**Electrocardiograms: a guide to rhythm recognition for emergency nurses**

**Introduction**

The electrocardiogram (ECG) is one of the most commonly performed investigations in the Emergency Department (ED), and is an extremely useful adjunct that guides diagnosis, prognosis and treatment. In the majority of cases, nurses are the first health care professional to assess the patient, and to record an ECG, yet anecdotal evidence would suggest that very few emergency nurses actively review, interpret and act on the findings from the ECG. This may be due to lack of confidence or knowledge as noted in a recent study of nurses working with an ambulance service which found that the nurses had deficiencies in their ECG interpretation skills (Werner et al 2016). This is supported further by a study in 2013 which showed that nurses in ED scored lower than those in other departments when it came to ECG interpretation (Zhang and Hsu 2013). A number of further studies have also highlighted that that medical and nursing staff knowledge of ECG interpretation is often limited due to lack of or inadequate training (Hernández-Padilla 2017, Richley, 2013). Therefore given the staffing pressures in UK hospitals at the current time, finding someone qualified to review an ECG can result in delay to the patient’s treatment. The development of ECG interpretation skills among ED nurses is therefore desirable. The usually starting place for nurses new to ECG interpretation is the development of rhythm recognition skills.

The aim of this guide is to enable emergency nurses to understand and interpret the cardiac rhythms that are commonly encountered in the ED, and therefore offer earlier diagnosis and treatment. We propose a simple, five step method that evaluates the key components of cardiac rhythm. This method should be underpinned by a sound understanding of the anatomy and physiology of the heart; readers may therefore want to revise this topic to get the most from this guide. As well as good quality Anatomy and Physiology books, our previous articles may be a useful starting point (Sampson and McGrath 2015a; Sampson and McGrath 2015b) in helping you understand the physiology behind am ECG. Gaining a good understanding of cardiac electrical function, and how this is displayed on the ECG, will help ED Nurses to understand how the ECG changes during rhythm disturbance.

**The five-step method**

It can be tempting to rush in and focus on abnormal complexes, however, by being systematic you will gain a lot more information from the ECG. This methodical approach will increase your understanding, and ability to interpret the ECG. There are a number of methods available to help practitioners to do this,

such as the colour-coded CRASH system (Jabbour and Touquet, 2014).

This guide will provide you with a simple and straightforward five-step approach to interpreting the ECG that believe is one the easiest ways of interpreting an ECG (EKG.Academy, 2017; Scribd 2017).

This approach requires you to look in turn at the heart rate, heart rhythm, QRS duration, the presence or absence of a P-wave, and the PR interval. By doing so you should be able to identify both the normal and abnormal components of an ECG which will guide you when interpreting rhythms.

**Graph paper**

It is important that you have a good understanding of the graph paper used when recording an ECG. The ECG is a graph displaying cardiac electrical activity (figure 1). Time is measured along the x-axis, while amplitude (voltage) is measured along the y-axis. The graph paper is divided into small squares (1mm x 1mm) and larger squares (5mm x 5mm). Each small square has a time interval of 0.04 seconds, while one large square measures 0.20 seconds (5 x 0.04 = 0.2).

 In terms of amplitude, one small square is equal to 0.1 millivolts (mV), so a large square is 0.5 mV. Time is especially important when it comes to rhythm recognition; this will become apparent as we work through the five step method.

***Figure 1. The ECG is a graph of cardiac electrical activity***

**Step 1: Heart Rate**

The normal heart rate at rest is between 60 and 100 beats per minute (bpm) (Hampton, 2013). When reading a rhythm strip, always read from left to right and begin by calculating the heart rate. For rhythms that are regular, this is relatively simple. Take two QRS complexes, and count the number of large (5mm x 5mm) squares between them. This number is then divided into 300. For example, in figure 2 (panel a) there are approximately 4 large squares between each QRS complex. 300 divided by 4 is 75; the heart rate is approximately 75 beats per minute.

