRS complex or P wave

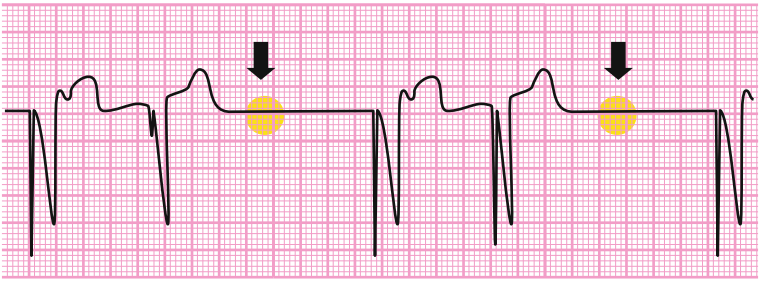


Fig. 8.8 Failure to Pace. *Arrows* show the presence of a gap in between complexes

Failure to Capture

The pacemaker fires but fails to pace the myocardium. This can again be dangerous for the patient as they can be subject to the condition the pacemaker was inserted to treat if the pacemaker fails to work correctly. This is seen on the ECG by the presence of pacing spikes without a subsequently paced QRS complex or P wave (Fig. 8.7).

Failure to Pace

This is the complete absence of pacing spikes and paced complexes where expected. The ECG often shows a gap in between complexes. This can be problematic for patients as it can decrease cardiac output (Fig. 8.8).

Over-Sensing

This is caused by the pacemaker misreading interference (i.e. muscular movement) or other parts of the cardiac cycle and assuming that they are the patient's intrinsic

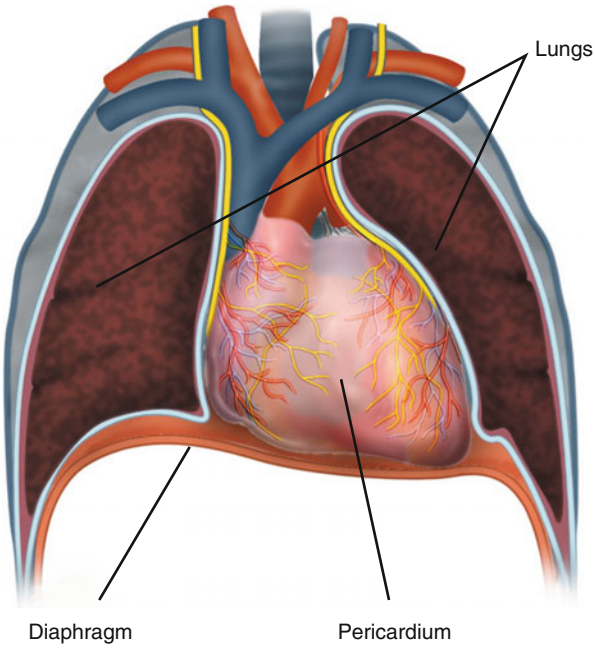


Fig. 8.9 The Pericardium

rhythm. The pacemaker then does not perceive the need to fire. This leads to the pacemaker not activating when required. This is seen on the ECG as no or insufficient pacemaker activity.

Pericarditis

Pericarditis is an inflammation of the pericardium, the sac surrounding the heart (Fig. 8.9). The condition can be classified as being either acute or chronic.

Signs & Symptoms of Pericarditis

Acute

- Retro-sternal chest pain
- Arrhythmias
- Fever/chills
- Reduction in pain on leaning forward
- Worsening pain on inspiration
- The presence of friction rub (sound generated by friction)

Chronic

- Retro-sternal chest pain
- Oedema
- Enlarged liver (Hepatomegaly)
- Build-up of fluid in the abdomen (Ascites)

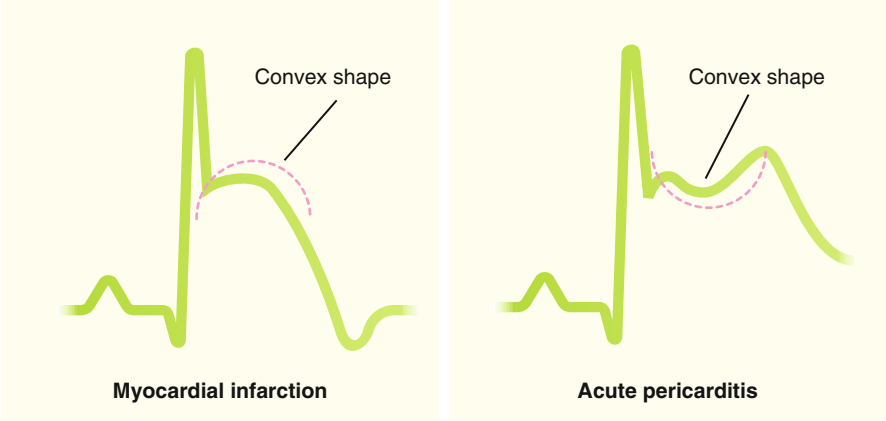


Fig. 8.10 Typical ST segment elevation shapes as seen in an MI and Pericarditis

Differentiating Pericarditis from Acute Myocardial Infarction

The main difference between Pericarditis and an MI is the shape of the ST elevation. Pericarditis has a concaved shape, as oppose to the convex shape seen in an MI (Fig. 8.10). In addition the ST segment changes in a MI to reflect the areas affected, as oppose to the more widespread ST elevation seen in Pericarditis (Fig. 8.11).

What Is ST Segment Elevation?

ST segment elevation refers to the elevation (rising) of the ST segment above the isoelectric baseline measuring 1 mm (1 small box) or more. ST elevation is measured from the baseline to the J point in mm, as shown in Fig. 8.12.

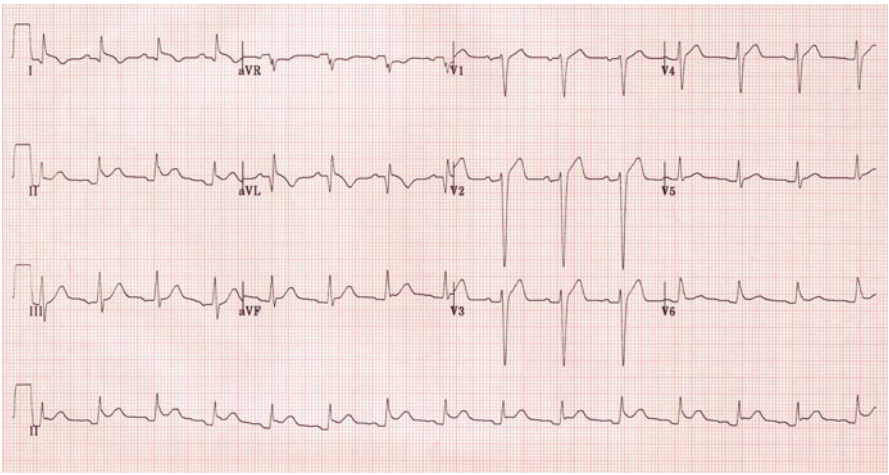


Fig. 8.11 Pericarditis. The classic saddle shaped (concave) ST elevation seen in multiple leads

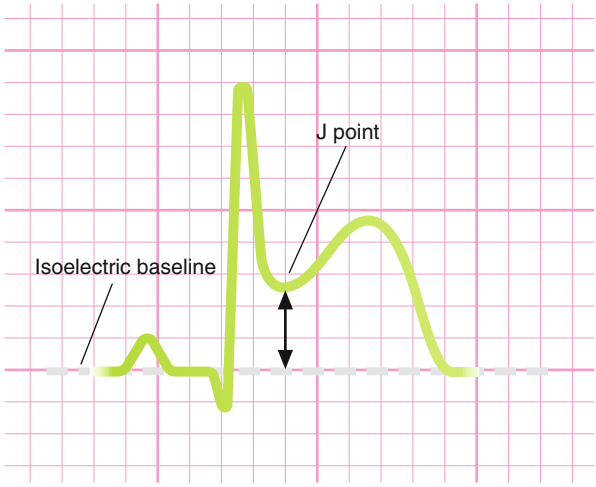


Fig. 8.12 ST segment elevation measurement

Other Causes of ST Elevation

Many people talk about ST elevation in the context of acute Myocardial Infarction (MI). It is important however to realise that there are several different causes of ST elevation.

