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Understanding the ECG. Part 4: Conduction blocks

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Introduction

In the normal heart, the electrical impulse arising from the sinus node is conducted to the ventricles via the atrioventricular (AV) node, bundle of His, and bundle branches (Klabunde, 2012) (*Figure 1*). The normal operation of this specialised system ensures that every impulse from the sinus node is conducted to both ventricles in a rapid and efficient manner (Marieb and Hoehne, 2015).

Unfortunately, this process of impulse conduction is not normal in every individual. The cardiac conduction system is subject to the same disease processes that impair the function of other parts of the heart, and the result is a range of conduction disturbances (Chow et al, 2012). Impulses from the sinus node may fail to conduct through the atria, may be delayed in their journey through the conduction system, or may fail to arrive in the ventricles at all. Conduction may be normal through one bundle branch, but be blocked in the other (Garcia, 2015). All of these conduction problems are referred to as 'blocks', although their effect on heart rate, rhythm, and patient well-being vary considerably.

In this fourth article of the *British Journal of Nursing's* ECG Interpretation Series, we examine the conduction blocks commonly encountered in clinical practice, and discuss how they can be recognised on the electrocardiogram (ECG). As with the previous article in this series (Sampson, 2016), we also place each block in context by briefly considering why it occurs, its clinical significance, and any treatment that might be necessary.

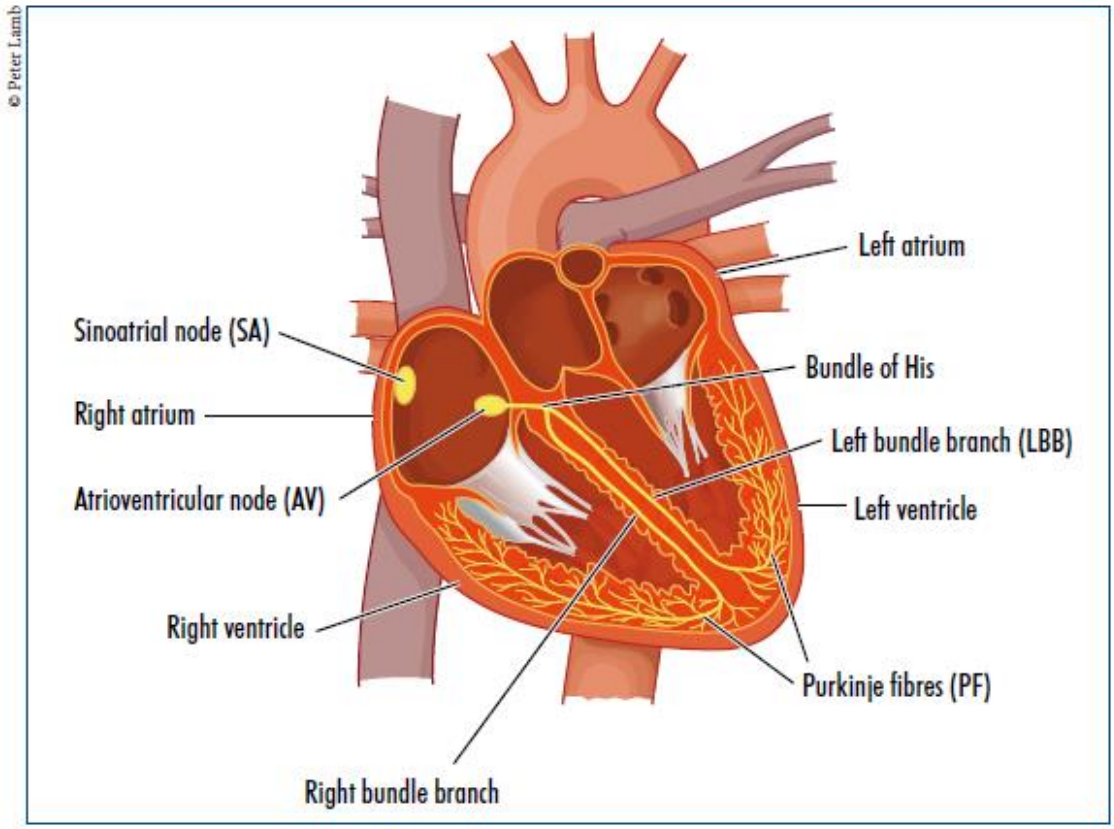

Figure 1. The cardiac conduction system: sites of conduction block include the sinoatrial (SA) node, atrioventricular (AV) node, and the left and right bundle branches.

