Clinical skills: the physiological basis and interpretation of the ECG

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Abstract

Electrocardiograph (ECG) interpretation is a complex subject that requires considerable experience. However, an understanding of the principles underlying generation of the ECG can make this learning process easier. This knowledge is necessary if interpretation is to be founded on understanding rather then being a pattern recognition-based process. The aim of this article is to introduce the practitioner to the basic processes and mechanisms that govern formation of the normal ECG. These principles will largely be applied to the normal ECG and include cardiac anatomy applied to the ECG, how the ECG leads look at the heart and how the normal ECG waveform is formed. Examples of how the ECG changes when the underlying mechanisms are disturbed will also be given. Together, this knowledge should help the practitioner to have a clearer understanding of interpreting the abnormalities seen on an ECG.

Key words: Anatomy and physiology ■ Cardiovascular system and disorders ■ Electrocardiography ■ Patient assessment

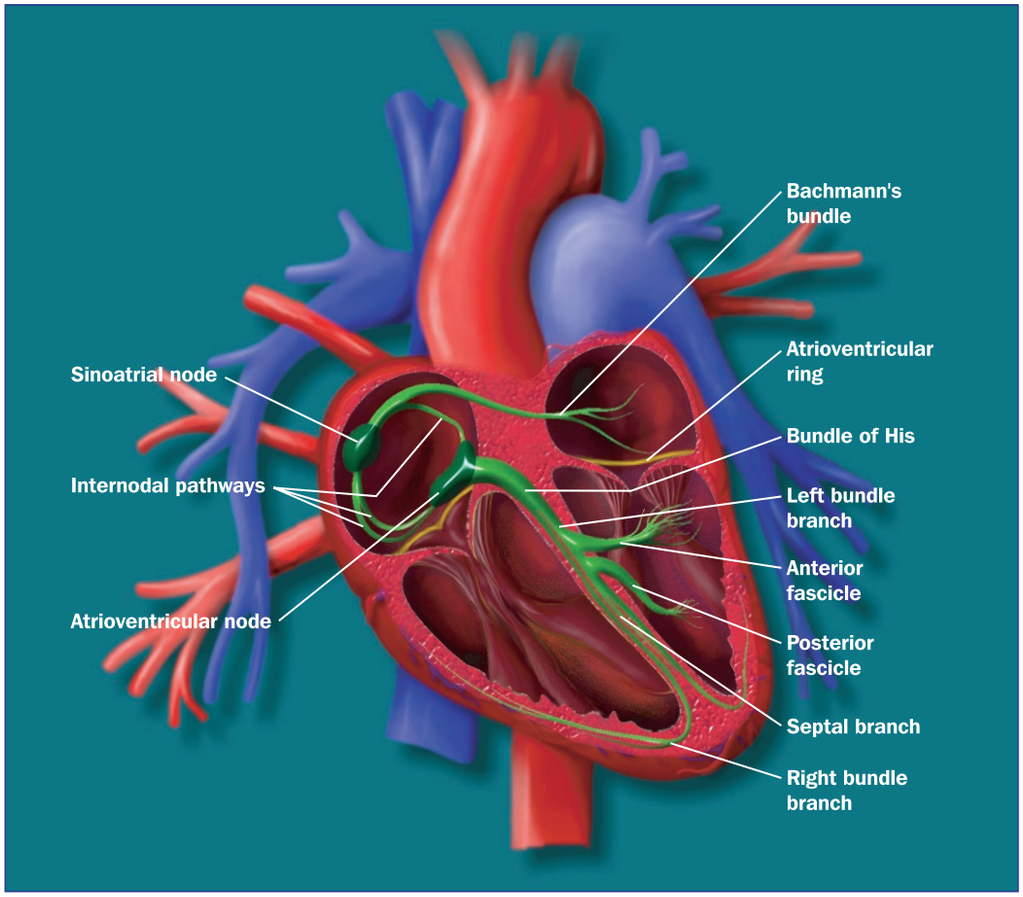

Figure 1. Diagrammatic representation of the conduction system of the heart.

With the improvement of computerized automation and decrease in prices of electronic technology, electrocardiograph (ECG) is now more accessible to nursing staff. Better understanding of disease is also now prompting healthcare professionals to use ECG interpretation skills in illnesses that are not directly associated with the cardiac patient (Booker et al, 2003). ECG monitoring is increasingly being implicated in mental health because of the Q–T interval-prolonging effect of many antipsychotic medications (Hennessy et al, 2002; O'Brien and Oyebode, 2003). Therefore, it is now becoming necessary that all nurses have a basic understanding of ECG.

Many articles have been published on ECG interpretation; however, few clearly discuss the physiological basis of the ECG waveforms. The ECG is a pictorial representation of electrical conduction and myocardial excitation in the heart (Despopoulos and Silbernagl, 2003). To understand this picture, one needs to understand the relationship between the waveforms seen in the ECG and the conduction events that cause them. This article will try to address these physiological events and apply them to ECG interpretation, in an attempt to make the ECG more comprehensible.

History of the ECG

Current generated by the heart was first recorded in humans by Augustus D Waller (1856–1922) in St Mary's Hospital, London. Unfortunately, Waller failed to recognize the clinical potential of his discovery (Sykes and Waller, 1887). It fell to Willem Einthoven of Leiden (1860–1927) to refine Waller's techniques and generate a clinically relevant ECG (Hurst, 1998). It is for this reason that Einthoven is generally recognized as the father of ECG. It was Einthoven who labelled the ECG waveform as PQRST (Snellen, 1995). It is suggested

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that he may have used the letters in the middle of the alphabet because he was not sure if he had determined all the waves present in the ECG and wanted to make sure there were enough letters available to accommodate any newly discovered waves (Hurst, 1998). Einthoven subsequently discovered the U wave a few years later (Snellen, 1995).