Causes of ST-Segment Elevation
- Acute Myocardial Infarction (MI)
- Acute Pericarditis
- High take off
- Brugada syndrome
- Left ventricular aneurysm
- Prinzmetals angina

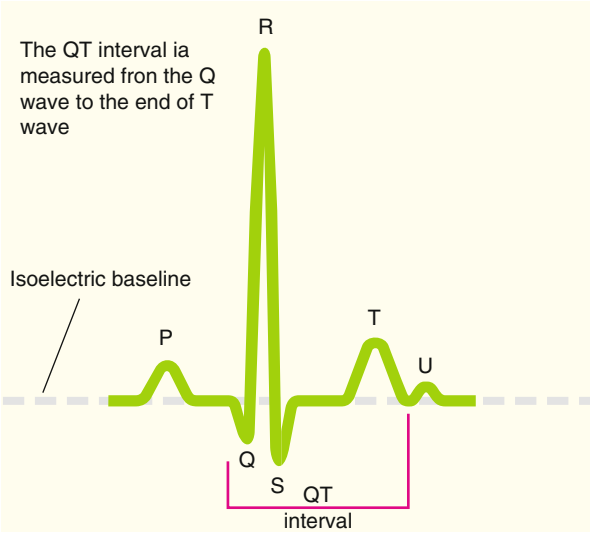


Fig. 8.13 The QT interval

Long QT Syndromes

There are several reasons why the QT interval may be elongated. The principle causes can include:

- The effect of certain drugs
- Hereditary syndromes
- A long QT interval may also be a feature of certain other conditions but not diagnostically definitive (e.g. cerebral injury).

The QT interval varies with the patient's heart rate (Fig. 8.13). To compensate for this a formula was developed to account for this and provide a corrected QT interval. The normal QTc should fall in the range of 0.35–0.43 s (just over two large

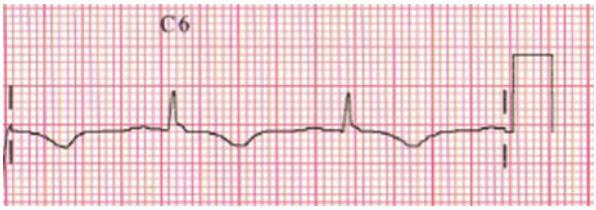


Fig. 8.14 A long QT interval

boxes). Most ECG machines will display both the QT and QTc interval data, it is however important that the interpreter of the ECG can work this out independently, which may be required with older machines.

A long QT interval (Fig. 8.14) can be dangerous for a patient as it can predispose them to ventricular arrhythmias leading potentially to sudden death. If the cause of the prolongation is due to drugs then the patient's drug regime should be reviewed and altered accordingly. If however the prolongation is due to a hereditary syndrome then the patient may have an implanted cardiac defibrillator fitted to shock them out of any dangerous arrhythmias.

Drugs that May Increase the QT Interval

- Some Tricyclic antidepressants
- Some Anti-arrhythmogenics
- Some antibiotics (e.g. Voriconazole)

Hereditary Conditions

- Romano-Ward syndrome, autosomal dominant with normal hearing
- Jervell and Lange-Nielsen syndrome, autosomal recessive with congenital deafness

Both conditions predispose the patient to dangerous ventricular arrhythmias (Ventricular Tachycardia/Fibrillation and/or Torsades).

Summary of Key Points

- Pacemakers can be identified on the ECG by the presence of pacing spikes.
- Pacing spikes indicate which of the heart's chambers are being paced.
- Pericarditis can be identified by widespread ST segment elevation in multiple leads, with a concave shape.
- ST segment elevation is measured from the isoelectric baseline to the J point in mm.

- Long QT syndromes can predispose patients to dangerous ventricular arrhythmias such as Ventricular Tachycardia/Fibrillation.
- Long QT syndromes can be caused by drugs or may be the result of a hereditary condition.

Quiz

Q1. Pacemakers are identified on the ECG by the presence of…

(A) *Pacing spikes*
(B) *Small dots above each QRS complex*
(C) *Small P waves*

Q2. The shape of ST elevation on ECGs associated with Pericarditis is said to be…

(A) *Convex*
(B) *Concave*

Q3. The normal corrected QT interval (QTc) is…

(A) *0.25–0.43 s*
(B) *0.35–0.43 s*
(C) *0.12–0.2 s*

Q4. A pacemaker with the code VVI…

(A) *Is used to treat advanced heart failure*
(B) *Paces both the atria and ventricles*
(C) *Senses the ventricle, paces the ventricle and inhibits if it detects the patients intrinsic rhythm*

Q5. ST segment elevation is measured from the Isoelectric base line to the…

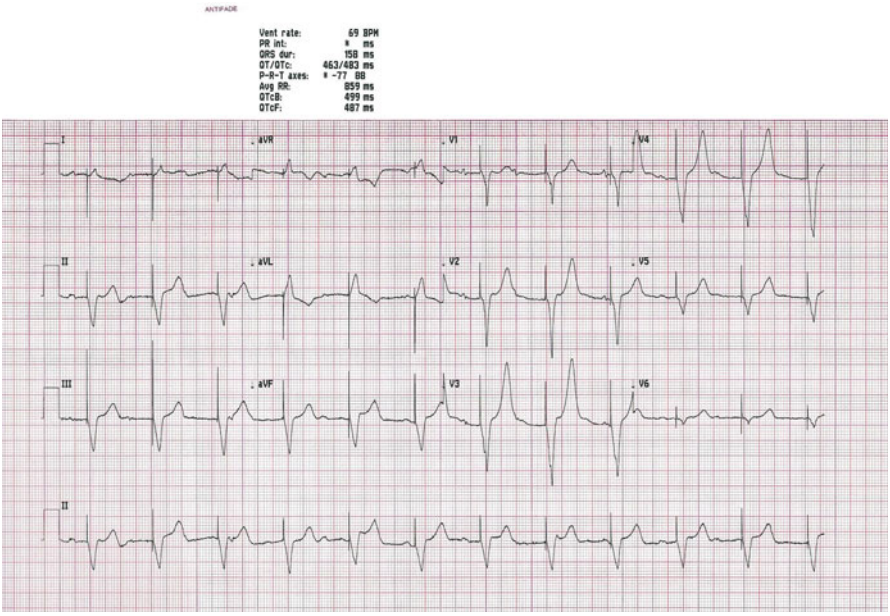
(A) *F point*
(B) *J point*
(C) *ST region*

Q6. A Bi-v pacemaker (CRT) is used to treat…

(A) *Mumps*
(B) *Heart failure*
(C) *Complete Heart Block* (*CHB*)

Interpret the following ECG.

Q7.



Answers: Q1 = A, Q2 = B, Q3 = B, Q4 = C, Q5 = B, Q6 = B, Q7 = Ventricular paced rhythm

Chapter 9
Non Cardiac Conditions Identifiable on the ECG

Abstract There are various conditions, diseases and pharmacological effects that can be identified on a 12-lead ECG, and are often seen in clinical practice. It is important to state that the ECG is usually only a guide in diagnosis for these conditions as further tests, such as bloods normally need to be carried out to confirm the diagnosis.