Rhythm evaluation

We have discussed systematic rhythm evaluation in a previous article (Sampson and McGrath, 2015). Readers may wish to refresh their memories of this information before reading further. For convenience, we have reproduced the key steps in *Box 1*.

Rhythm evaluation questions

1. Is the rhythm regular?
2. Is the heart rate between 60 and 100 beats per minute?
3. Are there upright P-waves, and are they all the same shape?
4. Is there one P-wave in front of each QRS complex?
5. Is the PR interval normal (3 to 5 small squares?)
6. Is the QRS complex narrow (less than 3 small squares wide?)

Box 1. Key steps in systematic rhythm evaluation

Sinoatrial block

The sinus node is a relatively large structure, divided into functionally different areas. The electrical impulse arises in one part of the node, and is conducted through the surrounding nodal tissue before reaching the atrial myocardium. Conduction block occurring in this surrounding tissue will prevent the impulse from exiting the sinus node (Monfredi et al, 2010). This phenomenon is referred to as sinoatrial block, or sinus exit block. Because the non-conducted beats do not reach the main body of the atria, no P-wave is generated. The electrical impulse does not arrive at the AV node, and is not conducted to the ventricles, so there is no QRS complex or T-wave either (Aehlert, 2011). On the ECG, this results in a single missed beat in an otherwise regular rhythm (*figure 2).* Because the sinus node is still firing regularly, the pause is a multiple of the usual cycle length. For example, if the sinus node is firing once every second, the pause will be two seconds if a single impulse is blocked (Houghton and Gray, 2014).

A similar pause occurs if the sinus node fails to fire at all. This is referred to as sinus arrest (*figure 3*). The pause produced by sinus arrest is variable in length, and does not correspond with cycle length as it does in sinoatrial block (Hampton, 2013). Otherwise, the two problems are very similar in ECG appearance.

Sinoatrial block and sinus arrest occur most commonly in older people, often in association with related disorders such as sinus bradycardia, chronotropic incompetence, and paroxysmal atrial fibrillation or flutter. Collectively, these problems are referred to as sick sinus syndrome (Ewy, 2014). The most common cause of sick sinus syndrome is age related degeneration of the sinus node, resulting in fibrosis or ion-channel dysfunction, although it may also be due to coronary heart disease (Monfredi et al, 2010). Its symptoms include dizziness, loss of consciousness, palpitations, breathlessness, and exercise intolerance (Jensen et al, 2014). Permanent pacemaker implantation is necessary in symptomatic individuals, especially if there is a history of syncope (Bennett, 2013).

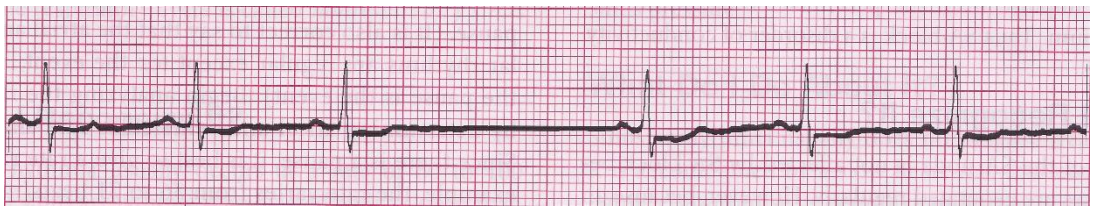

Figure 2. Sinoatrial block. The pause is exactly 2 cycle lengths.

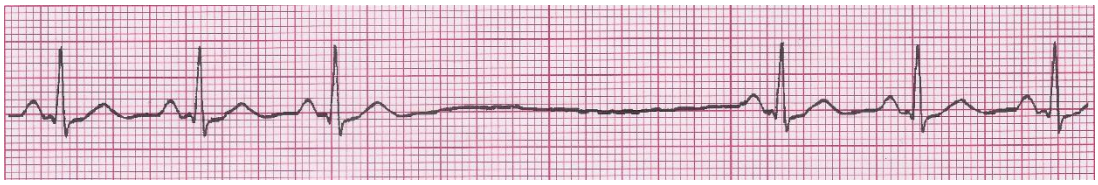

Figure 3. Sinus arrest. The pause is around 3.5 cycle lengths

Escape beats and rhythms

If sinoatrial block or sinus arrest result in a long enough pause, a subsidiary pacemaker in the AV junction may fire. This is referred to as an escape beat (Bennett, 2013). *Figure 4* shows a junctional escape beat that occurs following a sinus pause. Because the escape beat arises in the AV junction, the electrical wavefront travels upward through the atria instead of downward. This results in an inverted P-wave that may occur before or after the QRS, depending on conduction times and the location of the subsidiary pacemaker. In many cases, as in *figure 4*, the P-wave occurs at the same time as the QRS, and is therefore hidden by it. The QRS is narrow because conduction to the ventricles is normal (Aehlert, 2011).

