Electrocardiography (ECG)

Learning Objectives.

At the end of this section, you should be able to :

1. **Describe the electrical conduction pathway in the heart**
2. **Understand the basic principles of how an ECG functions.**
3. **Know the placement positions for a 12 lead ECG.**
4. **Interpret a basic, normal ECG trace, and identify the different components which make this up.**

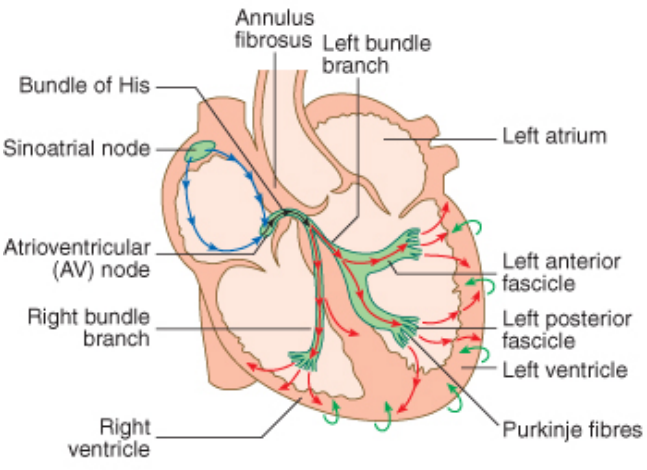
NB – there are no key learning points indentified in this short course as the questions posed in the accompanying workbook act as the key learning points.

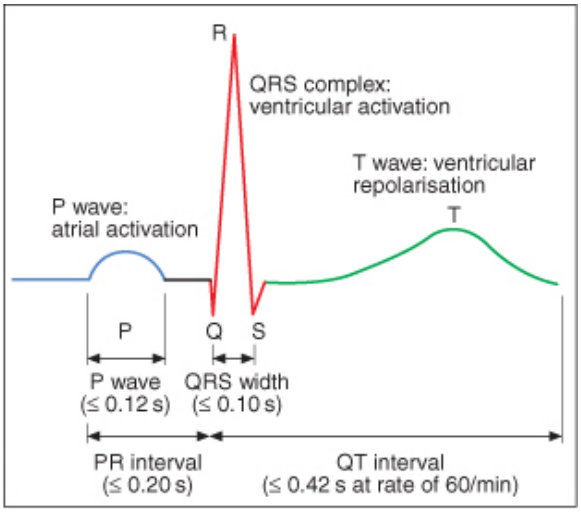
ECG is used to determine the cardiac rhythm and the condition of the conducting tissues. Information is also gained about chamber size and the presence of myocardial ischaemia and infarction, and about the effects of some drugs on the heart.

The fundamental basis for ECG is that the electrical activation of a heart muscle cell (cardiomyocyte) causes a depolarisation of its membrane. The depolarisation is propagated along the length of the cell or fibre, and transmitted to adjoining cells. The result is a moving wave front of depolarisation, which passes through the heart and sets up electrical currents; these can be detected by surface electrodes, amplified and displayed as the ECG.

From the electrical point of view, the heart acts as if it has only two chambers because the two atria and then the two ventricles contract together. In the electrical conduction system, the sinoatrial node is situated at the junction of the superior vena cava and right atrium, and is the origin of the impulses responsible for heart rhythm under normal conditions ('sinus rhythm'). Depolarisation of the sinoatrial node triggers a wave front of depolarisation which travels through the atria. Conduction directly to the ventricles is prevented by the annulus fibrosus, which insulates the atria from the ventricles.

The atrioventricular (AV) node, which is normally the only route of conduction from the atria to the ventricles, is situated beneath the right atrial endocardium at the lower end of the interatrial septum; it conducts slowly and regulates the frequency of conduction to the ventricles. The bundles of His passes from the AV node through the annulus fibrosus and divides into right and left bundle branches, which pass down the respective sides of the ventricular septum, radiating out as the Purkinje network. The left bundle branch is subdivided into anterior and posterior fascicles. Injury to one of the main bundles may manifest on the ECG as right or left bundle branch block, whereas selective injury of one of the left fascicles (hemiblock) produces deviation of the electrical axis.

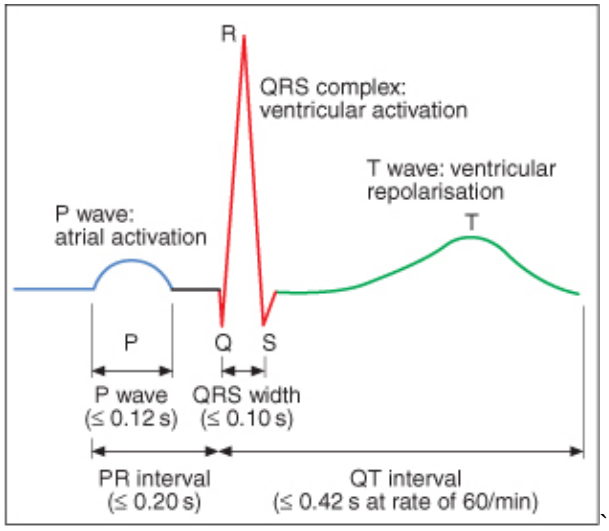




Normal conduction cycle : the blue, red and green sections of the trace on the right correlate with the areas of electrical activity show in the diagram on the left.