Keywords Electrolytes • Digitalis • Embolism • Hypothermia • Metabolic

Background

There are various conditions, diseases and pharmacological effects that can be identified on a 12-lead ECG, and are often seen in clinical practice. It is important to state that the ECG is usually only a guide in diagnosis for these conditions as further tests, such as bloods normally need to be carried out to confirm the diagnosis.

Electrolyte Imbalances

Electrolytes are electrically conductive substances containing free ions. The body requires a balance between the electrolytes inside and outside of cells (intracellular and extracellular ions). These electrolytes include Magnesium (Mg^{2+}), Sodium (Na^{+}), Potassium (K^{+}) and Calcium (Ca^{2+}). Imbalances in these electrolytes can cause serious physiological problems, so all electrolyte imbalances should be identified and treated promptly.

The online version of this chapter (doi:10.1007/978-1-4471-4962-0_9) contains supplementary material, which is available to authorized users.

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Table 9.1 The effects of hyperkalemia on the ECG dependent on level

Mild	Moderate	Severe
K+ ≥5.5 mmol/l	K+ ≥6.5 mmol/l	K+ ≥7.0 mmol/l
Tall peaked T waves	Prolonged PR interval	Broad QRS complexes
Flat (low amplitude) P waves	Wide P waves	Ventricular arrhythmias
	No P waves	Asystole
		ST segment elevation

Fig. 9.1 ECG changes in QRS complexes and T waves compared with a normal sinus beat

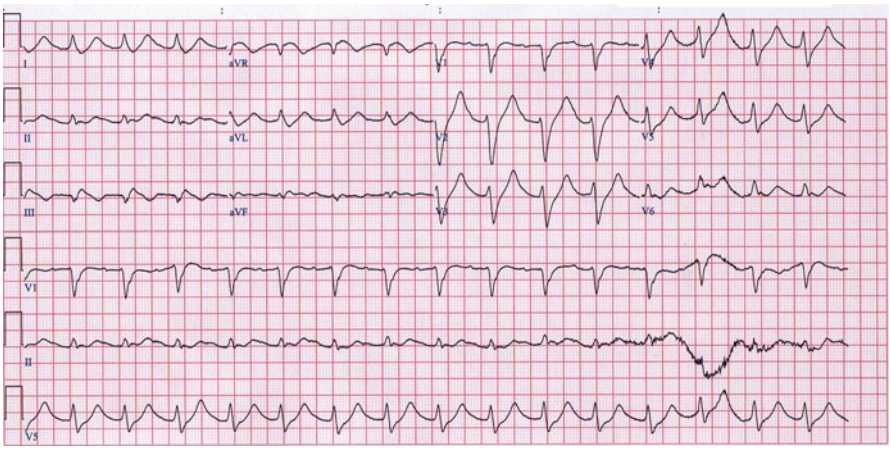

Fig. 9.2 ECG showing moderate to severe hyperkalemia

Common Causes of Electrolyte Imbalance

- Dehydration
- Starvation/malnutrition
- Diarrhoea
- Vomiting
- Drugs that increase electrolyte levels (i.e. IV Potassium)
- Drugs that decrease electrolyte levels (i.e. Diuretics)

Hyperkalemia

Is a condition defined by elevated levels of serum potassium. Normal potassium levels are around 3.5–5.5 mmol/l. An increase above the normal range can predispose individuals to dangerous ventricular arrhythmias and cardiac arrest. The ECG characteristics can be difficult to identify as different signs are present depending on the level of potassium (Table 9.1). The most obvious and consistent sign in the earlier stages is the presence of tall and peaked T waves (Figs. 9.1 and 9.2). As the levels of potassium increase, further changes can be seen on the ECG.

Hypokalemia

Is caused by lower than normal levels of potassium. As with hyperkalemia dangerous ventricular arrhythmias can be a consequence of hypokalemia. In contrast the T waves tend to be small (the opposite of hyperkalemia). Another key feature is the presence of prominent U waves.

ECG Findings in Hypokalemia
- In severe cases peaked P waves
- PR interval may also be prolonged
- QRS complex may be wide
- ST depression
- T waves reduced in amplitude (small T waves)
- Prominent U waves
- Prolonged QT interval

Hypercalcaemia

Is caused by an increase in calcium above the normal values. Normal calcium levels range from 8.5 to 10.5 mg/dl. Patients may experience bradyarrhythmias, ventricular arrhythmias and heart block (AV nodal block) when the levels of calcium are depleted below normal levels. ECG findings can be non-specific and difficult to distinguish.

ECG Findings in Hypercalcaemia
- Short QT interval
- Prolonged PR interval
- Wide QRS complex
- Ventricular arrhythmias

ECG Findings in Hypocalcaemia
- Prolonged QT interval
- Flat or inverted T waves
- Ventricular arrhythmias

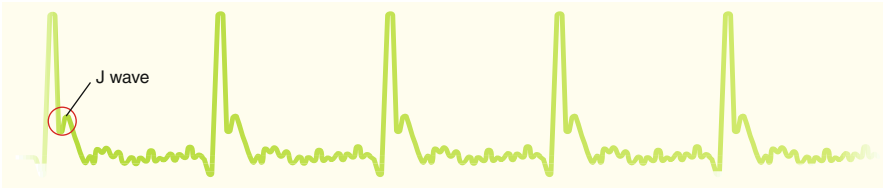

Fig. 9.3 What is a J wave? A positive deflection seen at the junction of the QRS complex and T wave

Hypocalcaemia

Caused by lower than normal levels of calcium. The main ECG feature that is probably the most specific is the prolonged QT interval. Flat or inverted T waves are less commonly seen.

Hypothermia

Normal body temperature is around 37 °C. If core temperature falls below 35 °C, a person experiences mild hypothermia. The patient normally feels intense cold and suffers bouts of shivering. If the core temperature falls below 32 °C the patient suffers severe hypothermia, which may cause cognitive impairment or even comma. Hypothermia can be as a result of environmental exposure to the cold. Many elderly patients that live alone and suffer falls can often be left on the floor for some time before being discovered, many of these patients develop hypothermia while they await discovery. Hypothermia is easily diagnosed by the use of a thermometer.

There are however several key features which are also seen on the 12-lead ECG. These include:

- Bradycardia
- A prolonged QT interval
- The presence of J waves (Fig. 9.3)

In many cases some or all of these details can be hard to discern, as many ECGs from hypothermic patients contain shivering artifact.

Digoxin Use

Digoxin is a cardiac glycoside derived from the Fox Glove plant (digitalis). This type of drug increases the force of cardiac contractions. Cardiac glycosides also slow down conductivity in the AV node. Digoxin is used to treat atrial fibrillation, atrial flutter and heart failure. One of the potential problems concerned with the use of Digoxin is the possibility of Digoxin toxicity, where levels of Digoxin build up in the body to toxic levels. Hypokalemia can also predispose patient's taking Digoxin to Digoxin toxicity. One of the prominent signs of Digoxin use on the ECG