Impulse conduction in the heart

The electrical impulses that cause contraction in the heart are conducted via specialized modified muscle tissues that make up the conduction system in the heart (Levick, 2000). Although the myocardium conducts electrical impulses, the specialized conduction fibres conduct the electrical impulse 2–3 times faster than the myocardium (Levick, 2000). The conduction system, therefore, produces an organized rapid progression of the electrical impulse throughout the heart. The main structures involved in this system are shown in *Figure 1*. They include the sinoatrial (SA) node, atrial conduction pathways, the atrioventricular (AV) node and the ventricular conduction pathways. The atria and ventricles are separated by a thick layer of fibrous tissue — the atrioventricular ring or annulus fibrosus (Marriot and Conover, 1998; Meek and Morris, 2002). This fibrous ring electrically insulates the atria from the ventricles, so that under normal conditions the only path of conduction from the atria to the ventricles is via the AV node (Marriott and Conover, 1998).

Sinoatrial node (SA node)

The SA node, which is found in the right atrium near the inlet of the superior vena cava, is composed of a group of specialized cells that have the property of automaticity (Opie, 1998; Huszar, 2001). Automaticity is the ability of the tissue to generate automatically an action potential (current) (Marriot and Conover, 1998). Automaticity occurs because of a small but continuous leak of positive ions into the cell (Waldo and Wit, 2001). The rate of automaticity in the SA node can be modulated by a number of mechanisms, including the autonomic nervous system and some hormones (Opie, 1998). As the SA node normally possesses the fastest rate of automaticity in the heart at 60–100 impulses/minute, it is the physiological pacemaker of the heart (Berne and Levy, 1992).

Internodal pathways

The impulse generated by the SA node is conducted throughout the atria via conduction pathways. There are four main atrial pathways: three in the right atrium and one in the left (see *Figure 1*). The three right atrial pathways are known as the internodal pathways and they carry the electrical impulse from the SA to the AV node, causing the atrial myocardium to contract as it travels down the atrium. The fourth atrial conduction pathway runs from the SA node to the left atrium and is known as the Bachmann's bundle (Berne and Levy, 1992).

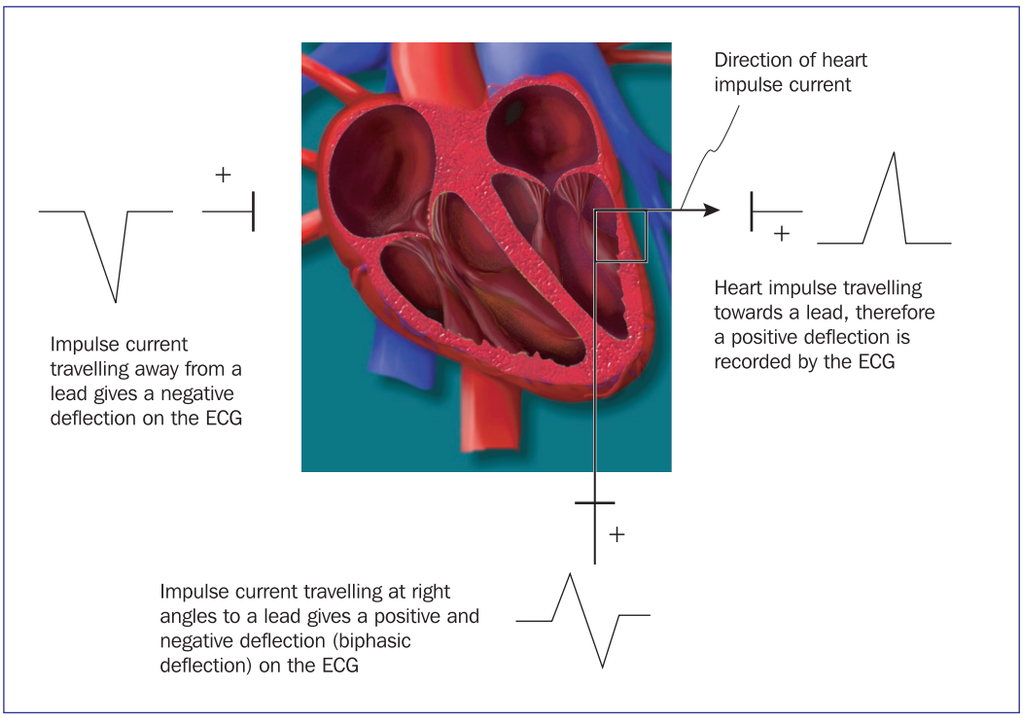


Figure 2. Principles of impulse recording by the electrocardiograph (Docherty, 2003).

Atrioventricular node (AV node)

The AV node is an important part of the conduction system. It has a number of specific functions. As mentioned, it acts as a bridge to allow current to cross the atrioventricular ring. The AV node has a slow conduction speed of approximately 0.2 m/second (Waldo and Wit, 2001); this slows down electrical impulses from the atria, delaying their entry into the ventricles, allowing time for adequate ventricular filling. The AV node does not possess the property of automaticity itself; however, the portion of conduction tissue joining the AV node to the bundle of His (the AV junction) does. Its intrinsic rate is less than that of the SA node with an approximate automaticity firing rate of 40–60 impulses/second (Berne and Levy, 1992). This enables the AV junction to become a secondary pacemaker in the event of an abnormal reduction in SA node firing rate.