***Figure 2. Calculating heart rate in regular (panel a) and irregular (panel b) rhythms.***

Although this method is a great way to estimate the heart rate for regular rhythms, it does not work for irregular rhythms, because the interval between QRS complexes is not consistent. An alternative method in this case is to average out the heart rate over six seconds, which is 30 large squares.

Start by marking off 30 large squares on the rhythm strip (figure 2, panel b). Next, count the number of QRS complexes within that section. This gives the heart rate over six seconds. Finally, multiply the number of QRS complexes by 10, to give the heart rate in beats per minute. In the example in figure 2, there are 9 QRS complexes, giving a heart rate of approximately 90 beats per minute.

**Step 2: Regularity**

A normal heart rhythm is regular, in other words, the QRS complexes appear at regular intervals. Rhythms, for the most part, can be divided into three categories: stable, too slow and those that are too fast (Kern, Halperin, & Field, 2001).Checking the regularity of a rhythm is therefore an important step in rhythm interpretation. A simple method of testing regularity is to use a blank piece of paper.

Place the paper against the rhythm strip so that the edge of the paper is above the rhythm . Make a mark on the paper above the R-wave of three successive QRS complexes. Now simply move the paper along the rhythm strip; if the rhythm is regular the points marked will continue to match up with the other R-waves. If the rhythm is irregular, the distance between each R-wave will be variable.

**Step 3: P-waves**

The normal P-wave is produced by depolarization of left and right atria, triggered by a regular impulse produced by the sinus node (Ashley and Niebauer 2004). A normal P-wave is a small, rounded or tent-shaped waveform that does not exceed 2.5mm in height or width (figure 4).

Each P-wave should have the same general appearance. When looking at the P-wave, always ask yourself the following questions; are there P-waves present? If so, are they regular? Is there a P-wave in front of every QRS complex? Are they smooth, upright and rounded in appearance or are they inverted? And finally do they all look the same?

***Figure 3. Waveforms and intervals***

**Step 4: QRS duration (width)**

The QRS complex represents depolarization of both ventricles (Bagliani 2017). During normal sinus rhythm, with normal conduction to the ventricles, the width of the QRS complex is less than 0.12 seconds (less than 3 small squares) (figure 3). A broad QRS (greater than 0.12 seconds) suggests that conduction to the ventricles is abnormal, for example there is bundle branch block, or that the rhythm has arisen within the ventricles themselves.

**Step 5: PR interval**

The PR interval is measured from the onset of the P wave to the onset of the QRS complex (figure 3)( Fleming 2012). It measures the interval between the onset of atrial contraction to the onset of ventricular contraction. A normal interval is within the range of 0.12- 0.20 seconds, which equates to 3-5 of the small squares on the ECG paper.

When looking at the PR interval always ask yourself the following questions. Is the interval greater than 0.20 seconds or less than 0.12 seconds? Is the interval consistent across the ECG? A shortened or prolonged PR interval suggests that conduction between the atria and ventricles is abnormal.

**Putting it all together**

In summary, a normal ECG strip will have

* A heart rate between 60 and 100bpm
* QRS complexes at regular intervals
* A normal shaped, upright P-wave in front of every QRS complex
* A QRS complex that is narrow (less than 0.12s / 3 small squares)
* A PR interval that is consistent, and between 0.12 and 0.20 seconds.

Before we move on let’s quickly recap normal sinus rhythm.

**Normal sinus rhythm**

Normal sinus rhythm is the recording of an impulse originating in the SA node, and passing through a normal conduction pathway to the ventricles (figure 4) (Garcia, 2015). You should use this rhythm as a baseline for all other rhythms that you are interpreting.

If we evaluate the rhythm strip in figure 4 using the five step method,

1. We can see that the heart rate is 75 bpm, and the rhythm is regular.
2. There is a small, rounded, upright P-wave in front of each QRS complex.
3. Each P-wave has more or less the same shape, which you would expect as every beat is coming from the SA node.
4. The QRS duration is normal at less than 0.12 seconds.
5. Finally, looking at the PR interval, you can see that there are not more than 5 small squares between the start of the P-wave and the beginning of the QRS complex. Anything above 5 squares would indicate first-degree heart block, which is discussed below (Aehlert, 2011).