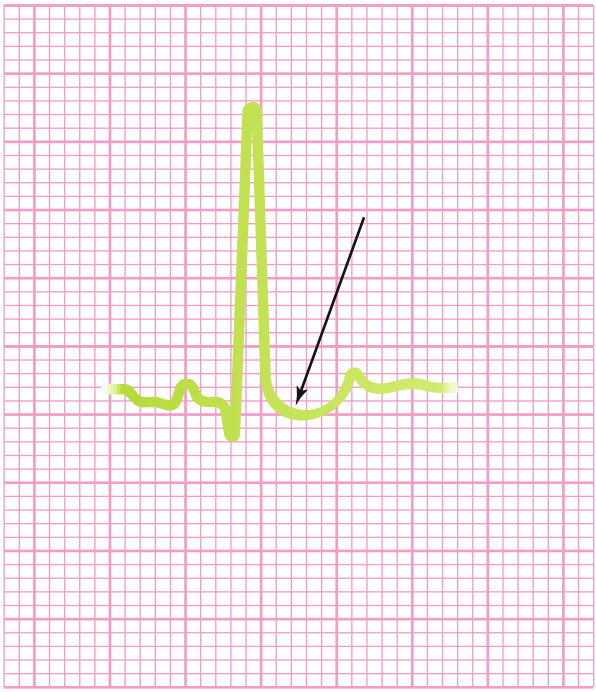

Fig. 9.4 The ST depression caused by Digoxin as seen on the ECG

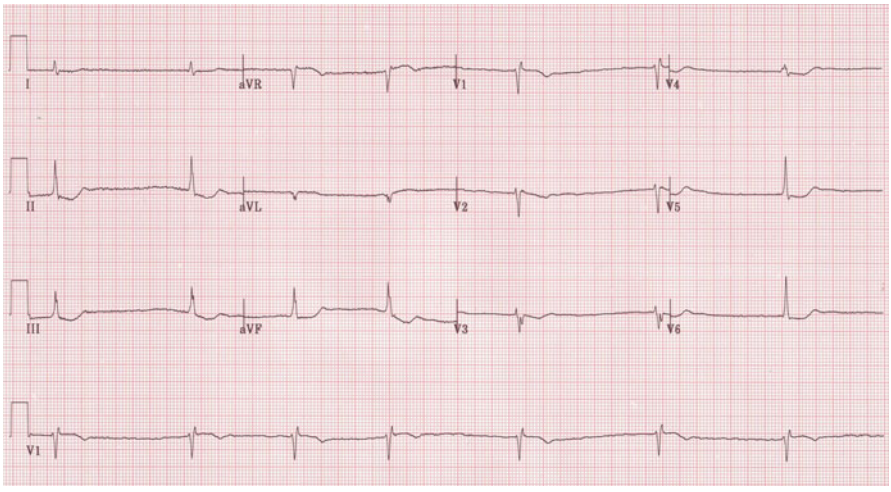

Fig. 9.5 Digitalis effect

is the presence of wide spread ST depression that resembles a 'reverse tick' in appearance, or a gradual sloping, known as the digitalis effect (Figs. 9.4 and 9.5).

It is important to understand that some of the ECG characteristics are present at therapeutic levels of Digoxin use. Arrhythmias and inverted T waves tend to appear at toxic levels. Distinguishing between therapeutic and toxic levels cannot be done using an ECG alone but the ECG findings coupled with the patient's medical history

could be helpful in determining if the patient is taking Digoxin. These sorts of clues are especially useful when dealing with confused patients or patients who cannot remember what medication they are prescribed or have taken.

ECG Characteristics of Digitalis
- ST depression (Digoxin effect)
- A slower atrial and ventricular rate
- Reduced T wave amplitude
- Shortened QT interval
- Arrhythmias
- Inverted T waves

Pulmonary Embolism (PE)

Is usually caused by a blood clot migrating from one part of the body to the lungs (Fig. 9.6). One of the primary reasons patients suffer from PE is secondary to a DVT (Deep Vein Thrombosis) a blood clot in the deep veins, usually in the legs. Prolonged bed rest, inactivity and long haul flights can increase the risk of developing a DVT. Patients often present with acute chest pain so an ECG is usually one of the first investigations to be carried out. If the chest pain is not found to be caused by an acute cardiac event there may be signs on the ECG that point towards PE.

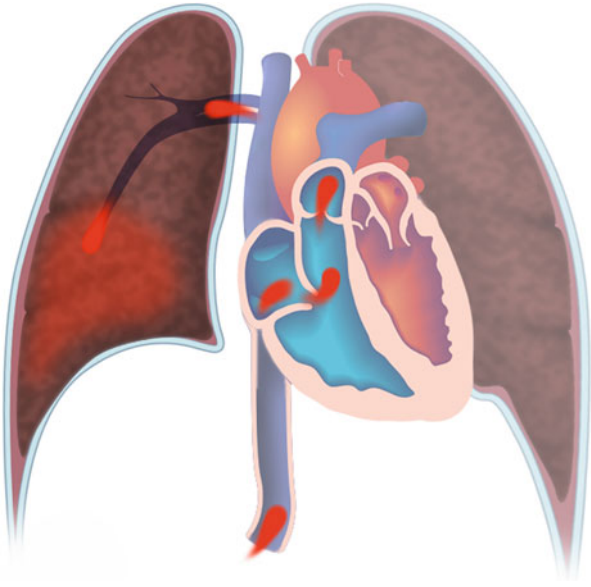

Fig. 9.6 Clot migration from lower body to the lungs causing a PE

The PE has to be large to show up on the ECG. A small PE may not show up on the ECG at all. The ECG should be used as a guide to diagnosis only. PE diagnosis is usually confirmed by bloods tests showing a raised D-dimer and subsequent CTPA (CT Pulmonary Angiography).

Primary Investigations for PE Diagnostic Confirmation
- Raised D-dimer
- CTPA (CT Pulmonary Angiography) visualising the PE

Treatment of PE
- O_2 therapy
- Analgesia
- Anti-coagulation

ECG Characteristics of PE
- Sinus tachycardia
- Complete or incomplete RBBB
- T wave inversion in leads V1 – V3
- An S1 Q3 T3 pattern (S wave in lead I, Q wave in lead III and T wave inversion in lead III)
- Right atrial enlargement
- Right axis deviation

Summary of Key Points

- Electrolyte imbalances cannot be diagnosed by the ECG alone and always require further investigation. The ECG may however be an aid in diagnosis.
- It is important to distinguish between the toxic and therapeutic effects of Digoxin in patients taking the drug.
- Hypokalemia can predispose patients taking Digoxin to Digoxin toxicity.
- Electrolyte imbalances require prompt investigation and treatment.
- Non cardiac specific conditions may cause ECG changes, prompting further investigation.

Quiz

Q1. One of the ECG characteristics associated with hyperkalemia is…

(A) *Prominent U waves*
(B) *Tall peaked P waves*
(C) *Tall peaked T waves*

Q2. One of the ECG characteristics associated with hypokalemia is…

(A) *Prominent U waves*
(B) *Tall peaked T waves*
(C) *Bifid 'notched' P waves*

Q3. A Pulmonary Embolism can always be diagnosed using an ECG alone

(A). *True*
(B) *False*

Q4. Hypokalemia can predispose patients…

(A) *Who are taking Aspirin to suffer from gastric ulcers*
(B) *Who are taking Ramipril to renal failure*
(C) *Who are taking Digoxin to Digoxin toxicity*

Q5. J waves can often be seen on ECGs of patients suffering from…

(A) *Dextrocardia*
(B) *Hypothermia*
(C) *Hyperthermia*

Q6. Electrolyte imbalances are always caused by drugs

(A) *True*
(B) *False*

Answers: Q1 = C, Q2 = A, Q3 = B, Q4 = C, Q5 = B, Q6 = B

Chapter 10
Acute Coronary Syndromes

Abstract Although the heart pumps blood around the entire body, it too requires a supply of blood, oxygen and nutrients to perform its action as a pump. The heart gets its blood supply from the coronary arteries (Fig. 10.1).

Background

Although the heart pumps blood around the entire body, it too requires a supply of blood, oxygen and nutrients to perform its action as a pump. The heart gets its blood supply from the coronary arteries (Fig. 10.1).

There are two coronary arteries: the right coronary artery which supplies blood to the inferior (bottom) part of the heart and the left coronary artery, which branches off into two further arteries, known as the left anterior descending artery, which supplies blood to the anterior (front) portion of the heart and the circumflex artery which supplies the lateral (side) of the heart.