Finally, the AV node acts as a frequency filter, by reducing the number of atrial beats entering the ventricles when the atrial firing rate is very rapid. This characteristic of the AV node is only evident when the atrial firing rate is above 180–200 impulses/minute (Berne and Levy, 1992). Fast atrial firing rates are hardly ever owing to SA node activity as the SA node firing rate does not normally exceed 170–190 impulses/minute as the maximum sinus tachycardia is normally calculated as 220–age (University of California San Francisco, 2003). They are likey to be owing to an area of abnormal automaticity that may be present in the conduction fibres or myocardium of the atria (Huszar, 2001).

The degree to which impulses are inhibited by the AV node depends on the impulse rate, such that the atrial–ventricular conduction ratio (atrial waves: ventricular beats) may be 2:1, 3:1, etc. as the atrial firing rate increases (Marriot and Conover, 1998). Therefore, the AV node acts as a frequency filter (Opie, 1998). It is this frequency filtering function of the AV node that allows only a proportion of the fast atrial waves to enter the ventricles resulting in a lower ventricular rate, thus resulting in the appearance of atrial flutter (Puech, 1956).

Ventricular conduction fibres

Ventricular conduction fibres consist of the bundle of His and its branches (see *Figure 1*). The bundle of His is divided into two main branches, the right and left bundle branches. The left bundle branch is then further divided into two and sometimes three other branches; the sub-branches of the left bundle branch are named according to the path they take in the left ventricular wall. They are the anterior

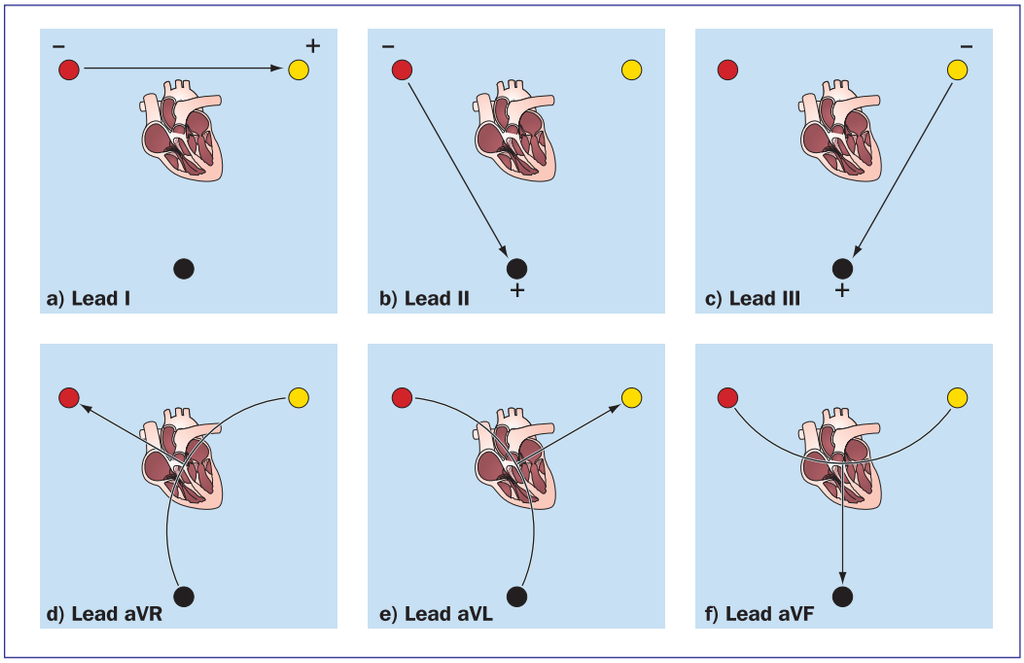


Figure 3. Bipolar and augmented lead recording; modified from Despopoulos and Silbernagl (2003).

superior fascicle; this is a thin branch that radiates anteriorly and superiorly across the left ventricular wall. The other main sub-branch of the left bundle branch is the posterior inferior fascicle, which, as its name suggests, radiates posteriorly and inferiorly across the left ventricular wall. This fascicle is quite thick compared with the anterior fascicle. The final sub-branch of the left bundle branch is the mid-septal fascicle (Demoulin and Kulbertus, 1972). This branch is present in approximately a third of the population (Kulbertus and Demoulin, 1976).

The Purkinje fibres

These are the final part of the conduction system and result from subdivisions of the bundle branches. The Purkinje fibres send forth branches or extensions into the myocardium and run through the subendothelial connective tissue layer. Thus contraction of the myocardium occurs from deep within the heart and follows an endocardial–epicardial or inside–outward direction (Schamroth, 1990).

The ECG and what it records

The ECG records electrical activity generated by the heart, by recording current from terminals or leads placed on specific areas of the body. Different leads look at the heart from different directions. Depending on which direction the current is travelling in respect to the observing lead (i.e. the ECG lead in question, e.g. lead I, aVF, V1, etc.), the lead will record a positive wave when the current is moving towards it, a negative wave when the current is moving away from it and a biphasic deflection (positive and negative) wave when the electrical impulse is at right angles (90°) to the observing lead (Docherty, 2003) (*Figure 2*).

This is the principle by which the ECG records the electrical activity of the heart. What is actually seen on an ECG recording is more complex. A surface ECG tracing records the net electricity generated by the heart during the cardiac cycle (Huszar, 2001).

Before the origin of the ECG waveforms is looked at in terms of cardiac contraction, a brief description of the position and perspectives of the various leads would be beneficial.