***Figure 4. Normal sinus rhythm***

**Sinus Tachycardia**

Sinus tachycardia is a fast heart rhythm originating in the SA node, with a heart rate above 100bpm (Marieb and Hoehn, 2015). This rhythm is a normal response to exercise, and may also result from anxiety, pain and fever. Other causes include hyperthyroidism, heart failure, and the use of substances including alcohol, caffeine and cocaine (Houghton and Gray, 2014)

As with normal sinus rhythm, the rhythm is regular and the QRS duration is normal. A P-wave can be seen before each QRS complex, and the PR interval is normal. The only abnormality is the increase in heart rate.

**Sinus Bradycardia**

Sinus bradycardia is also a common variant of sinus rhythm, but in this case the heart rate is less than 60bpm (Swift, 2013). This rhythm is common in athletes, whose hearts produce a greater stroke volume, and therefore an adequate cardiac output at lower heart rates (Kenny et al, 2015). Sinus bradycardia also occurs when vagal outflow to the heart is high, for example during sleep or as the result of vagal manoeuvres such as carotid sinus massage (Klabunde, 2012). Other causes include hypoglycaemia, hypothermia, hypothyroidism, raised intracranial pressure, and medications including beta blockers and digoxin (Hampton, 2013).

When reviewing a rhythm strip that shows sinus bradycardia it will fit all the criteria for sinus rhythm; it is regular, the QRS duration is normal, there is a P-wave before each QRS complex, and the PR interval is at the upper limit of normal at around 0.2s.The only difference will be that the heart rate is less than 60".

**Junctional rhythm**

All parts of the cardiac conduction system are able to produce an electrical impulse, and act as subsidiary pacemakers (Hampton, 2013). If the SA node fails to fire, or its impulses are blocked, these secondary pacemakers take over, ensuring that the heart does not stop beating (Bennett, 2013). This phenomenon is referred to as escape rhythm (Chugh 2014).

Junctional rhythm is an escape rhythm originating in the area comprising the AV node and bundle of His. This area is known as the AV junction (Aehlert, 2011). On the ECG, the heart rate is between 40-60 beats per minute, the rhythm is regular, and the QRS duration normal (Garcia, 2015). In ECG lead II, the P-waves are inverted, or may be concealed by the QRS complex (figure 5).

***Figure 5. Junctional rhythm. In this example, the P-waves are hidden the QRS complex.***

**Atrial fibrillation (AF)**

During AF, the electrical impulse does not originate in the SA node. The rhythm is triggered by rapid and abnormal electrical activity, usually from around the pulmonary veins in the left atrium (Haisseguerre et al, 1998). The AV node is bombarded by up to 600 impulses per minute, some of which are conducted to the ventricles in an irregular pattern (Elliot, 2014). The heart rate during AF is variable, and depends on conduction speed through the AV node. Ventricular rates between 100 and 160bpm are common, although heart rates up to and beyond 200bpm are possible (Bennett, 2013). Heart rates below 100bpm are seen when AV node conduction is slowed by drugs or conduction system disease (Kirchof et al, 2016). AF often occurs in people with hypertension or structural heart disease, and may also be caused by hyperthyroidism, alcohol excess and acute infection (NICE, 2014).

On the ECG, the key findings are an irregularly-irregular rhythm (in other words, there is no obvious pattern to the irregularity), and an absence of normal P-waves (Aehlert, 2011) (figure 6). P-waves are replaced by fibrillation waves, which appear as a continuous and chaotic undulation of the baseline (Garcia, 2015). Because the atria are fibrillating continuously, fibrillation waves often distort the shape of the T-waves, as seen in figure 6. We cannot use the large square method to calculate heart rate during irregular rhythms, but using the 30-square method we can see that there are 14 QRS complexes in figure 6, which is 30 large squares long. This gives us a heart rate of approximately 140bpm. Note that it is impossible to measure the PR interval during AF, because normal P-waves are not present.