The standard 12-lead ECG

Normally, cardiac activation starts in the sinoatrial node, but this cannot be detected on the ECG. Depolarisation then spreads through the atria, creating the P wave and triggering atrial contraction. The PR interval represents the delay from the onset of atrial depolarisation to the onset of ventricular depolarisation. Electrical activity then spreads rapidly through the bundle of His and the bundle branches, triggering ventricular contraction and creating the QRS complex. The muscle mass of the ventricles is much larger than that of the atria, and the QRS complex is therefore correspondingly larger than the P wave. Repolarisation is a slower process that occurs from the epicardium to the endocardium, and produces the T wave. The QT interval represents the total duration of ventricular depolarisation and repolarisation.



The 12-lead ECG is generated from chest and limb electrodes which view the heart from different directions. There are four limb electrodes: one on each wrist and one on each ankle, connected to a central terminal which is electrically neutral. The

signal recorded from the electrode on the left arm is augmented relative to the central terminal and is therefore designated lead aVL (a = augmented). Similarly augmented signals are obtained from the right arm (aVR) and left leg (aVF). These leads record the electrical activity of the heart within the frontal plane, with each lead 120° apart. Readings for leads I, II and III (the bipolar leads) are generated by the difference in potential between two adjacent electrodes. Lead I is the difference between the left arm and the right arm, lead II is the difference between the left leg and the right arm, and lead III is the difference between the left leg and the left arm. By convention lead I is designated as 0° within the frontal plane axis. The other leads are referenced from this point so that aVF becomes +90°, aVL -30°. Further description of lead placement and designation can be found later in this course.

When depolarisation spreads towards an electrode it produces a positive deflection in that lead; when it moves away a negative deflection is registered. The principal direction of depolarisation in the heart is known as the electrical vector or axis. When the vector is at right angles to a lead, the depolarisation in that lead is equally negative and positive (isoelectric).

There are six chest leads, V_1-V_6, from electrodes placed on the anterior and lateral side of the chest over the heart. Leads V_1 and V_2 lie approximately over the right ventricle, leads V_3 and V_4 over the interventricular septum, and V_5 and V_6 over the left ventricle. The left ventricle has the greater muscle mass and contributes the major component of the QRS complex. Depolarisation of the interventricular septum occurs first and moves from left to right; this generates an initial negative deflection in V_6 (Q wave) and an initial positive deflection in V_1 (R wave). The second phase of depolarisation is activation of the body of the left ventricle, which creates a large positive deflection or R wave in V_6 (with reciprocal changes in V_1). The third and final phase of depolarisation involves the right ventricle and produces a small negative deflection or S wave in V_6.

The ECG in infarction and ischaemia.

When an area of the myocardium is ischaemic or undergoing infarction, repolarisation and depolarisation become abnormal relative to the surrounding myocardium. In transmural infarction there is initial ST segment elevation (the current of injury) in the leads facing or overlying the infarct; Q waves (negative deflections) will then appear as the entire thickness of the myocardial wall becomes electrically neutral relative to the adjacent myocardium. In myocardial ischaemia the ECG typically shows ST segment depression and/or T wave inversion; it is usually the subendocardium which most readily becomes ischaemic. Other conditions, such as left ventricular hypertrophy and electrolyte disturbances, can cause similar ST and T wave changes.

Exercise (stress) ECG

A 12-lead ECG is recorded during exercise on a treadmill or bicycle ergometer. The limb leads are placed on the shoulders and hips rather than the wrists and ankles. The “Bruce Protocol” has been well validated and is the most widely used test format for treadmill testing. Blood pressure is recorded and symptoms assessed regularly throughout the test. A test is 'positive' if anginal pain occurs, blood pressure falls or fails to rise, or there is ST segment shifts of > 1 mm. The results of an exercise tolerance test (ETT) are not always conclusive. Some patients with a negative test will have underlying coronary disease (false negative) and, conversely, some with a positive test will not have coronary disease (false positive). Exercise testing is an unreliable population screening tool because in low-risk individuals (e.g. asymptomatic young or middle-aged women) an abnormal response is more likely to represent a false positive than a true positive test. In patients with symptoms suggestive of angina, exercise testing has much better sensitivity and specificity, and is clinically very useful. Stress tests are contraindicated in the presence of un-stable angina, decompensated heart failure and severe hypertension.

Ambulatory ECG (Holter monitoring)

Continuous recordings of one or more ECG leads may be obtained by attaching them to a small portable solid state or tape recorder for 24 hours or more. This

technique is useful for detecting transient episodes of arrhythmia or ischaemia, which seldom occur fortuitously during the short time taken for routine 12-lead ECG recordings. A variety of hand-held or implantable patient-activated devices can be used to record the ECG during symptomatic episodes and are particularly suitable for investigating patients with infrequent but potentially serious symptoms. Many of these devices have the facility to transmit ECG recordings to a cardiac centre through the telephone.

ECG Lead Placement

The system of positioning of leads for performing a 12-lead ECG is universal. This helps to ensure that, when a person's ECGs are compared, any changes on the ECG are due to cardiac injury, not a difference in placement of leads, which becomes extremely important with the increasing use of foreign travel. There are universal standards in place throughout the world. Interpretation of the findings can vary from doctor to doctor but methods for obtaining the information are the same the world over.