ECG leads

Limb leads

There are six limb leads that can be recorded using a combination of three leads or electrodes. The three leads used in recording the limb lead waveforms are placed as follows:

- Right arm: (normally red lead)
- Left arm: (normally yellow lead)
- Left foot: (normally green lead)

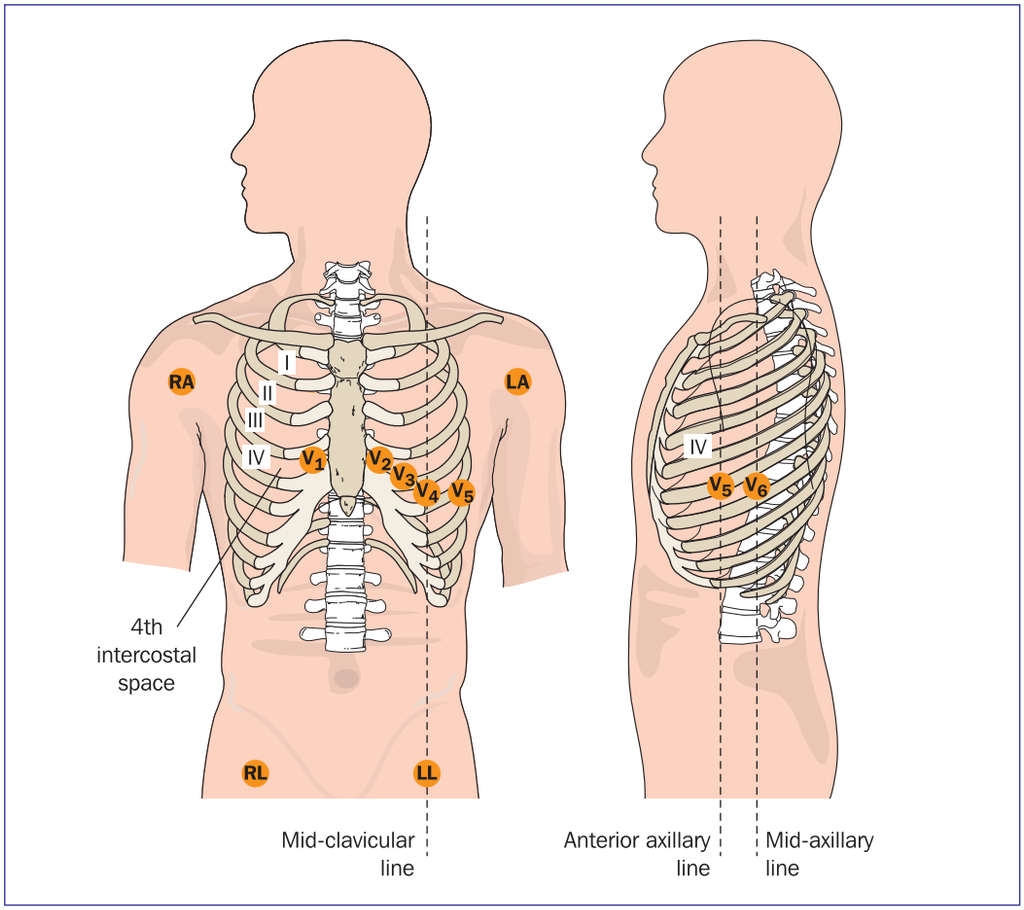
(Right foot is normally black lead, which is neutral).

These leads are further divided into bipolar and unipolar or augmented leads.

Bipolar limb leads

These leads are called bipolar because they have two definite poles: a positive and a

Figure 4. Intercostal spaces/12-lead electrocardiograph placement (from Docherty, 2003).



negative electrode. The lead that picks up the current is known as the exploring, observing or facing lead. Conventionally, the exploring or facing lead is always positive (Schamroth, 1990). The limb leads include:

Lead I: This lead is made of the terminal on the right arm and the terminal on the left arm. The right arm is the negative terminal and the left arm the positive (*Figure 3a*). This lead looks at the heart on the frontal plane and picks up activity from the lateral side of the heart, i.e. the left lateral ventricle wall.

Lead II: Lead II runs from the right arm (negative) to the left foot (positive) (*Figure 3b*). It again is a frontal lead and looks at the inferior aspect of the heart. It is normally positive and usually shows the greatest deflection of all the limb leads in a normal heart (Schamroth, 1990).

Lead III: This lead runs from the left arm (negative) to the left foot (positive) (*Figure 3c*). This frontal lead also looks at the inferior surface of the heart, but at a more righthanded aspect than lead II (Huszar, 2001).

Unipolar leads

The unipolar leads include the augmented leads and the chest leads. These are called unipolar because they only have one definite pole. For ease of understanding, the lead receiving the electric current will be referred to as the positive or exploring lead. There is no real negative terminal as the combination of all three limb leads forms a 'neutral' terminal; this 'terminal' is as if there were a terminal in the centre of the chest, a null reference point (Docherty, 2003). In all the unipolar leads the current generated by the heart is measured from the centre of the chest to the exploring lead.

Augmented leads

aVR: In the aVR unipolar lead, the positive (exploring) terminal is the right arm (*Figure 3d*). This lead looks at the heart from the right upper surface of the heart. This lead is nearly always negative, as it looks down onto the cavity of the heart, thus the P wave, QRS and T waves are normally negative. This lead is positive when the patient is dextrocardiac (heart on the right side) or when the left and right arm leads have been swapped over (Ho and Ho, 2001).

aVL: In this lead the positive (exploring) terminal is on the left arm (*Figure 3e*). This lead looks at the left lateral aspect of the heart; however, it looks at the lateral wall from an angle of approximately 30°, whereas lead I looks at the lateral wall on a horizontal plane (Huszar, 2001).

aVF: In this lead the positive (exploring) terminal is the left leg lead (*Figure 3f*). This lead looks at the inferior aspect of the heart (Huszar, 2001).