***Figure 6. Atrial fibrillation***

**Atrial flutter**

Atrial flutter is caused by a rapid but coordinated beating of the atria. This is due to an abnormal electrical impulse that sweeps continuously around the walls of the left or right atrium (Kircher et al, 2014). Most commonly the right atrium is involved, and the impulse circulates around the annulus of the tricuspid valve (Feld et al, 2008). This is referred to as typical atrial flutter, and is characterized by an atrial rate of approximately 300bpm (Houghton and Gray, 2014). The causes of atrial flutter are similar to AF, and the two arrhythmias may be seen at different times in the same individual (Kirchof et al, 2016).

In typical atrial flutter, P-waves are replaced by flutter waves with a sawtooth appearance in the inferior ECG leads (Aehlert, 2011) (figure 7). The heart rate is often rapid, and the rhythm may be regular, or regularly-irregular. This appearance depends on the ratio of flutter waves that are conducted to the ventricles. In the example in figure 7, AV conduction is variable, producing a regularly-irregular rhythm. At acute presentation, 2:1 conduction is more common, resulting in a regular rhythm with a heart rate around 150bpm (Bennett, 2013). QRS duration is usually within the normal range. The PR Interval is not measurable.

***Figure 7. Typical atrial flutter. Note the sawtooth flutter waves. There is variable conduction to the ventricles.***

**Supraventricular tachycardia (SVT)**

SVT refers to a rapid, regular rhythm with a narrow QRS complex (figure 8). The heart rate is typically between 140 and 220bpm (Nolan & Pitcher, 2015). P-waves are often not seen; where they are discernable, they occur after the QRS complex, and may distort the ST-segment or T-wave (Houghton and Gray, 2014). The PR interval is often impossible to determine.

SVT can be caused by several different pathologies. In most cases, it results from extra electrical pathways that are present in the heart at birth (Medi et al, 2009). The most common abnormality affects the AV node, which has two distinct electrical pathways leading into it, instead of one. This is referred to as dual AV node physiology (Mullord and Sargeant, 2011). Less commonly, individuals are born with a strand of myocardium bridging the AV ring, the layer of connective tissue that separates the atria from the ventricles. This is known as an accessory pathway, and provides an additional pathway for electrical conduction between the upper and lower chambers (Chugh et al, 2008).

During SVT, the electrical impulse is conducted back into the atria using the additional pathway in a process known as “re-entry”. This produces the rapid, regular heart rate seen during SVT (Lee and Linker, 2014). The P-wave is often concealed by the QRS complex because the atria and ventricles are depolarized almost simultaneously. SVT can affect people of all ages, including children (Page et al, 2015). Episodes of SVT may be triggered by stress, hypoxia, physical exertion, hypokalaemia and stimulants such as caffeine (Aehlert, 2011).

***Figure 8. SVT. Note the rapid, regular rhythm and absence of P-waves.***

**First-degree atrioventricular (AV) Block**

First-degree AV block occurs when the electrical impulse is delayed in its passage from the SA node to the ventricles. This is usually due to slow conduction in the AV node, although delay in the bundle of His or bundle branches is also possible (Bennett, 2013). When reviewing the ECG, all the components of normal sinus rhythm are present, however the PR interval is prolonged (>5 small squares) (figure 9). First-degree AV block may be caused by age related degeneration of the conduction system, or drugs such as calcium channel blockers, beta blockers and digoxin (Chow et al, 2012). It rarely has any clinical significance.

***Figure 9. First degree AV block. The PR interval is 340 milliseconds.***

**Second-degree AV block**

In second-degree AV block, some impulses are conducted to the ventricles, while others are blocked (Hampton, 2013). This results in a regular or regularly-irregular rhythm with a slow or normal heart rate. Second-degree AV block is divided into two distinct types, according to its ECG appearance; Mobitz type I, also known as Wenckebach, and Mobitz type II (Aehlert, 2011).