Atherosclerosis

Is a process leading to a blockage or narrowing of the arteries. 'Fatty streaks' composed of cholesterol are deposited on the arterial walls. Although this is a normal process associated with aging, in some individuals there is an excess increase of cholesterol that can lead to Coronary Heart Disease (CHD). This process can also lead to thrombosis, which is the formation of a blood clot within the arteries (Fig. 10.2).

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A. Davies, A. Scott, *Starting to Read ECGs*,
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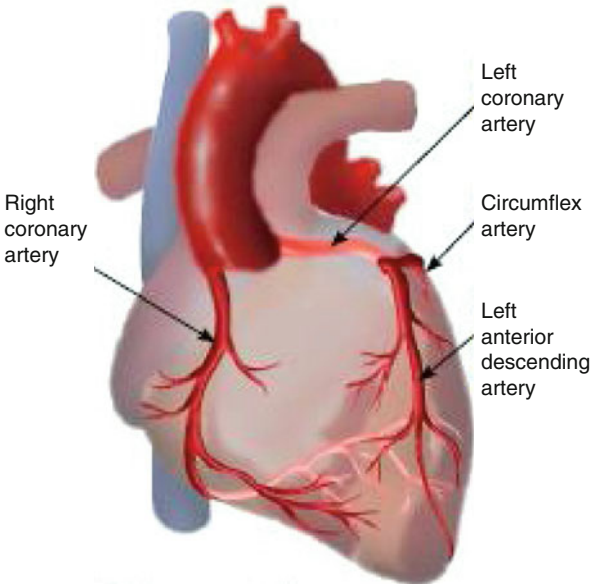


Fig. 10.1 The coronary arteries

Fat is deposited on the walls of the artery, a fibrous cap then forms over the top of the lipid core. The cap can then become dislodged exposing the inner core. As a result of this, platelets aggregate (collect) forming a mesh trapping blood cells leading in turn to the formation of a blood clot.

Modifiable and Non-modifiable Risk Factors for CHD

There are factors contributing to CHD that are non-modifiable (Table 10.1). These are things a person has no control over, such as their age and race. Males are more likely to develop CHD than women. The incidence increases with age. Certain groups, such as African Americans are more prone to CHD. There is also a genetic link with people with parents suffering from CHD are more likely to suffer problems themselves. Contrasting this there are also modifiable risk factors, these are things that can be altered, usually through making lifestyle changes. Leading an active life, eating a well-balanced diet and not smoking are all things people can do to reduce their risk of CHD.

Angina

A partial blockage of a coronary artery may cause angina pectoris. This typically presents as retrosternal (behind the sternum) chest pain. This can also be accompanied by sweating, pain in the neck, jaw and/or arms. Many patients describe it as a 'tightness' in the chest.

This partial blockage of the artery reduces the flow of blood to the section of the heart muscle beyond the narrowing. This means not enough blood and oxygen can

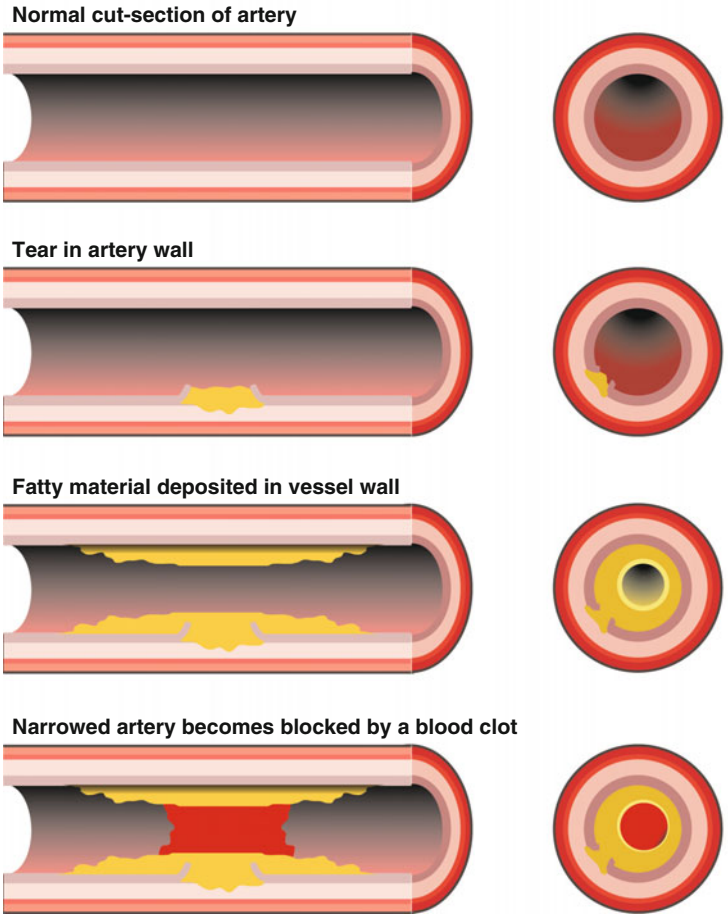


Fig. 10.2 Atherosclerosis

Table 10.1 Non-modifiable and modifiable risk factors contributing to CHD

Non-modifiable risk factors	Modifiable risk factors
Age	Smoking
Race	High blood cholesterol
Sex	High blood pressure
Genetics	Obesity and inactivity
	Diabetes

reach that part of heart muscle. This lack of oxygen and nutrients to cells is called ischemia. Angina can be split into two main subgroups:

- Stable angina
- Unstable angina

Stable angina is a type of angina that is normally felt only when the patient exerts themselves physically. At rest the reduced blood flow does not cause too much of a

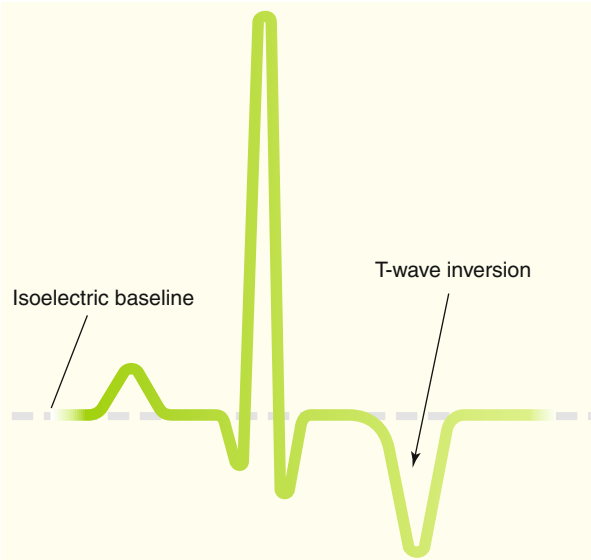


Fig. 10.3 T wave inversion

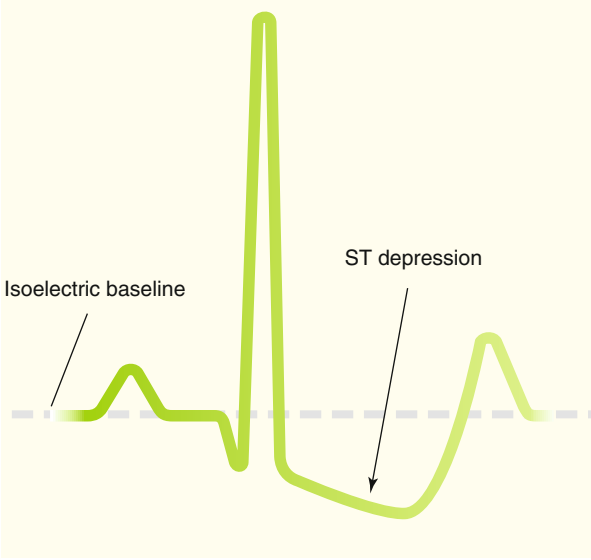
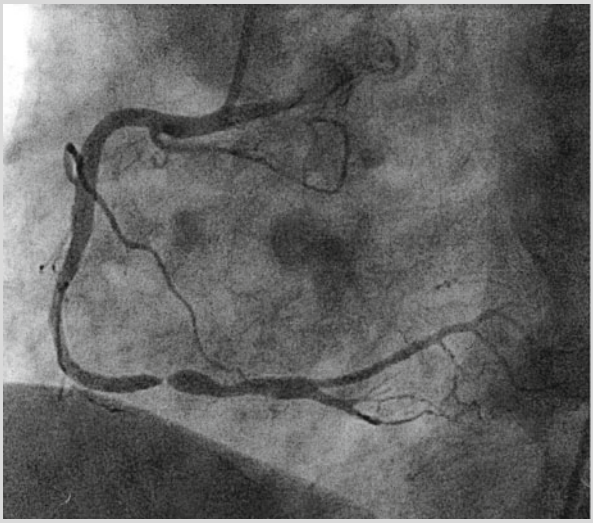