Unipolar chest leads

Also called precordial leads, the chest leads are made up of a combination of leads. In this case, although all three limb leads combine to make up a single neutral lead, the exploring lead is the chest lead that is normally placed on one of six positions on the chest wall (*Figure 4*).

The chest leads are placed in a horizontal plane along the chest wall. The placement for these leads is as follows (Huszar, 2001):

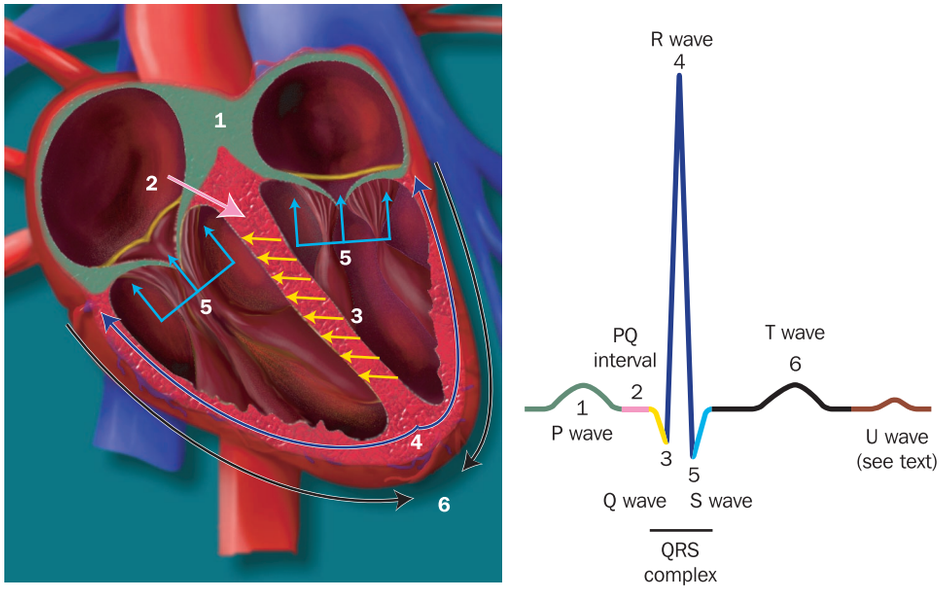


Figure 5. Origin of the waveforms seen on the electrocardiograph (ECG). Events during the cardiac cycle are colour coded to the waveforms seen on the ECG.

V1 Right side of sternum on sternal margin, fourth intercostal space

V2 Left side of sternum on sternal margin, fourth intercostal space

V3 Before putting on lead V3, V4 should be located then V3 is placed in a direct line between V2 and V4

V4 Fifth intercostal space in the mid-clavicular line. It has been suggested that adipose tissue causes minimal aberration to ECG recording (Rautaharju et al, 1998). Therefore, the electrode should be placed on top of the breast in women so as to attain the appropriate placement

V5 Placed on the fifth intercostal space on the anterior axillary line

V6 Same plane as V4 and V5 but placed on the mid-axillary line.

The ECG and its origins from cardiac contraction

Expanding a little more on what has already been discussed, the production of the ECG waveform will now be explored. When observing the ECG limb lead II is usually used because the cardiac axis is normally directed towards this lead (Schamroth, 1990). The electrical events that lead to contraction in myocardial cells are known as depolarization and the relaxation of myocardium results in electrical activity known as repolarization (Schamroth, 1990). Thus, depolarization pertains to contraction of myocardium and repolarization to its relaxation.

Cardiac contraction starts in the right atrium from the SA node. First the right atrium contracts via the internodal pathways, then the Bachmann's bundle carries the impulse into the left atrium and this contracts (red brackets, *Figure 5*). This activity forms the P wave (Schamroth, 1990).

The impulse now enters the AV node where it is delayed; this generates a flat segment on the ECG, called the PQ interval (*Figure 5*, orange arrow). The segment is flat because the small amount of tissue involved in conduction only generates a small amount of electrical activity that cannot be perceived by surface ECG recordings (Waldo and Wit, 2001). Following the AV node delay, the impulse enters the bundle of His and travels down both bundle branches. As the impulse travels through the bundle of His, the septum contracts. The septal muscle mass is small, thus its contraction results in a comparatively small-sized wave. As the mean direction of electrical activity generated during septal contraction is from the bottom left of the septum upward, the waveform recorded in lead II is negative, as the current is moving away from