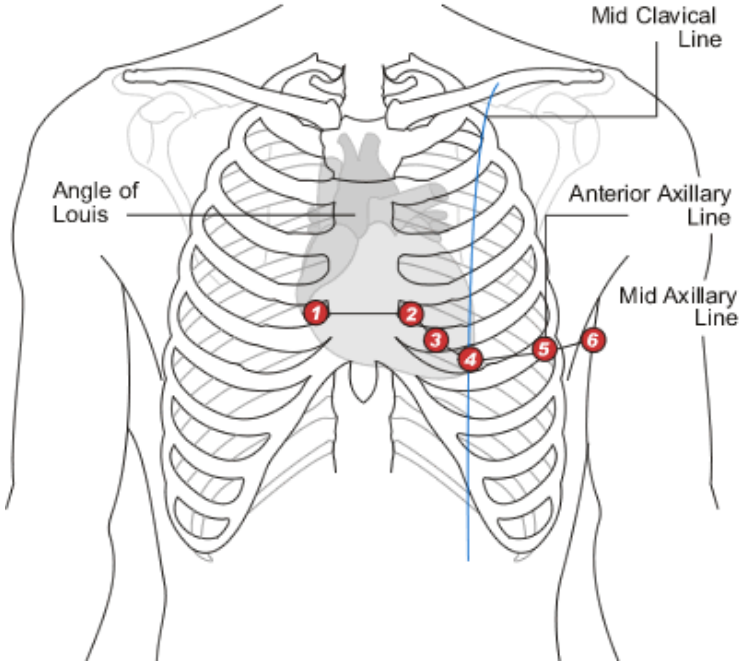
These positions may also differ if a patient is shaking (maybe due to Parkinson's Disease or hypothermia) or has muscle tremors. In this situation the leads may be moved onto the thighs and forearms.

Chest Leads

There are 10 wires on an ECG machine that are connected to specific parts of the body. These wires break down into 2 groups:

1. 6 chest leads
2. 4 limb or peripheral leads (one of these is "neutral")

The 6 chest leads are positioned as below:



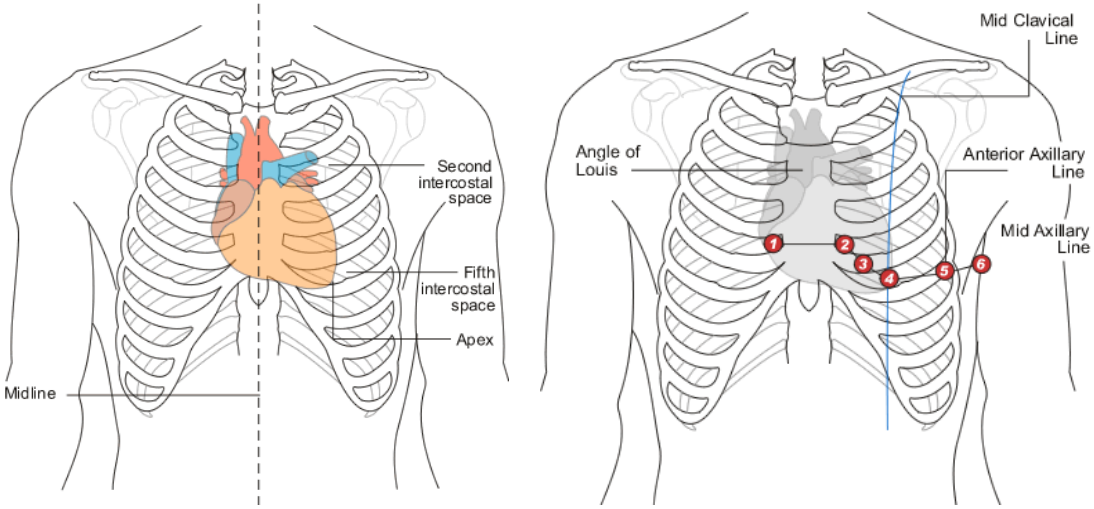
The 6 leads are labelled as "V" leads and numbered V1 to V6. They are positioned in specific positions on the rib cage. To position then accurately it is important to be able to identify the ***angle of Louis***, or ***sternal angle***.

This is found by placing the fingers gently at the base of the throat in a central position and moving the fingers downward until the top of the sternum is felt (sternal notch). From this position, the fingers are moved downward until a bony lump is felt - the angle of Louis. The angle of Louis is most easily found when the patient is lying down as the surrounding tissue is tighter against the rib cage.

From the angle of Louis, the fourth intercostal space on the right is located, and the area where is meets the sternum is the position for V1. V2 is placed in the corresponding area on the left.

The midclavicular line is identified, and V4 is placed in line with this in the fifth intercostal space. V3 is placed midway between V2 and V4. Following the 5th intercostal space laterally to a point in-line with the beginning of the axilla, V5 is placed. V6 is placed at the same level as V5, but on the mid-axillary line.

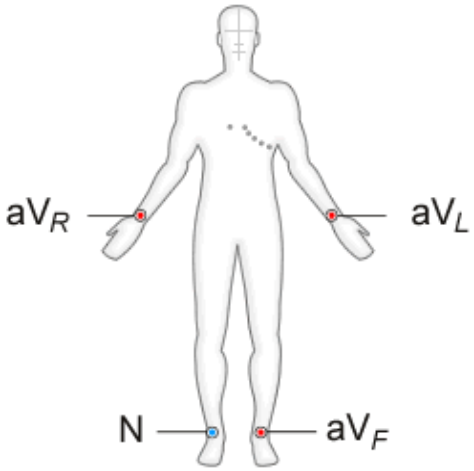
The picture below indicates the position of the heart in relation to the rib-cage, illustrating the relationship between heart position and lead placement.



