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 than 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

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

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

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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 (Marriott and Conover, 1998; Meek and Morris, 2002). This fibrous ring electrically isolates 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) (Marriott 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).

**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 likely 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 (Marriott 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
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 subepithelial connective tissue layer. Thus contraction of the myocardium occurs from deep within the heart and follows an endocardial–epicardial or inside–outside 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: (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
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):

- **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
- **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.

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 Wü, 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 the inferior aspect of the heart (Huszar, 2001).
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 x 0.04 = 0.2). Therefore, five large squares is equal to 1 second (5 x 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.

**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). Short PR intervals (<0.12 sec) 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 disparity between 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**

<table>
<thead>
<tr>
<th>P wave</th>
<th>PR interval</th>
<th>QRS duration</th>
<th>QT</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.1 sec</td>
<td>0.18–0.2 sec</td>
<td>0.07–0.1 sec</td>
<td>Rate dependent</td>
</tr>
</tbody>
</table>

**Rhythm:** Similar to rhythm strip analysis.

**Rate:** Similar to rhythm strip analysis.

**Major shape of the complexes**

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

**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 (Schamroth, 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
endocardium to epicardium (outwards), the axis is normally directed downwards and as the left-hand 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.

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.