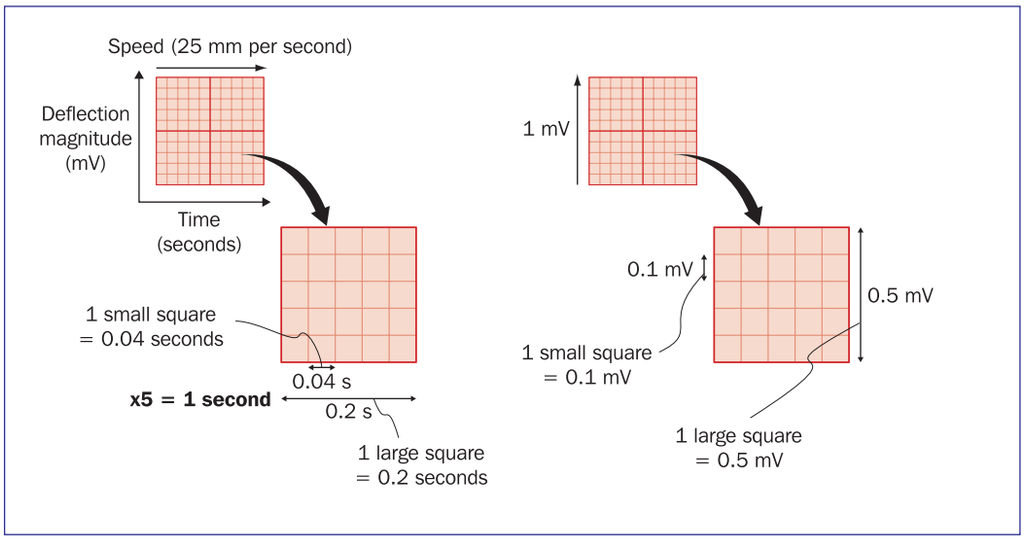


Figure 6. Electrocardiograph graph paper measurements.

it (*Figure 5*, pink arrows). This first negative deflection on the ECG after the P wave is known as the septal q wave and is most clearly seen in V5–V6 and limb lead II (Huszar, 2001).

Following septal contraction, the impulse enters both ventricles almost simultaneously and causes contraction of the ventricles from the apex of the heart upwards (*Figure 5*, green arrows). Contraction of the myocardium occurs from the endocardium outwards (Schamroth, 1990). The net direction of the electrical activity generated during ventricular contraction is, therefore, downward towards the apex of the heart and a little towards the left. This is because the left side of the heart (left ventricle) consists of more muscle than the right side and, therefore, when it contracts it generates more electrical activity. As the lead that faces this orientation is lead II, ventricular contraction is most clearly seen in this lead; hence healthcare professionals normally monitor in lead II and lead II is also the average normal conduction. The contraction of the main part of the ventricles results in the formation of the R wave on the ECG (Schamroth, 1990).

Finally, the muscle around the major valves contracts (*Figure 5*, purple arrows). As this electrical activity is directed upwards towards the base of the heart and away from lead II, the deflection formed on the ECG is negative and small in size because of the small amount of muscle in this area. This negative deflection is known as the S wave (Schamroth, 1990). The ventricles relax and repolarize, which is seen on the ECG as a positive waveform after the S wave — the T wave (*Figure 5*, blue arrows) (Schamroth, 1990).

In some cases, an additional wave may be seen after the T wave — the U wave (*Figure 5*). The origin of this wave is unclear; however, it has been suggested that it is produced after depolarizations in the ventricles, by repolarization of the His-Purkinje system, or by prolonged repolarization of a specific cell layer (M-cells) in the mid-myocardium (Ritsema van Eck et al, 2003). This wave is seen best in the rightward chest leads, especially in V3 (Ritsema van Eck et al, 2003).

ECG paper

Now the way cardiac conduction forms the different ECG waveforms has been examined, normal dimensions can be assigned to these waves. To understand ECG waveform dimensions, one needs to understand the graph paper on which the ECG is recorded. Time is plotted along the horizontal axis (X-axis) of the ECG graph paper and along the vertical axis (Y-axis), the paper shows deflection magnitude in mV (*Figure 6*). The graph paper is divided into small squares of 1 mm each and five small squares make up a large square. As the normal running speed of ECG paper is 25 mm/second, one small square on the X-axis equals 0.04 seconds and one large square (five small squares) equals 0.2 seconds ($5 \times 0.04 = 0.2$). Therefore, five large squares is equal to 1 second ($5 \times 0.2 = 1$) (Huszar, 2001).

The vertical plane of the ECG paper describes the deflection magnitude of the cardiac conduction. The ECG recorder is calibrated such that 10 small squares or two big squares give a deflection of 1 mV (Huszar, 2001). This is important as changing this calibration may lead to misinterpretation of the ECG.

Deflections on the ECG

Although the physiological basis of the ECG waveforms has been described, these waveforms sometimes have a slightly different meaning from an interpretational point of view (*Figure 7*). These definitions are given below — see Huszar (2001) for a detailed discussion.

P wave: Normally the first positive deflection of the ECG cycle, usually symmetrically rounded.

Q wave: First negative deflection that precedes the first positive deflection (normally the P wave).

R wave: First positive deflection of the QRS complex. ECGs may have more than one positive deflection in the QRS. The first deflection is labelled R or r depending on the size of the deflection. If the deflection is large then the wave will be called R, if it is small it will be r. The next positive deflection in a QRS complex is denoted R' or r' (R prime). Subsequent positive deflections are described as R" or r" and so on.

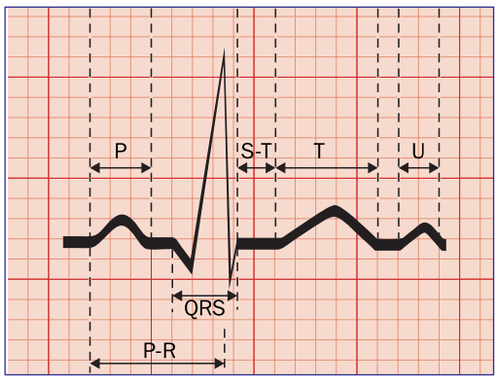


Figure 7. Electrocardiograph waveform nomenclature.