Limb Leads

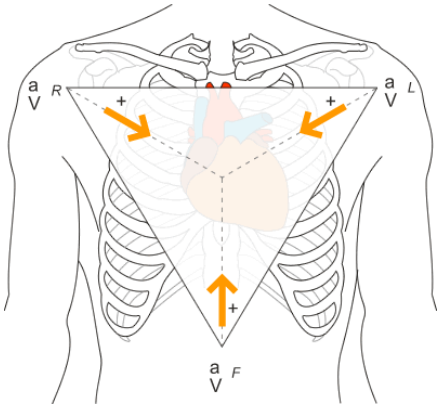
Limb leads are made up of 4 leads placed on the extremities: left and right wrist; left and right ankle.

The lead connected to the right ankle is a neutral lead. Its role is to complete an electrical circuit and plays no role in the ECG itself.



Unipolar Leads

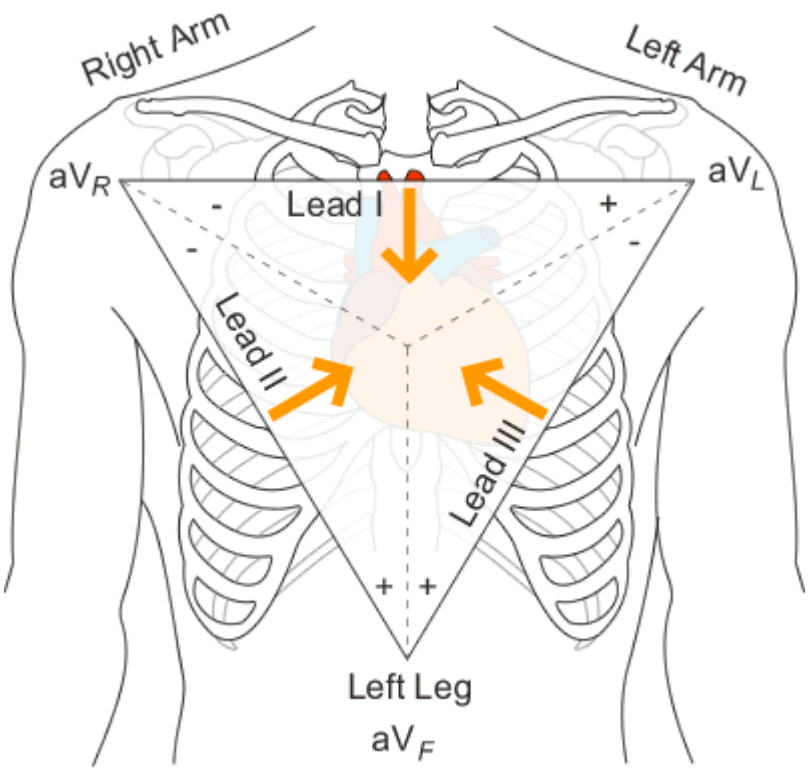
At this point, only nine leads have been discussed. They all look directly at the heart with tunnel vision, only giving information based on what is immediately in front of them. These nine wires are known as ***unipolar leads***. The three active peripheral leads are aVr, aVL, and aVf. These 3 leads create a triangle with the heart in the middle, as below. The lines into the centre indicate the line of sight of these leads.



Bipolar Leads

The 2 leads situated on the right and left wrist (or shoulders), aVr and aVL respectively, and the lead situated on the left ankle (or left lower abdomen) aVf, make up a triangle, known as ***Einthoven's Triangle***". Information gathered between these leads is known as ***bipolar***. It is represented on the ECG as 3 bipolar leads. Therefore:

- information between aVr and aVl is known as lead I
- Information between aVr and aVf is known as lead II
- Information between aVl and aVf is known as lead III



This now completes the 12 leads.

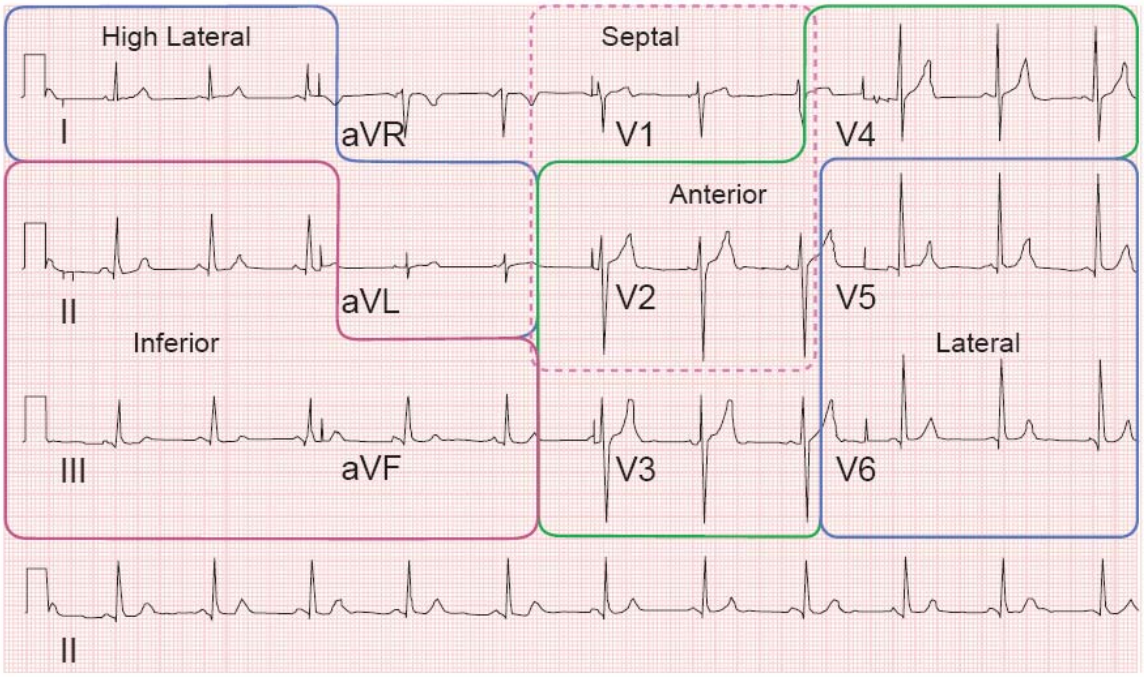
Regions of the Heart

It is important to know which regions of the heart each lead is looking at and what groups they make up.