Escape beats are a normal back up feature of the heart, and ensure that prolonged asystole does not occur (Klabunde, 2012). If the sinus node fails entirely, an escape rhythm from the AV junction will usually take over. This is referred to as junctional rhythm, and appears as a series of junctional escape beats (*figure 5*). The rate is usually slow, as pacemakers in the AV junction fire more slowly than those in the sinus node (Jowett and Thompson, 2007). The ECG features of junctional rhythm are

- A regular rhythm, often between 40 and 60 beats per minute
- P-waves are either inverted, or hidden by the QRS
- The QRS is narrow

If a complete block in conduction occurs lower in the conduction system, for example in the AV node, a subsidiary pacemaker below the level of the block usually ensures that the ventricles continue to depolarise. If the subsidiary pacemaker is within the bundle of His, the resulting escape rhythm will have a narrow QRS. If it is in the bundle branches or Purkinje fibres, the QRS will be broad. Subsidiary pacemakers in the lower parts of the conduction system are much slower than those in the AV junction, and are more unreliable. If they fail, asystole occurs (Bennett, 2013).

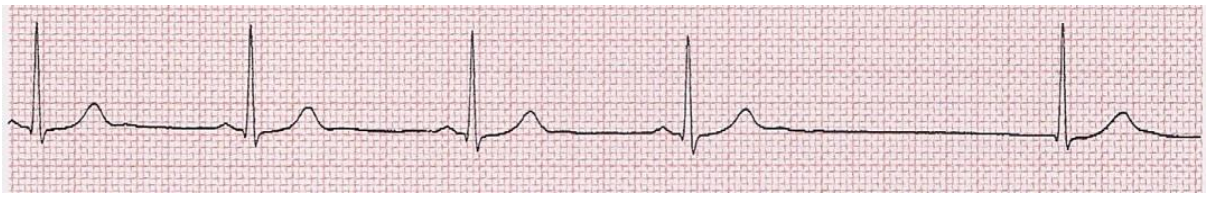

Figure 4. Sinus pause followed by a junctional escape beat.

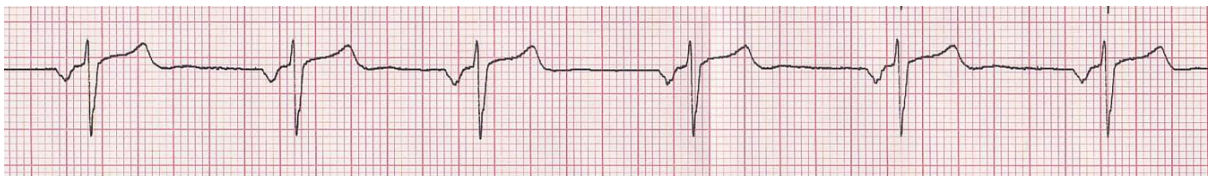

Figure 5. Junctional rhythm. In this example, there is an inverted P-wave before the QRS.

Atrioventricular blocks

AV blocks occur when conduction between the atria and ventricles is delayed, intermittently blocked, or fails completely. Unlike sinoatrial block, the electrical impulse exits the sinus node normally and is conducted through the atria, creating a P-wave on the ECG (Vogler et al, 2012). Conduction failure occurs further down the system, in the AV node, bundle of His or bundle branches. On the ECG, the effects of AV block can be seen in changes in the PR interval, in P-waves that are not followed by QRS complexes, or in complete electrical dissociation of the atria and ventricles (Hampton, 2013). Based on the degree of conduction block, AV blocks are classified as first-, second- or third-degree in nature (Chow et al, 2012).