Fig. 10.4 ST depression

problem. When there is a demand for more blood and oxygen to the heart during physical activity, pain is felt as the blood/oxygen cannot be delivered quickly enough. Supply does not meet demand. Unstable angina occurs when the blockage is so severe that the pain can be felt on minimal or no activity at all. This can be an acute emergency.

Angina itself cannot be seen on an ECG. Examination of the ECG will often however show signs of cardiac ischemia. This can be seen on the ECG as either T wave inversion (T waves are upside down/flipped) as seen in Fig. 10.3 or ST depression where the ST segment is depressed below the Isoelectric baseline (Fig. 10.4).

Angiography
Confirmation of arterial narrowing (stenosis) is found by carrying out a cardiac catheterisation (angiogram). A procedure where plastic catheters are fed into the coronary arteries. Access is usually gained through the radial or femoral artery. The catheters are positioned by x-ray camera (fluoroscopy). A contrast medium is then injected into the arteries allowing doctors to see any blockages or narrowing of the arteries.



The image above shows a blockage in the Right Coronary Artery.

Acute Coronary Syndromes (ACS)

Narrowing or obstruction of any part or parts of the coronary arteries causing acute symptoms can be described as an acute coronary syndrome.

These include:

- ST Elevation Myocardial Infarction (STEMI)
- Non ST Elevation Myocardial Infarction (NSTEMI)
- Unstable angina

STEMI

An ST elevation Myocardial Infarction (heart attack) is defined by the raising of the ST segment on the 12-lead ECG in more than one lead coupled with the history of the presenting complaint and the patient's presentation.

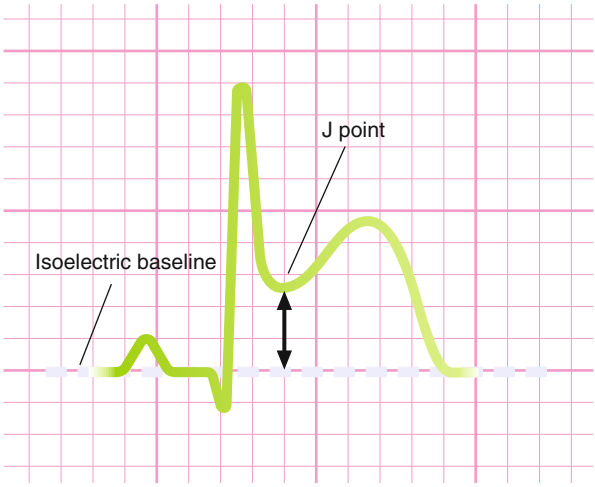


Fig. 10.5 ST elevation

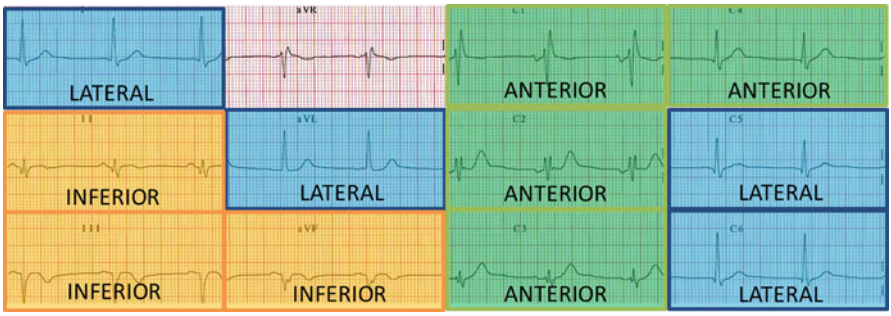


Fig. 10.6 The ECG split into regions (inferior, lateral and anterior)

As seen in the Fig. 10.5 the ST segment is elevated above the baseline. This is measured in mm from the isoelectric baseline to the J point. Sometimes the elevation can be so pronounced that the T wave and QRS complex appear to merge. ST elevation is not only seen in acute MI patients. The main way of telling the difference is based on a combination of factors, including patient history, presenting condition and ECG findings. When ST elevation is associated with an MI the elevation is seen in more than one lead. The 12-lead ECG is split into regions as seen in Fig. 10.6 and Table 10.2.

This is one reason why a 12-lead ECG is a vital test for patients with chest pain. The patient could have elevation in leads V_1, V_2, V_3 and V_4 making it an anterior MI, but if the patient was only being monitored with a single lead ECG, say lead II, there may be no elevation at all in that lead and the heart attack could go undiagnosed.

Table 10.2 The regions of the heart

Leads	Colour Code	Area of Heart (region)	Probable Artery Involved
II, III and aVF	Orange	Inferior (bottom)	Right Coronary Artery (RCA)
I, aVL, V5 and V6	Blue	Lateral (side)	Circumflex artery (Cx)
V1, V2, V3, V4	Green	Anterior (front)	Left Anterior Descending (LAD)

NB: Sometimes due to anatomical variance the artery involved does not always correspond to the ECG region

Causes of ST-Segment elevation
- Acute Myocardial Infarction (MI)
- Acute Pericarditis
- High take off
- Brugada syndrome
- Left ventricular aneurysm
- Prinzmetals angina
- Normal variant

There may also be combinations of regions involved. If there was for example elevation in the inferior and lateral leads this would be an inferiolateral MI or anteriolateral MI if it were the anterior and lateral leads that were involved.

Common Symptoms of Myocardial Infarction
- Chest pain possibly spreading to the neck, jaw and left arm
- Feeling or being sick
- Shortness of breath
- Clammy pallor
- Light headedness and dizziness
- Less commonly may present as dull pain
- or epigastric pain (sometimes in women)

Immediate Management of Chest Pain Patient
- Sit/lay patient down if standing
- Reassure patient
- High flow oxygen
- 12-Lead ECG
- Obtain a full history of the pain including;
- location, severity, what makes it worse/better, radiation and pain score.
- Analgesia