- aVL is on the left wrist or shoulder and looks at the upper left side of the heart.
- Lead I travels towards aVL creating a second high lateral lead.
- aVf is on the left ankle or left lower abdomen and looks at the bottom, or inferior wall, of the heart.
- Lead II travels from aVr towards aVf to become a 2nd inferior lead
- Lead III travels from aVL towards aVf to become a 3rd inferior lead.

- V2 V3 and V4 look at the front of the heart and are the anterior leads.
- V1 is often ignored but if changes occur in V1 and V2 only, these leads are referred to as Septal leads.
- V5 and V6 look at the left side of the heart and are the lateral leads.

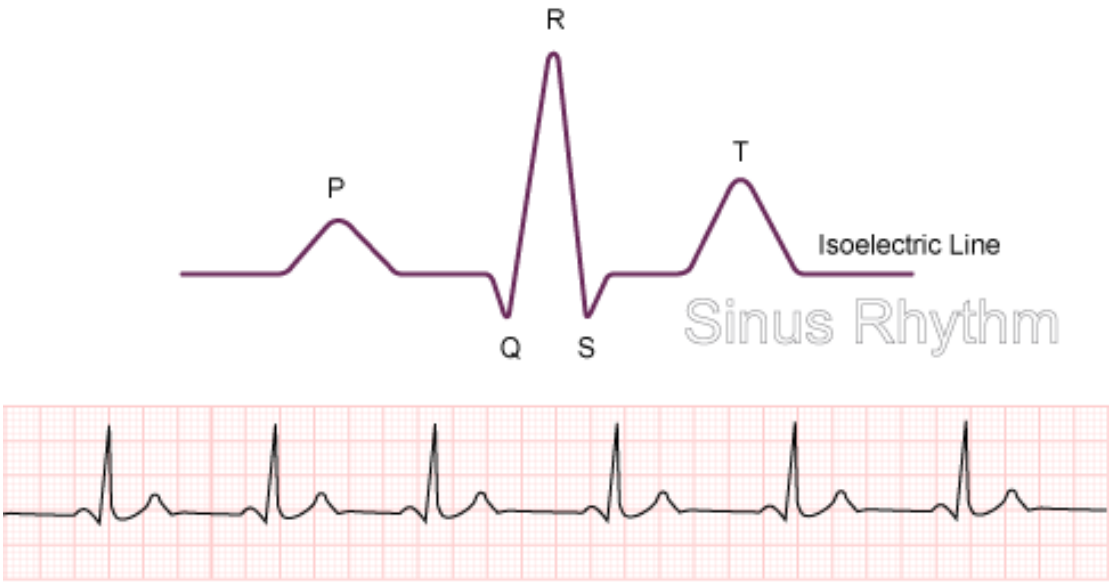
The ECG below shows where these leads are when printed.



Sinus Rhythm

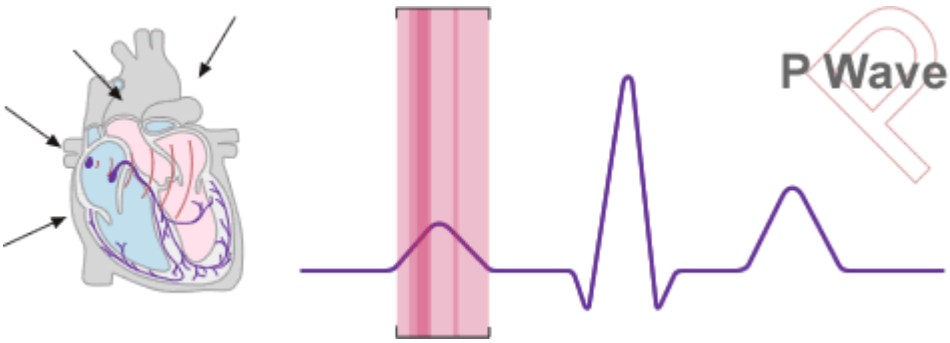
Sinus rhythm is the name given to the normal rhythm of the heart where electrical stimuli are initiated in the SA node, and are then conducted through the AV node and bundle of His, bundle branches and Purkinje fibres.

Depolarisation and repolarisation of the atria and ventricles show up as 3 distinct waves on ECG. A unique labelling system is used to identify each wave : P, Q, R, S, and T. The baseline voltage of the ECG is known as the isoelectric line, and is usually measured as the portion following a T wave and preceding the next P wave.



The more cells there are in an area, the more voltage is required. A the walls of the atria are thinner than those of the ventricles, more voltage is required, and therefore the size of the waves relative to these areas is different - less muscle means less cells, which means less voltage.