First-degree AV block

In first-degree AV block, the electrical impulse is delayed on its journey from the sinus node to the ventricles. It is not, therefore, a block at all but rather a form of conduction delay (Aehlert, 2011). Most commonly, the delay occurs within the AV node, however slow conduction through the atria or His-Purkinje system may also be responsible (Vogler et al, 2012). On the ECG, first-degree AV block is characterised by prolongation of the PR interval (*figure 6*). In every other respect the rhythm is normal, and can easily be mistaken for normal sinus rhythm (Houghton and Gray, 2014). The key ECG findings are therefore

- A regular rhythm with a normal P-wave in front of every QRS
- A long PR interval (>200 milliseconds or 5 small squares)
- The PR interval is the same for every beat
- The QRS is narrow unless ventricular conduction block is present (*e.g.* bundle branch block)

First-degree AV block does not affect the heart rate or rhythm, and does not generally cause symptoms. It is often considered benign, and is a normal finding in young people and athletes, in whom it is usually due to high vagal tone (Bennett, 2013). Early research into first-degree AV block supports this benign view, demonstrating a low rate of progression to higher degrees of block, and no increase in mortality (Packard et al, 1954; Mymin et al, 1986). However, these findings were recently challenged by Cheng et al (2009) who point out that the early studies were based on healthy young men, and are not generalizable to a wider population. Their study, which followed 7575 people over five decades, found that individuals with first-degree AV block at baseline were twice as likely to develop atrial fibrillation, and three times as likely to need permanent pacing. Mortality rates were also moderately increased (Cheng et al, 2009).

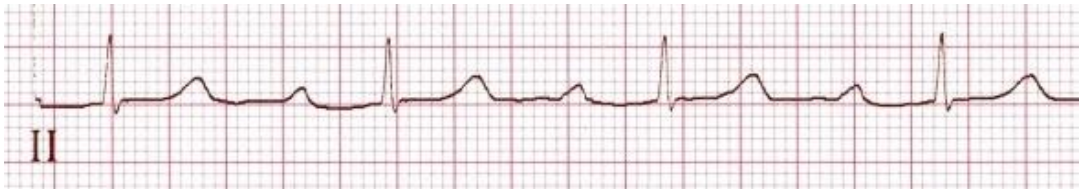

Figure 6. First-degree AV block. The PR interval is 340 milliseconds.

Second-degree AV block

In second-degree AV block, there is intermittent conduction between the atria and ventricles. In other words, some impulses are conducted through the electrical system successfully, while others fail to conduct. On the ECG, this results in some P-waves that are not followed by QRS complexes. Second-degree AV block is further subdivided into two distinct types according to ECG appearance: Mobitz type-I, or Wenckebach, and Mobitz type-II. There are prognostic differences between these types of second-degree block (Aehlert, 2011).

Mobitz type-I (Wenckebach)

In Mobitz type-I, conduction from the sinus node to the ventricles becomes progressively slower with each beat, until an impulse fails to conduct at all. On the ECG, this results in progressive lengthening of the PR interval until a P-wave is not followed by a QRS complex (*figure 7*). After the dropped beat, the PR interval returns to its shortest length before starting to lengthen again. The dropped beats make the rhythm regularly irregular (Houghton and Gray, 2014). The ECG findings in Mobitz type I are therefore:

- There are more P-waves than QRS complexes
- The PR interval gets longer with each beat, until a P-wave is not followed by a QRS
- After a dropped beat, the PR interval 'resets' and the cycle starts again
- The rhythm is regularly irregular (areas of regular rhythm are separated by dropped beats)
- The QRS complex is narrow, unless there is concurrent bundle branch block.

Like first-degree AV block, Mobitz type-I may be caused by slow conduction within the AV node. In young, healthy people this is a normal finding at night, and is caused by high vagal tone. In older people, and during the day, Mobitz type-I is less likely to be harmless, and more likely to be caused by damage to the distal conduction system. In this case, it carries a similar prognosis to Mobitz type-II (Bennett, 2013).

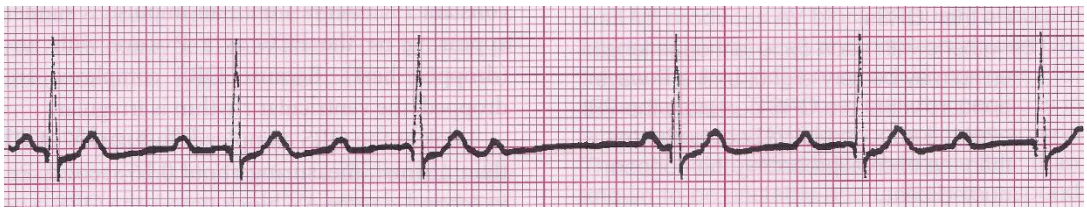

Figure 7. Mobitz type-I. The PR interval lengthens progressively until a beat is dropped.