**Mobitz type-I (Wenckebach)**

In Mobitz type-I, there is increasing conduction delay with each successive impulse from the SA node, until an impulse fails to conduct (Houghton and Gray, 2014). On the ECG, the PR interval prolongs with each successive beat, until a QRS complex is dropped (figure 10). The heart rate may be normal, but it is more likely to be slow. The rhythm is regularly irregular. The P-wave is normal, but there are more P-waves than QRS complexes because of the non-conducted impulses. Like first-degree AV block, Mobitz type-I is usually caused by slowing of AV node conduction. This is a normal phenomenon in younger people, especially during sleep (Bennett, 2013). In older individuals, or in the acutely unwell, it is more likely to be due to damage to the conduction system, or the effect of drugs such as beta-blockers (Vogler et al, 2012).

***Figure 10. Second-degree AV block, Mobitz type I. The PR interval prolongs until a beat is dropped***

**Mobitz type-II**

In Mobitz type-II, there is also intermittent failure of conduction, resulting in dropped beats (Swift, 2013). Unlike Mobitz type-I, however, the PR interval does not prolong prior to a dropped beat, and is the same for every conducted beat (figure 11). When you review the ECG, you'll note that the rate is normal or slow, and the rhythm is regular or regularly-irregular. The QRS duration is often prolonged. There are more P-waves than QRS complexes, because of the dropped beats (Aehlert, 2011). Mobitz type-II is usually caused by degeneration or acute damage to the bundle of His or bundle branches, for example by an inferior wall myocardial infarction (Bennett, 2013). Cardiac output may be diminished due to a slow ventricular rate, and there is a high risk of progression to complete AV block (Chow et al, 2012).

***Figure 11. Second-degree AV block, Mobitz type II. Note the broad QRS complex, and consistent PR interval.***

**Third-degree AV block (complete heart block)**

Third-degree AV block is also referred to as complete heart block, and occurs when conduction between the atria and ventricles is completely blocked. In other words, none of the impulses from the sinus node are conducted to the ventricles (Houghton and Gray, 2014). A subsidiary pacemaker below the level of the block takes control of the ventricles, resulting in a slow, regular escape rhythm that is often between 20 and 40 beats per minute (Swift, 2013). The QRS complex is usually broad.

On the ECG, you will see a slow rhythm with regular P-waves, and regular QRS complexes (figure 12). There is no relationship between the P-waves and QRS complexes, and there will be more P-waves than QRS (Vogler et al, 2012). Because the P-waves are not related to the QRS complexes, the PR interval is variable, and P-waves may be seen before or after the QRS, or distorting the T-waves. Like Mobitz type-II, third-degree AV block usually results from acute or chronic damage to the bundle of His or bundle branches. Cardiac output is often impaired, and there is a risk of asystole, making the management of this rhythm a medical emergency (Pitcher and Nolan, 2015).

***Figure 12. Third-degree AV block. There are regular P-waves, and regular QRS complexes, but no relationship between them. The QRS complex is wide.***

**Ventricular ectopics (premature ventricular complexes)**

Ectopic beats are produced by single electrical impulses arising outside of the SA node (Aehlert, 2011). Ventricular ectopics, also known as premature ventricular complexes (PVCs), originate in the conduction tissue or muscle of the ventricles, and are commonly seen on otherwise normal ECGs (Bennett, 2013). Because the PVC does not originate in the atrium, it is not preceded by a P-wave (figure 13). For the same reason, the QRS complex is wide and bizarre looking (Garcia, 2015). Ectopic beats always occur early, in other words before the next sinus beat would be expected. Although ventricular ectopics are a normal and benign finding in healthy individuals, frequent PVCs in critically unwell people can be a harbinger of sustained ventricular arrhythmia (Lee et al, 2012; John et al, 2012).