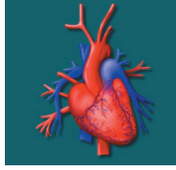
S wave: The first negative deflection after the R wave. Similar to the R wave, the S wave may be written as S or s depending on its size. Again, as in the R wave, the second negative deflection following an R wave is called S' (S prime) and the third S", etc.

T wave: Generally, the T wave is the next deflection to follow the QRS complex and is normally in the same direction as the preceding QRS complex. It tends to be less symmetrical than a P wave and is normally sloped on one side.

U wave: Small, normally positive deflection following the T wave. Its presence or absence is not pathological but it is important to recognize it because it may be mistaken as a P wave.

Time durations associated with the above waveforms are given in *Table 1*. It should be noted that the QT interval and the ST segment durations are rate-dependent. Out of these two measurements the QT interval (or QTc, as it is known once its duration has been corrected for heart rate) is the most important (Huszar, 2001).

QT interval: Prolongation of this interval leads to a specific form of ventricular tachycardia known as torsades de pointes. This form of VT has an oscillating pattern in that the



magnitude of the ventricular waveforms oscillate from a small amplitude to a large amplitude waveform (Huszar, 2001). Prolongation of the QT interval may be caused by genetic congenital abnormalities in the heart or by drug induction (Khan, 2002).

ECG interpretation

Before starting to interpret an ECG, a few rules need to be observed. These include proper labelling of the ECG with the patient's name, the time and date of the recording and, if the ECG is one of a series, the series number of the ECG information. The presence and type of chest pain is important in assessing ischaemic heart disease, as this may allow a differential diagnosis between cardiac and non-cardiac chest pain. Care should be taken to ensure a good-quality recording devoid of artefacts — tracings on the ECG from sources other than cardiac in nature, e.g. skeletal muscle movement or tremors and external electrical activity (Huszar, 2001). Interpretation of an ECG may be in one of two forms: rhythm strip interpretation or 12-lead interpretation. A single rhythm strip only contains information recorded from one perspective of the heart, whereas a 12-lead ECG contains information recorded from many different perspectives.

When doing any kind of ECG interpretation, it is important to be systematic, otherwise subtle changes may be missed that lead to misinterpretation of the ECG (Schamroth, 1990; Marriott and Conover, 1998). Ventricular activity is normally the most important feature of an ECG (Marriot and Conover, 1998) because a rhythm strip has limited use in detailed analysis of ventricular beats. Therefore, in the absence of obvious ventricular arrhythmias an anatomical approach is normally sufficient for rhythm strip analysis.

Rhythm strip interpretation

The following need to be analysed: general QRS shape; rate; rhythm; P wave; PR interval; P–R ratio.

General QRS wave shape: Little can be said for certain from a rhythm strip about the QRS waveform, apart from the gross changes of ventricular rhythms, hypertrophy and infarction. For an indepth analysis of the QRS, the precordial leads from a 12-lead ECG should be used. However, the rhythm strip can yield important information about the QRS duration, e.g. qRS widening associated with bundle branch blocks may be seen here (Huszar, 2001).

Rate: Bradycardic, normal, tachycardic.

Rhythm: Regular or irregular — remember all regular rhythms are not normal. Sinus tachycardia, ventricular tachycardia and first degree AV block are all examples of regular rhythms (Schamroth, 1990).

P wave duration and height: Prolonged and humped P waves may indicate left atrial hypertrophy, whereas tall P waves (>2.5 mm) may signify right atrial hypertrophy (Schamroth, 1990).

PR interval: Short PR intervals (<0.12 second) signify a bypass of the AV node, a phenomenon known as pre-excitation, which can be associated with some supraventricular tachycardias (Schamroth, 1990). Prolonged PR intervals (>0.2 second) may be caused by AV node ischaemia and increased parasympathetic stimulation (Huszar, 2001).

P–R ratio (number of P waves to R waves): There should always be a one-to-one relationship between the P wave and the qRS complex. Arrhythmias in which there are more P waves per QRS complex include second and third degree AV block. Atrial flutter and atrial fibrillation are also associated with a disparately high atrial activity when compared with the QRS waveform. However, in this case these waves are not called P waves, they are known as flutter and fibrillation waves, respectively (Huszar, 2001).

Table 1. Durations of some waves and intervals of the ECG

P wave	PR interval	QRS duration	QT
0.1 sec	0.18–0.2 sec	0.07–0.1 sec	Rate dependent

Rating	Adult male (msec)	Adult female (msec)
Normal	<430	<450
Borderline	430–450	450–470
Prolonged	>450	>470

Source: Huszar (2001); Strnad (2002); ECG = electrocardiograph

12-lead ECG interpretation

All the initial recording considerations given in rhythm strip interpretation remain the same for 12-lead interpretation, but as mentioned, when performing a 12-lead ECG, analysis begins with the precordial wave forms. Precordial waveforms are used because they are the closest leads to the heart and are, therefore, more sensitive leads.

The following interpretation tool is an adaptation of the 12-lead ECG interpretation tool described by Huszar (2001), with detail from Schamroth (1990), Thaler (2003) and others as indicated.

12-lead ECG analysis

- Analyse major shape of QRS complexes
- Rate (ventricular and atrial if necessary)
- Precordial lead analysis
- Limb lead analysis (including axis determination)
- P wave analysis
- PR interval analysis.

Major shape of the complexes

Look for obvious signs of ventricular abnormality, such as ventricular tachycardia and signs of infarction; ST segment elevation.

Rate: Similar to rhythm strip analysis.

Rhythm: Similar to rhythm strip analysis.