The P Wave



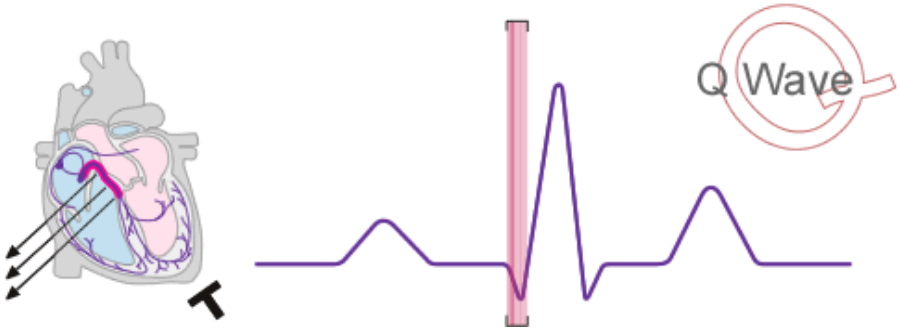
The first wave (P wave) represents atrial depolarisation. When the valves between the atria and ventricles open, 70% of the blood in the atria falls through with the aid of gravity, but mainly due to suction caused by the ventricles as they expand.

Atrial contraction is required only for the final 30% and therefore a relatively small muscle mass is required, and only a relatively small amount of voltage is needed to contract the atria.

After the first wave there follows a short period where the line is flat and is known as the **PR interval.** This is the point at which the stimulus is delayed in the bundle of His to allow the atria enough time to pump all the blood into the ventricles. It usually lasts for between 120 – 200ms, which corresponds to 3-5 small squares on the trace paper. An interval over 200ms may indicate a first degree heart block. A short PR interval may indicate a pre-excitation syndrome, such as Wolff-Parkinson-White syndrome. PR interval depression can indicate atrial injury or pericarditis.

As the ventricles fill, the growing pressure causes the valves between the atria and ventricles to close. At this point the electrical stimulus passes from the bundle of His into the bundle branches and Purkinje fibres. The amount of electrical energy generated is recorded as a complex of 3 waves known collectively as the **QRS complex**. Measuring the waves vertically shows voltage. More voltage is required to cause ventricular contraction and therefore the wave is much bigger.

The Q Wave

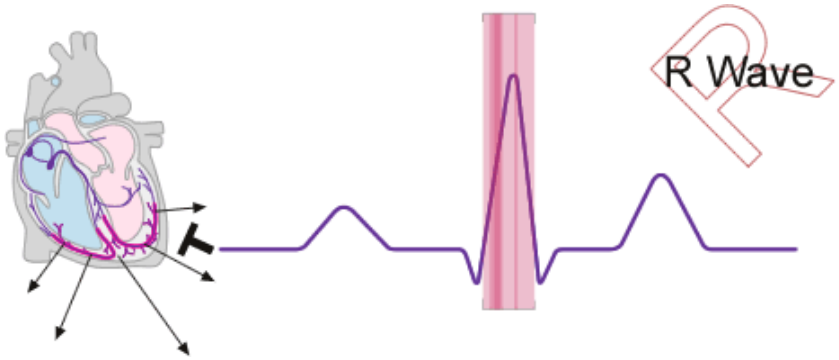


The diagram shows a small negative wave immediately before the large QRS complex. This is known as a Q wave and represents depolarisation in the septum.

Whilst the electrical stimulus passes through the bundle of His, and before it separates down the two bundle branches, it starts to depolarise the septum from left to right. This is only a small amount of conduction (hence the Q wave is less than 2 small squares on the trace paper), and it travels in the opposite direction to the main conduction (right to left) so the Q wave points in the opposite direction to the large QRS complex. Q waves greater than one third of the height of the R wave, or longer than 40ms, are considered abnormal and may indicate myocardial infarction.

The R Wave

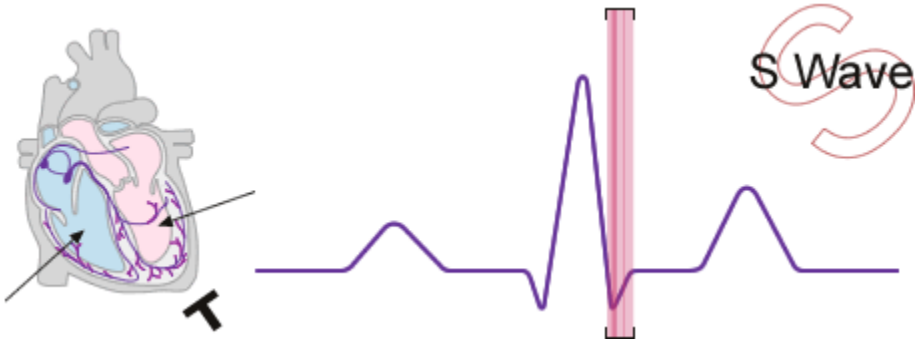
The QRS complex is made up of three waves. These waves indicate the changing direction of the electrical stimulus as it passes through the heart's conduction system. The largest wave in the QRS complex is the R wave.



The R wave represents the electrical stimulus as it passes through the main portion of the ventricular walls. The ventricle walls are very thick due to the amount of work they have to do and, consequently, more voltage is required. This explains why the R wave is by far the biggest wave generated during normal conduction - More muscle means more cells; more cells means more electricity; more electricity leads to a bigger wave.

The S Wave

The small negative wave following the large R wave is known as an S wave and represents depolarisation in the Purkinje fibres. The S wave travels in the opposite direction to the large R wave because, as can be seen on the earlier picture, the Purkinje fibres spread throughout the ventricles from top to bottom and then back up through the walls of the ventricles.



From the above information, it can now be understood how the QRS complex is broken down into 3 distinct waves:

- Q wave representing septal depolarisation
- R wave representing ventricular depolarisation
- S wave representing depolarisation of the Purkinje fibres.