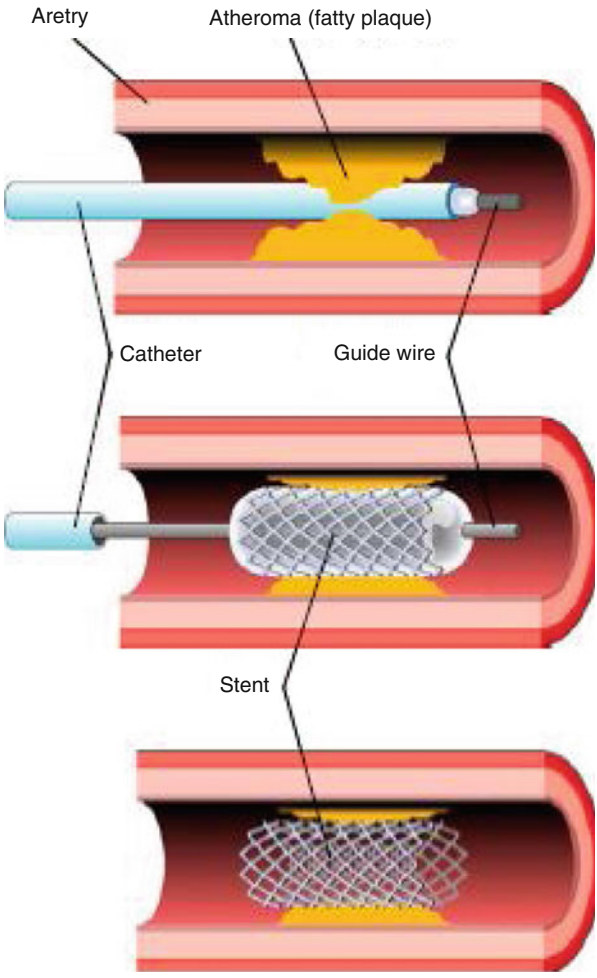


Fig. 10.7 Percutaneous coronary intervention

There is a commonly used phrase in cardiology: 'time is muscle' which equates to the fact that if a patient is having an MI, the more time that passes the more heart muscle will be damaged. Prompt identification and treatment of an MI is essential. The treatment of choice where possible is now primary PCI (Percutaneous, Coronary, Intervention). This involves using small balloons and metal cages called 'stents' to open and reinforce the blocked or narrowed artery (Fig. 10.7).

This procedure is carried out on conscious patients without general anaesthetic in cardiac catheterisation labs. On occasions where there is no access to a catheter lab, thrombolytics as still used. These are powerful drugs which are used to break down clots. The disadvantage is they may remove the clot but they don't treat the underlying narrowing in the artery as a PCI does.

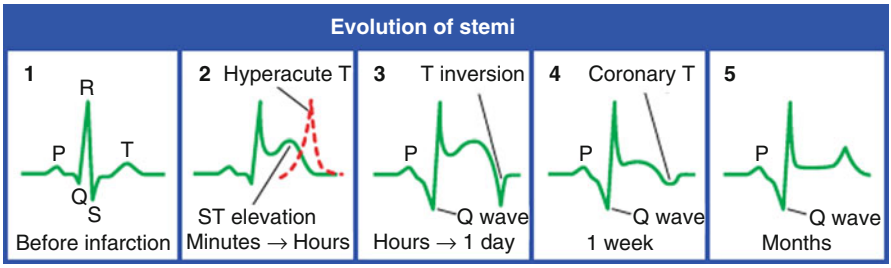


Fig. 10.8 The changes taking place to the PQRST waves over time during a STEMI

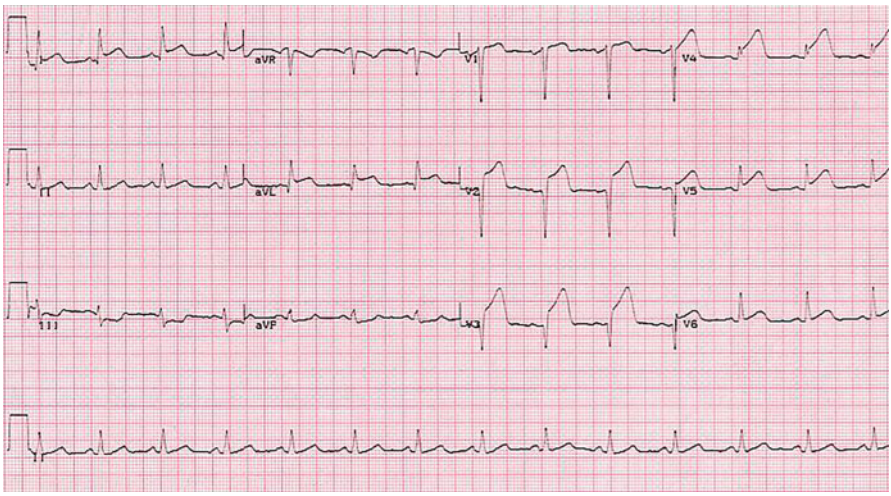


Fig. 10.9 Anterolateral STEMI

Evolution of STEMI

As time passes the STEMI is said to evolve going through various stages that can be clearly seen on the ECG (Fig. 10.8).

Within minutes to hours after the start of a STEMI the ST segment begins to rise. Alternatively hyperacute T-waves can be seen. These are large tall pointed T waves. After several hours to a day the T wave is inverted and the Q wave deepens. Q waves are likely to be pathological when occurring in more than one lead, deeper than two small squares (2 mm) and/or 25 % the height of the R wave in depth. Within a week the elevation is often reduced leaving an inverted T wave and deep Q wave. Months later the ECG may look quite normal, with the exception of the deep Q waves seen in the region of the ECG where the heart attack occurred (Figs. 10.9, 10.10, and 10.11).

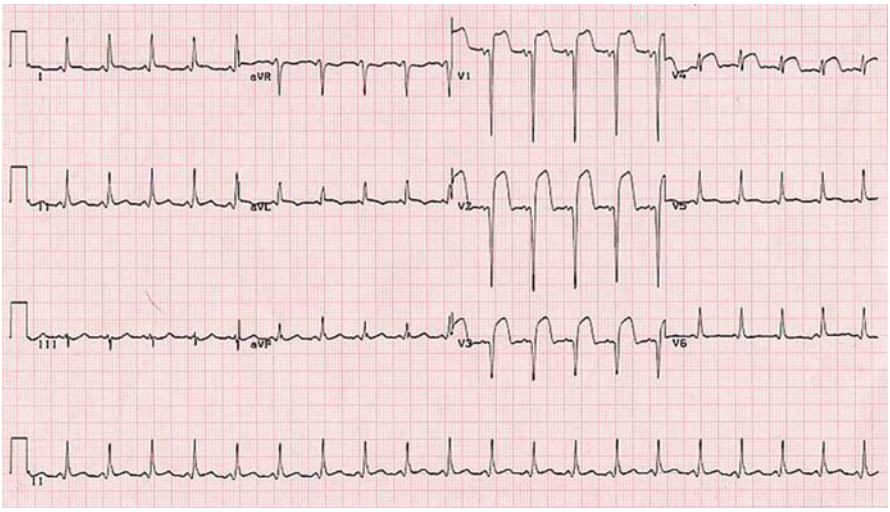


Fig. 10.10 Anterior STEMI

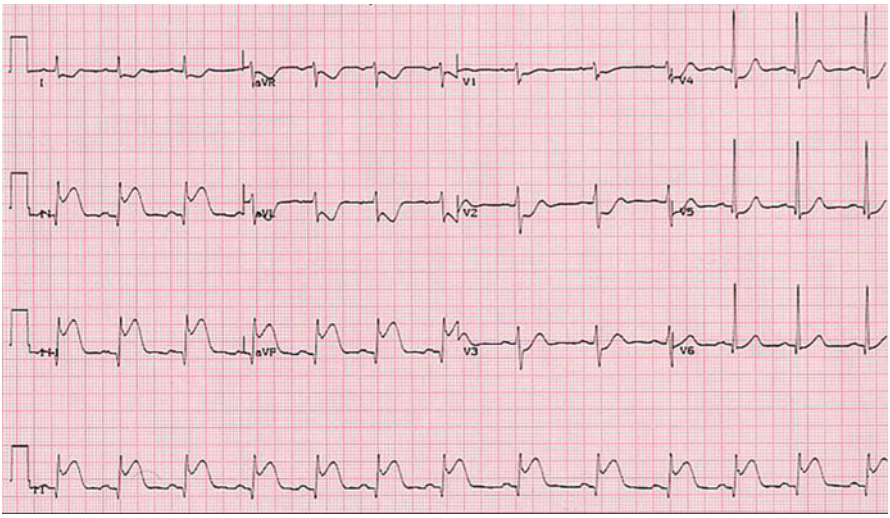


Fig. 10.11 Inferior STEMI

NSTEMI

Sometimes a patient presents with all the symptoms of a heart attack but there is no ST elevation or pathological Q waves on the ECG. The ECG often however shows signs of ischemia (ST depression or T wave inversion), although not always. Where a heart attack is suspected, blood can be taken to check levels of a protein called

Troponin T, or Trop T for short. If this level is raised it can point to a heart attack alongside other clinical observations. Trop T can also however be raised for other reasons such as a tachycardia like AF or renal problems. Often an angiogram is performed during the patients stay to check for underlying CHD.