***Figure 13. The fourth beat is a PVC. The underlying rhythm is sinus bradycardia.***

**Ventricular Tachycardia (VT)**

Ventricular tachycardia is a rapid, regular rhythm arising within the ventricles (Houghton and Gray, 2014). The heart rate is greater than 100bpm, and the QRS complex is broad. P-waves do not precede the QRS complexes (figure 14) (Aehlert, 2011). VT may occur in short bursts (non-sustained VT) or may be continuous. If VT is sustained, loss of consciousness or cardiac arrest may occur (Bennett, 2013). If the patient is unconscious, and there is no pulse present, they will need immediate defibrillation (Soar et al, 2015).

The most common cause of VT is myocardial ischaemia due to coronary heart disease (John et al, 2012). This arrhythmia is also common in critically ill people, in whom hypoxia, acidemia or electrolyte imbalance result in myocardial irritability (Bench and Brown, 2011). In 10% of cases, VT occurs in the context of normal health, in which case it rarely causes cardiac arrest (Prystowsky et al, 2012).

***Figure 14. Ventricular tachycardia.***

**Ventricular Fibrillation (VF)**

VF is a chaotic and disorganized pattern of electrical activity arising in the ventricles (Bench and Brown, 2011). This causes the lower chambers to quiver instead of contract, and cardiac output is lost (Klabunde, 2012). The patient with VF requires immediate defibrillation and cardiopulmonary resuscitation, as the blood is no longer being pumped around the body (Soar et al, 2015). On the ECG, the heart rate is fast, usually above 300bpm, and the rhythm is disorganized and irregular. The QRS duration is not recognizable, and P-waves are not seen (figure 15) (Hampton, 2013).

***Figure 15. Ventricular fibrillation. The rhythm is fast and disorganized.***

**Asystole**

Asystole occurs when there is nocardiac electrical activity (Aehlert, 2011). Myocardial contraction ceases, resulting in complete loss of cardiac output (Bench and Brown, 2011). Immediate cardiopulmonary resuscitation is required as the patient is in cardiac arrest. Because there is no cardiac electrical activity, defibrillation is not effective, and should not be attempted (Soar et al, 2015). When you look at the ECG you will see a flat line; there are no QRS complexes or P-waves (figure 16) (Aehlert, 2011). The outlook for patients with asystole is poor; only one in ten individuals survives to discharge following an in-hospital, asystolic arrest (Nolan et al, 2014).

***Figure 16. Asystole.***

**Conclusion**

This guide has put forward the five-step method as one of the easiest ways to interpret the rhythm strip, and has provided a breakdown of the common rhythms seen in the ED. Use of this method can help emergency nurses to evaluate cardiac rhythms in a systematic way, enabling the early detection of abnormal rhythms, and facilitating timely treatment. This can play a significant role in improving patient outcomes, and ensures that emergency nurses are fully equipped to provide the best possible care for their patients.

Words 4113

**References**

Aehlert B (2011) *ECGs made easy*, fourth edition, Maryland Heights: Mosby Elsevier.

Ashley EA, Niebauer J. Cardiology Explained. London: Remedica; 2004. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK2204/>

## [Bagliani G,](http://www.sciencedirect.com/science/article/pii/S1877918217300679%22%20%5Cl%20%22%21) [De Ponti R,](http://www.sciencedirect.com/science/article/pii/S1877918217300679#!)  [Gianni C,](http://www.sciencedirect.com/science/article/pii/S1877918217300679%22%20%5Cl%20%22%21) [Padeletti L (2017)](http://www.sciencedirect.com/science/article/pii/S1877918217300679%22%20%5Cl%20%22%21) The QRS Complex: Normal Activation of the Ventricles: [Cardiac Electrophysiology Clinics](http://www.sciencedirect.com/science/journal/18779182), [Volume 9, Issue 3](http://www.sciencedirect.com/science/journal/18779182/9/3), September 2017, Pages 453-460

Bench, S. & Brown, K. (2011) *Critical care nursing: learning from practice*, Chichester, West Sussex : Wiley-Blackwell

Bennett DH (2013) *Bennett’s Cardiac Arrhythmias: Practical notes on interpretation and treatment*, 8th edition, London: Hodder Arnold.