Mobitz type-II

Mobitz type-II is also a form of intermittent heart block. Unlike type-I, however, there is no progressive slowing of conduction prior to a dropped beat (*figure 8*). In conducted beats, the PR interval remains constant (Hampton, 2013). On the ECG, its key features are:

- There are more P-waves than QRS complexes
- The PR interval does not changes from beat to beat
- Some P-waves are not followed by QRS complexes

- The rhythm is regularly irregular
- The QRS may be narrow, but is often broad.

Mobitz type-II is usually due to conduction failure within the bundle branches, and is a more sinister finding than Mobitz type-I. There is a significant risk that it will progress to third degree heart block (Vogler et al, 2012).

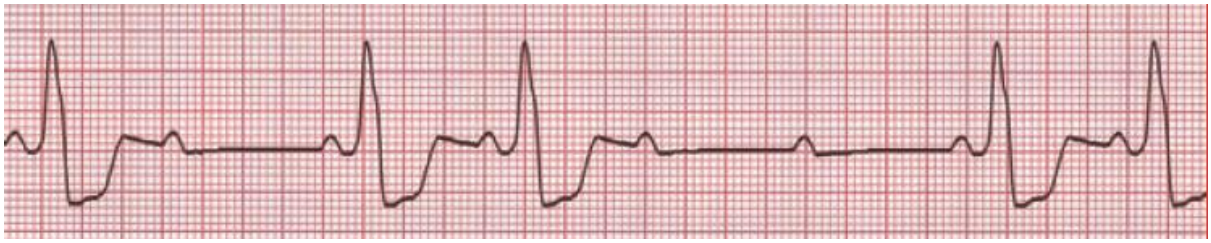

Figure 8. Mobitz type-II. The PR interval is constant. Note the frequent dropped beats, and broad QRS complex.

Second-degree AV block with 2:1 conduction

Second-degree AV block may occur in a pattern where every other beat is blocked, resulting in 2:1 conduction to the ventricles (*figure 9*). So is this Mobitz type-I or Mobitz type-II? It is impossible to say. The key feature that separates the two types of second-degree AV block is the progressive lengthening of the PR interval in type-I. In 2:1 block, we have only one PR interval to examine before conduction fails. Thus, we cannot say whether the PR interval is getting longer (Aehlert, 2011). This leaves us with a certain amount of prognostic uncertainty. Bennett (2013) suggests that 2:1 block with a broad QRS is likely to be type-II, and should be treated as such. Further testing, for example Holter monitoring, may be useful in determining which type of block is present (Vogler et al, 2012).

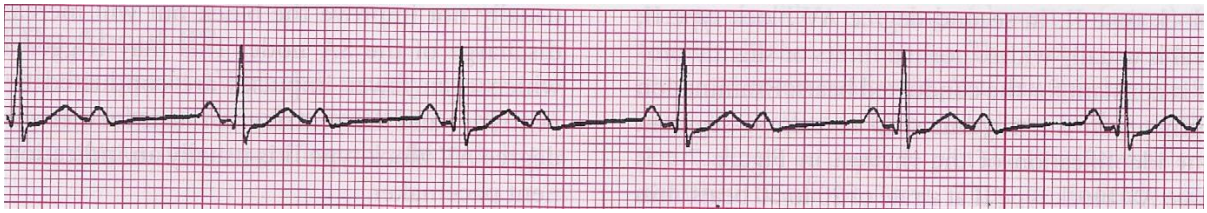

Figure 9. Second-degree AV block with 2:1 conduction. The QRS complex is narrow.

Third degree (complete) AV block

Third-degree AV block is also referred to as complete heart block. It occurs when there is no conduction between the atria and ventricles. Impulses arise in the sinus node as normal, and are conducted through the atria, creating regular P-waves on the ECG. Every impulse is blocked in the conduction system, however, and none arrive in the ventricles. In most cases, a subsidiary pacemaker below the block starts to fire, resulting in a slower ventricular escape rhythm. The QRS complexes may be broad or narrow, depending on the level of the block, and therefore the site of the subsidiary pacemaker (Garcia, 2015).