Left Bundle Branch Block (LBBB) and Chest Pain

A LBBB normally makes it extremely difficult to see signs of ischemia and elevation. As such if the patient has a new LBBB and is experiencing chest pain they should receive immediate treatment, usually primary PCI to investigate and treat the underlying cause.

Warning: Chest Pain and LBBB

If a patient is experiencing chest pain and has a newly diagnosed LBBB, this should be treated as medical emergency. The patient could be having a Myocardial Infarction and should be treated as such.

Summary of Key Points

- There are three types of Acute Coronary Syndrome, consisting of; NSTEMI, STEMI and unstable angina
- Primary PCI is the current gold standard of treatment for patients presenting with STEMI
- Cardiac enzymes such as Troponin T are used to aid diagnosis in NSTEMI patients
- Prompt treatment of a MI can reduce damage caused to the heart muscle
- Chest pain and a new LBBB should be considered a potential medical emergency and receive prompt treatment
- There are risk factors increasing the risk of developing CHD that are both modifiable and non-modifiable. Good patient education and lifestyle changes can reduce the risk of a patient developing CHD.

Quiz

Q1. A patient presenting with acute chest pain and a new Left Bundle Branch Block (LBBB) should…

(A) *Be treated as a potential medical emergency*
(B) *Can wait for routine medical review at the next ward round*

Q2. ST elevation in leads II, III and aVF would indicate the patient may have…

(A) *An anteriolateral STEMI*
(B) *An inferior STEMI*
(C) *An anterior STEMI*

Q3. The gold standard treatment for patients presenting with a STEMI is…

(A) *Thrombolytic drugs*
(B) *Primary PCI*
(C) *Bed rest*

Q4. Flipped T waves and ST segment depression are signs of…

(A) *Complete Heart Block (CHB)*
(B) *Myocardial ischemia*
(C) *WPW syndrome*

Q5. Raised Troponin T is always a sign of an NSTEMI

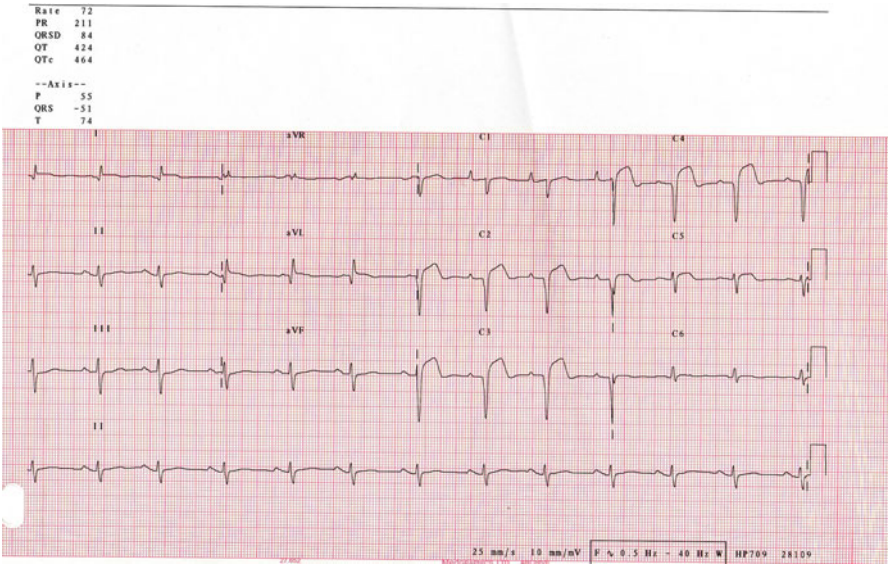
(A) *True*
(B) *False*

Q6. Modifiable risk factors for CHD are…

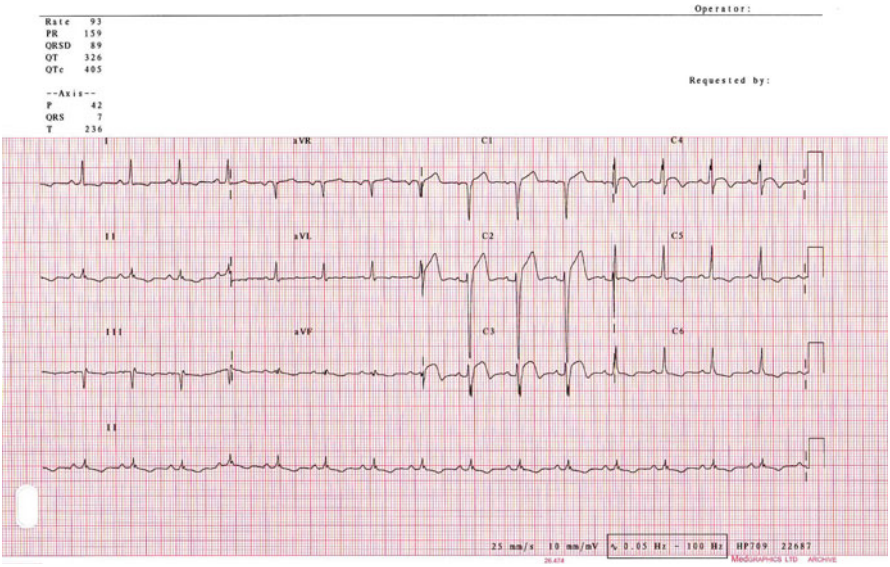
(A) *Risk factors that cannot be changed*
(B) *Risk factors that can be reduced by making changes*
(C) *Not a factor in CHD*

Now interpret the following ECGs:

Q7.



Q8.



Answers: Q1 = A, Q2 = B, Q3 = B, Q4 = B, Q5 = B, Q6 = B, Q7 = Probable anterior lateral STEMI, Q8 = Probable anterior STEMI.

The Authors

Alan Davies graduated from Birmingham City University with a first class honours degree. He also won a first place prize for adult nursing student in the inaugural Health Care Awards. At the time of writing this book, Alan Davies was working as a nurse in the cardiac catheterisation labs at the Royal Devon and Exeter Hospital. Alan has experience dealing with routine and emergency angiograms including

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primary PCI. Alan has also assisted in pacemaker/AICD insertion, transoesopheageal echo (TOE), cardioversion and electrophysiological studies. He has also authored three articles, two concerning ECGs and one about permanent pacemakers, which were published in the *British Journal of Nursing* and the *British Journal of Cardiac Nursing*, respectively. Alan also has a post-graduate degree module in ECGs and arrhythmias.

Alwyn Scott graduated from Birmingham University and has worked in the NHS since 1995. He is currently employed as Senior Staff Nurse for the Cardiology High Dependency Department at Papworth Hospital in Cambridgeshire. His role is predominantly that of working with patients who are in the acute stages of having heart attacks both pre and post primary PCI. Alwyn is also heavily involved in staff education. Alongside specialist knowledge in acute cardiac nursing and ECG interpretation, Alwyn also has a vast experience in emergency care, working in Accident and Emergency departments and also frontline for the ambulance service responding to 999 calls.

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