The QRS complex lasts between 60 – 100 ms (three small squares or less), but any conduction abnormality will lengthen this space.

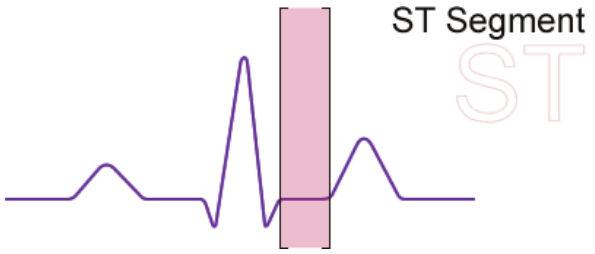
The T Wave

Both ventricles repolarise before the cycle repeats itself and therefore a third wave (T wave) is visible, representing ventricular repolarisation. The interval from the beginning of the QRS complex to the apex of the T wave is known as the absolute refractory period, whilst the last half of the T wave is known as the relative refractory period.

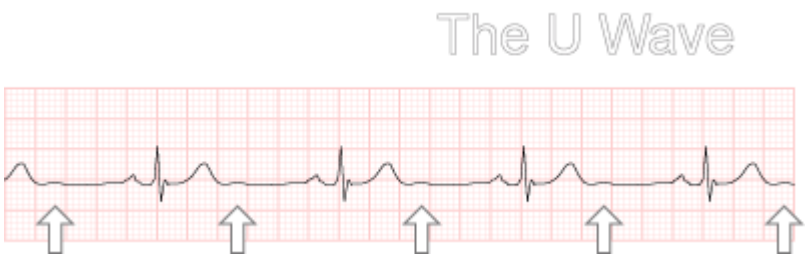
Inverted T waves can be a sign of coronary ischaemia, left ventricular hypertrophy, or a CNS disorder. Taller T waves may indicated high levels of potassium (hyperkalaemia), and flat T waves indicate hypokalaemia.

The ST Segment

There is a brief period between the end of the QRS complex and the beginning of the T wave where there is no conduction and the line is flat, lasting 80-120ms. This is known as the ST segment and it is a key indicator for both myocardial ischaemia and necrosis if it goes up or down. Normal ST segments have a slight upwards concavity, but flat or down-sloping ST segments may indicate coronary ischaemia, but elevate ones can be indication of myocardial infarction.



The U Wave

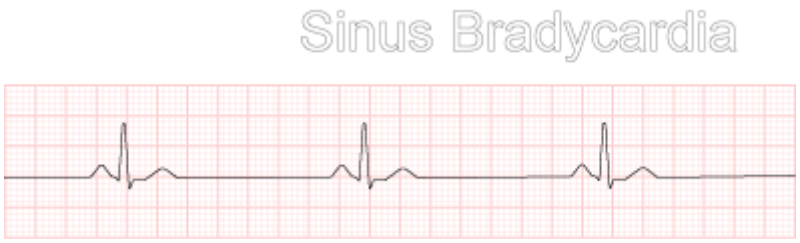


This is not always seen, but it is typically small, and follows the T wave. It is thought to represent repolarisation of the papillary muscles of Purkinje fibres. Prominent U waves are more frequently seen in hypokalaemia, but may also occur with hyper calcaemia, thyrotoxicosis, or exposure to digitalis, adrenaline, and Class 1a and 3 antiarrhythmics. An inverted U wave may represent myocardial ischaemia or left ventricular volume overload.

Sinus Bradycardia

The job of the heart is to pump blood around the body carrying oxygen and nutrients to organs, muscles and tissues, and transporting waste such as carbon dioxide to the lungs for expiration. The cardiovascular system is under the control of the nervous system which increases and decreases the workload of the heart depending on the body's requirements.

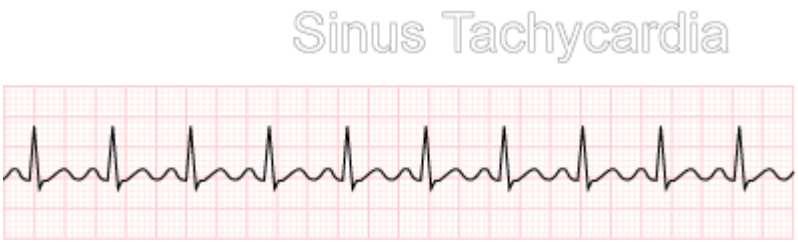
A heart rate below 60 beats per minute (bpm) is known as Bradycardia. Sinus Bradycardia is not a changed rhythm, it is simply normal sinus rhythm slowed down.



- R-R intervals constant and regular
- All waveforms are present, and there is 1 P-wave to each QRS complex
- The rate is <60bpm but not usually <40bpm
- Patients usually asymptomatic and no treatment is required
- Often caused by beta-blockers / calcium channel blockers
- May also be seen in athletes and occur during sleep.

Sinus Tachycardia

During exercise, it is common for a person's heart rate to go above 100bpm. This is known as Tachycardia and, if the rhythm is sinus, it is known as Sinus Tachycardia. Sinus tachycardia is not a different rhythm, it is simply sinus rhythm going faster than 100 bpm.



- R-R intervals constant and regular
- One P-wave per QRS complex
- All waveforms present
- Rate is > 100bpm, but not usually > 130bpm at rest
- Occurs normally in exercise / stress. Patient is usually asymptomatic.
- Other causes may be hypovolaemia / underlying medical problems

The passage of conduction through the heart in both sinus tachycardia and sinus bradycardia is exactly the same as in sinus rhythm. Both rhythms are naturally occurring but can also be brought on by an overdose of both legal and illegal drugs.