Check for appropriate R wave progression: The first precordial lead (V1) should be predominantly negative and leads V5–V6 should be predominantly positive (Huszar, 2001). This gives an indication of the average direction of the contractile force in the ventricles. R wave progression changes in the presence of hypertrophy and intraventricular conduction blocks (Schamroth, 1990).

Precordial leads

The duration should not be more than 0.1 seconds. Anything over this, especially durations over 0.12 seconds suggest significant intraventricular conduction delay (Schamroth, 1990). Such delayed conduction is commonly seen with ventricular conduction blocks and with ventricular ectopic beats (Schamroth, 1990).

The height of the R wave should not exceed 26 mm and the depth of the S waves should not be more than 30 mm. Abnormally tall R waves or deep S waves indicate hypertrophy of the ventricles (Huszar, 2001).

The Q wave depth should not be more than one quarter the ensuing R wave height. Abnormally deep Q waves in any lead normally signify an infarction in the part of the heart facing those leads (Schmaroth, 1990).

T wave

The T wave is the most unstable part of the ECG recording and is, therefore, found changed with a number of ECG abnormalities (Schamroth, 1990). The T wave in V1 may be

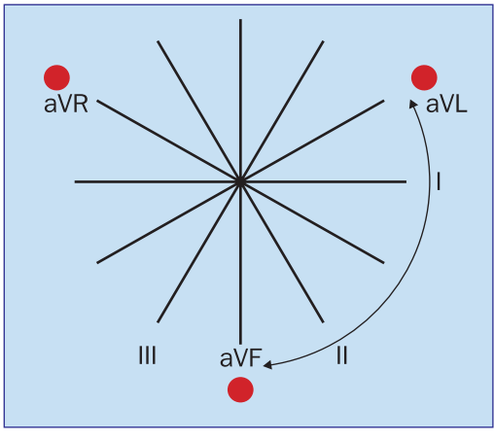


Figure 8. Hexaxial reference system. ECGs that have an axis between the arrows have a normal axis.

upright or may be biphasic or inverted, unless originally upright in the same patient. Normally V2 is similar to V1 and V3–V6 are normally upright. Inverted T waves may be a sign of an infarction (Morris and Brady, 2002).

Precordial ST segment

The ST segment should be on the baseline in most cases. The ST segment should be measured 2 mm from the J point (Huszar, 2001). The J point is the point where the S wave comes up to the baseline and begins the ST segment (Schamroth, 1990). This may sometimes be difficult to see, as there may be no clear demarcation between the beginning of the ST segment and the end of the S wave. An ST segment 0.5 mm below the baseline commonly signifies ischaemia (Jones, 2003). A downward slurred ST segment may be seen with ventricular muscle strain found in hypertrophy (Schamroth, 1990). An elevated ST segment in two consecutive leads indicates an acute infarction: >1 mm in limb leads and >2 mm in chest leads (Morris and Brady, 2002; Jones, 2003). Persistent ST elevation may occur for a number of reasons, including formation of a ventricular aneurysm (Thaler, 2003). Pericarditis may also cause persistent ST segment elevation; however, this tends to be found throughout the chest leads commonly associated with a notch on the end of the QRS (Smith et al, 2002).

Analyse limb leads

Analysis of limb leads is similar in most cases to precordial leads, although the magnitude of the waveforms is slightly less as they are further away from the heart.

Cardiac axis

The cardiac axis is determined from the limb leads. The cardiac axis shows the mean direction of the flow of electrical activity generated in the heart. As the myocardium contracts from endocardium to epicardium (outwards), the axis is normally directed downwards and as the lefthand side of the heart is more muscular than the right, the axis is shifted slightly towards the left (Schamroth, 1990). Therefore, the cardiac axis is normally directed towards the left foot (towards lead II). The normal axis is found between limb lead aVL and aVF on the hexaxial reference system (*Figure 8*) (Huszar, 2001). The hexaxial reference system is made up by superimposing the directions of the six limb leads on top of each other using the heart as a central point (see *Figure 8*).

The direction of the normal cardiac axis depends on normal muscle contraction, which in turn depends on an intact conduction system and myocardium. Therefore, axis shifts (movement away from lead II but still within normal limits) or deviations (axis outside the normal limits) may be seen in conduction blocks such as bundle branch, hemiblocks or in hypertrophies, where one side of the heart becomes abnormally dominant in terms of contractility than the other (Schamroth, 1990). For a simple method to determine axis see Docherty (2003).

P wave analysis and PR interval analysis

This is similar to the analysis found in rhythm strip analysis. As a result of the effects of pre-excitation on precordial waveforms, it may be appropriate to preclude the presence of pre-excitation before analysing the precordial leads.

Conclusion

Confident ECG interpretation takes experience and practice. When setting out to learn ECG interpretation, it might help to enlist the help of a knowledgeable senior. However, a basic understanding of the physiology underlying the waveforms is helpful. Coupled with this, an understanding of the 'view' ECG leads have on the heart will help the practitioner to make a logical interpretation of the ECG. This article has tried to give a foundation for both these principles. BJN

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KEY POINTS

- Electrocardiograph (ECG) recording and interpretation is becoming more prevalent in many healthcare environments.
- Intelligent ECG interpretation necessitates an understanding of the basic physiology behind the waveforms.
- Proper ECG analysis requires an understanding of the different views a 12-lead ECG has on the heart.
- To ensure a comprehensive analysis of the ECG, a systematic approach to ECG interpretation needs to be adopted.
- To gain confidence and expertise in ECG interpretation, nurses should practice under the supervision of a proficient practitioner.