Chow GV, Marine JE & Fleg JL (2012) Epidemiology of Arrhythmias and Conduction Disorders in Older Adults, *Clinical Geriatric Medicine*, 28(4), 539–553.

Chugh A, Bogun F & Morady F (2008) Catheter ablation of accessory pathways, in Wilber DJ, Packer DL & Stevenson WG (eds) *Catheter ablation of cardiac arrhythmias: Basic concepts and clinical applications*, Third edition, Oxford: Blackwell Publishing.

Chugh SN (2014) Textbook of Clinical Electrocardiography: For postgarduates, residents and practicing physicans ( 3rd Ed) Jaypee Brothers Publishers Ltd New Delhi

Ekg.academy. 2017. */learn-ekg*. [ONLINE] Available at: [https://ekg.academy?courseid=315&seq=2](https://ekg.academy/?courseid=315&seq=2). [Accessed 13 September 2017].

Elliott K (2014) The nurse’s role in the management and treatment of atrial fibrillation, *British Journal of Cardiac Nursing*, 9(12), 586-591.

Feld GK, Birgersdotter-Green U & Narayan S (2008) Diagnosis and ablation of typical and reverse typical (type 1) atrial flutter, in Wilber DJ, Packer DL & Stevenson WG (eds) *Catheter ablation of cardiac arrhythmias: Basic concepts and clinical applications*, Oxford: Blackwell Publishing, p. 173-192.

[Fleming](https://www.google.co.uk/search?tbo=p&tbm=bks&q=inauthor:%22J.S.+Fleming%22) J.S.( 2012) Interpreting the Electrocardiogram, Springer Science & Business Media, London.

Garcia TB (2015) *12-lead ECG: The Art of Interpretation.*  2nd Edition. Burlington, Ma; Jones and Bartlett

Haisseguerre M, Jais P, Shah DC, Takahashi A, Hocini M, Quiniou G, Garrigue S, Le Mouroux A, Le Metayer P & Clementy J (1998) Spontaneous initiation of atrial fibrillation by ectopic beats originating in the pulmonary veins, *New England Journal of Medicine*, 339, 659-666.

Hampton JR (2013) *The ECG made easy*, 8th edition, London: Churchill Livingstone.

Hernández-Padilla JM, Granero-Molina J, Márquez-Hernández VV, Suthers F, López-Entrambasaguas OM, Fernández-Sola C. (2017) [Design and validation of a three-instrument toolkit for the assessment of competence in electrocardiogram rhythm recognition.](https://www.ncbi.nlm.nih.gov/pubmed/28045334) Eur J Cardiovasc Nurs. 2017 Jun;16(5):425-434.

Houghton AR & Gray D (2014) *Making sense of the ECG: A hands-on guide*, 4th edition, Boca Raton: CRC Press.

Jabbour R, Touquet R (2014) A stepwise approach to reading ECGs using colour-coded electricalviewpoints. *British Journal of Cardiac Nursing***9**(6): 293–6.

John RM, Tedrow UB, Koplan BA, Albert CM, Epstein LM, Sweeney MO, Miller AL, Michaud GF & Stevenson WG (2012) Ventricular arrhythmias and sudden cardiac death, *Lancet*, 380: 1520–29.

Kern, K., Halperin, H., & Field, J. (2001). New guidelines for cardiopulmonary resuscitation and emergency cardiac care.Changes in the management of cardiac arrest. Journal of the American Medical Association, 285, 1267-1269.

Kenney WL, Wilmore J, Costill D (2015) *Physiology of Sport and Exercise*. 6th edn. Human Kinetics, Champaign

Kircher S, Rolf S, Hindricks G, Sommer P (2014) Ablation of typical atrial flutter using a novel non-fluoroscopic electromagnetic catheter tracking system*, Interventional Cardiology*, 6(2):149-58.

Kirchhof P, Benussi S, Kotecha D et al (2016) 2016 ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS, *Eur Heart J*, 37, 2893–2962.