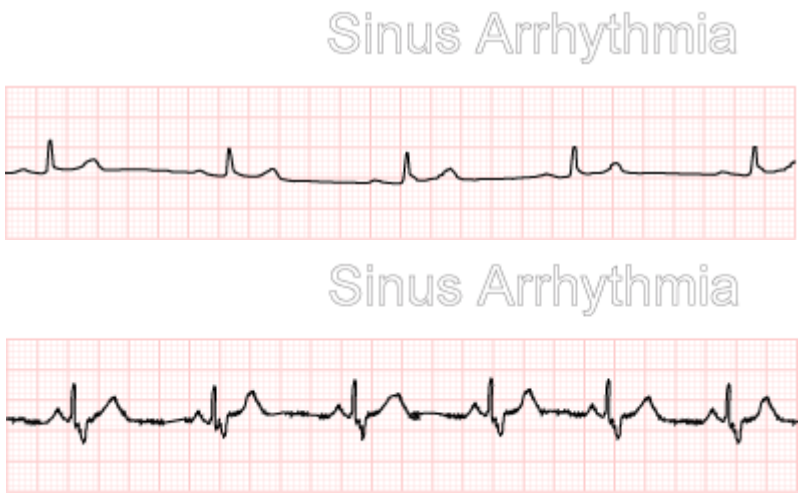
Sinus Arrhythmia

Sinus Arrhythmia, or regularly irregular sinus rhythm, is a variation on sinus rhythm where the P-P interval (the distance between consecutive P waves) varies by more than 10%. It can be naturally occurring or due to heart damage.

Causes can be:

- Respiratory - where the P-P interval lengthens and shortens with inspiration and expiration.
- Non-respiratory - where the process occurs seemingly for no reason.
- Sometimes seen in association with Complete Heart Block.

Below are two examples of sinus arrhythmia. The first is less obvious than the second.



Sinus arrhythmia is not commonly seen but the same evaluation procedure is used:

- Is there a P wave?
- Is each P wave the same shape?
- Is each P wave followed by a QRS complex?
- Is the P-R interval between 3-5 small squares?
- Is the rhythm regular?

If the answer to the first four questions is "yes", but the answer to the last is "no" then you have sinus arrhythmia.

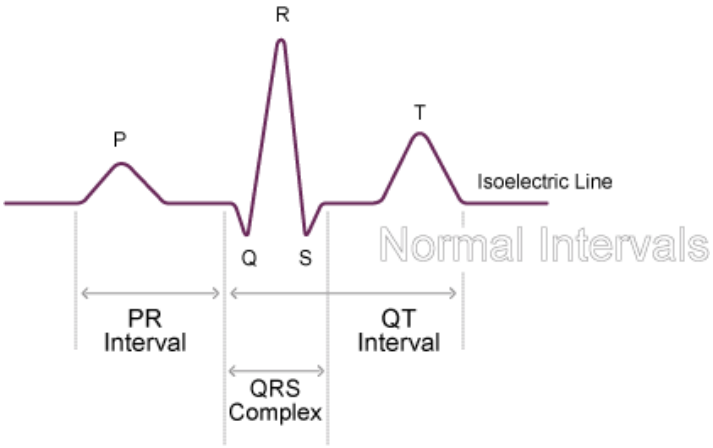
Normal Duration Times

As well as being able to recognise the 3 different parts of the cardiac cycle, each stage should be completed within a specific time period to be considered normal. Although

these measurements are in fractions of a second, the ECG paper allows time to be counted in small squares. Measurement in small squares is more universally used than tenths and hundredths of seconds.

The P-R Interval

The first measurement is the P-R interval and is measured from the beginning of the upslope of the P wave to the beginning of the QRS wave. This measurement should be 0.12-0.20 seconds, or 3-5 small squares in duration. The second measurement is the width of the QRS which should be less than 3 small squares, or less than 0.12 seconds in duration.

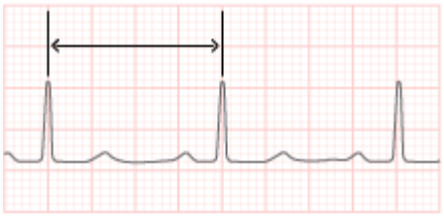


- P-R interval = 0.12 - 0.20 sec (3 - 5 small squares)
- QRS width = 0.08 - 0.12 sec (2 - 3 small squares)
- Q-T interval 0.35 - 0.43 sec
- * The PR interval should really be referred to as the PQ interval; however it is commonly referred as the PR interval

Regularity

The third measurement is to check for regularity by measuring the P-P interval or the R-R interval. The distance between the centre of 2 consecutive P waves is measured and compared with the measurement with the next 2 P waves. If the measurements are the same then the rhythm is regular. There are 300 large squares per minute. If

the rhythm is regular count the number of large squares between two QRS complexes and divide it into 300.



$$\text{Heart Rate} = \frac{300}{4} = 75 \text{ per minute}$$

Is it Sinus Rhythm?

To ascertain whether a rhythm is sinus or not key features need to be identified:

- There must always be a p wave.
- The P wave should be a rounded shape
- Each P wave should be the same shape
- Each P wave should be followed by a QRS
- The P-R interval should be 3-5 small squares and constant
- The rhythm should be regular.

T waves do not have to be identified for it to be sinus rhythm. Many abnormalities obscure the T wave. A good rule of thumb is that if the patient is still alive, then the ventricles are definitely repolarising !