Klabunde RE (2012) C*ardiovascular physiology concepts*, 2nd edition, Baltimore, MD; Lippincott Williams & Wilkins.

Lee V, Hemingway H, Harb R, Crake T & Lambiase P (2012) The prognostic significance of premature ventricular complexes in adults without clinically apparent heart disease: a meta-analysis and systematic review, *Heart.,* 98:1290-1298 doi:10.1136/heartjnl-2012-302005

Lee D. and John Linker, N (2014) Electrophysiology study in narrow complex tachycardia. *British Journal of Cardiac Nursing*, *9*(1), 25-29.

Marieb EB & Hoehn KN (2015) *Human Anatomy & Physiology*, 10th edition, Cambridge: Pearson

Medi C, Kalman JM, Freedman SB (2009) Supraventricular tachycardia, *Med J Aust*, 90(5):255–60

Mullord P & Sargent A (2011) Pharmacological conversion of AV nodal re-entry tachycardia with adenosine, *British Journal of Cardiac Nursing*, 6(4), 178-183.

National Institute for Health and Care Excellence (2014) *Atrial fibrillation: the management of atrial fibrillation. NICE clinical guideline 180.* Available at <https://www.nice.org.uk/guidance/cg180> (Accessed 09/05/2017).

Nolan JP, Soar J, Smith GB, Gwinnutt C, Parrott F, Power S, Harrison DA, Nixon E, Rowan K (2014) Incidence and outcome of in-hospital cardiac arrest in the United Kingdom National Cardiac Arrest Audit, *Resuscitation*, 85(8):987-92.

Page RL, Joglar JA, Halperin JL & Levine GN (2015) 2015 ACC/AHA/HRS Guideline for the Management of Adult Patients with Supraventricular Tachycardia, *Circulation*, <http://dx.doi.org/10.1016/j.hrthm.2015.09.019>

Pitcher D, Nolan J (2015) *Peri-arrest arrhythmias.* Resuscitation Council UK, London. http://tinyurl. com/ogeh2jt (accessed 09/05/2017)

Prystowsky EN, Padanilam BJ, Joshi S & Fogel RI (2012) Ventricular arrhythmias in the absence of structural heart disease, *Journal of the American College of Cardiology*, 59(20), 1733-44.

Richley D (2013) New training and qualifications in electrocardiography. *British Journal of Cardiac* *Nursing* **8**(1): 38–42

Sampson M, McGrath A (2015q) Understanding the ECG. Part 1: Anatomy and physiology. *British Journal of Cardiac Nursing* **10**(11): 548–54.

Sampson M, McGrath A (2015b) Understanding the ECG. Part 2: ECG basics. *British Journal of Cardiac Nursing* **10**(12): 588–593.

Scribd. 2017. *5-Steps-to-Rhythm-Strip-Interpretation*. [ONLINE] Available at: [https://www.scribd.com](https://www.scribd.com/). [Accessed 13 September 2017].

Soar J, Deakin C, Lockey A, Nolan J & Perkins G (2015) Adult advanced life support, Resuscitation Council UK, London <https://www.resus.org.uk/resuscitation-guidelines/adult-advanced-life-support/> (accessed 09/05/2017).

Swift (2013) Assessment and treatment of patients with acute unstable bradycardia, *Nursing Standard,* 27 (22), 48-56.

Vogler J, Breithardt G & Eckardt (2012) Bradyarrhythmias and conduction blocks, *Rev Esp Cardiol*, 65, 656-667.

Werner K, Kander K, Axelsson C. (2016) [Electrocardiogram interpretation skills among ambulance nurses.](https://www.ncbi.nlm.nih.gov/pubmed/25548395) Eur J Cardiovasc Nurs. 2016 Jun;15(4):262-8.

Zhang H, Hsu LL (2013)[The effectiveness of an education program on nurses' knowledge of electrocardiogram interpretation.](https://www.ncbi.nlm.nih.gov/pubmed/23266113). Int Emerg Nurs. 2013 Oct;21(4):247-51.