On the ECG, third-degree heart block appears as a regular rhythm (*figure 10*). There are regular P-waves, however these have no relationship with the QRS. In effect, there are two rhythms superimposed on each other; one from the sinus node, and one from the ventricles (Hampton, 2013). P-waves may distort T-waves or QRS complexes, or may be hidden by them. Mapping out the P-wave rhythm with a piece of paper can be useful in predicting where P-waves should be. The key ECG features of complete heart block are:

- P-waves are regular, but may be superimposed on the T-waves or QRS complexes
- QRS complexes are also regular, and occur at a slower rate than the P-waves
- There is no relationship between the P-waves and the QSR complexes
- The QRS may be narrow if the block is within the AV junction, but broad if it is lower down

Complete heart block is the most alarming of the AV blocks, and warrants immediate assessment and treatment because of the risk of haemodynamic compromise and cardiac arrest (Pitcher and Nolan, 2015).

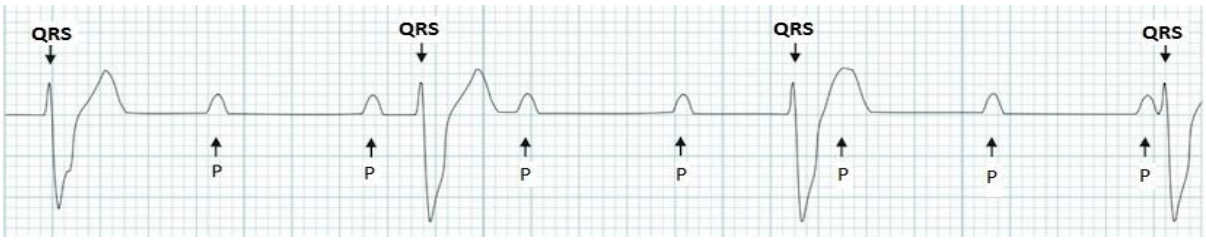

Figure 10. Third degree AV block. Note the regular P-waves and QRS complexes. There is no association between them. The QRS is wide, and the rate slow. There is a P-wave buried in the third T-wave.

Causes and treatment

As discussed above, high vagal tone may account for first-degree or Mobitz type-I AV blocks in younger, healthy people, in which case no treatment is required. In older people, these lower level AV bocks may be due to age related degeneration of the conduction tissues, or structural heart disease. Nonetheless, treatment is not generally necessary unless significant bradycardia is documented, and the patient is symptomatic (Bennett, 2013). In the acute setting, first-degree heart block and Mobitz type-I can occur during myocardial infarction (MI), in which case there is a risk of progression to higher degrees of block. These patients should be closely monitored (Aehlert, 2011).

Mobitz type-II and complete heart block are never benign findings. Both can cause significant bradycardia or result in sudden cardiac death as a result of asystole. As with sick sinus syndrome, idiopathic degeneration of the conduction system is a common cause, accounting for around 50% of cases (Vogler et al, 2010). Rarely, AV blocks occur as an isolated congenital abnormality, but more often are found in association with congenital heart disease or acquired disorders such as coronary heart disease, endocarditis and cardiac surgery (Tunaoglu et al, 2010). Drugs that alter cardiac conduction, for example beta-blockers, may also precipitate heart blocks, as may electrolyte disturbances and hypoxia (Fogoros, 2007). AV blocks are also a common complication of MI (Hreybe and Saba, 2009).

In the acute setting, any patient with second- or third-degree AV block should be assessed using an A-to-E approach, and placed under regular observation with continuous ECG monitoring. A 12-lead ECG should be recorded (Swift, 2013). Severe bradycardia is possible, which can cause significant haemodynamic compromise. Initial treatment in this case is atropine, given intravenously in 500 microgram doses, and repeated every three to five minutes to a maximum of 3 grams (Pitcher and Nolan, 2015). If atropine fails to increase the heart rate, temporary pacing should be initiated. Transcutaneous pacing may be tried initially, although it is not always successful, and is painful for the patient (Swift, 2013). A temporary transvenous pacing wire, placed via the femoral, brachial or jugular vein, is a more reliable alternative, but insertion requires advanced skills that are not available in every practice setting (McNaughton and Davison, 2013; Sharma et al, 2012). Transfer to a more specialised setting may be necessary.

In the longer term, patients who remain in Mobitz type-II or third-degree AV block will need permanent pacing. This may be delayed in acute conditions, such as MI, if there is a chance that normal conduction will return with recovery. Otherwise, a pacemaker should be implanted without delay, given the risk of collapse or sudden death (Bennett 2013).

Bundle Branch Blocks

A bundle branch block occurs when conduction through either the left- or right-bundle branch fails entirely. Because conduction continues normally in the other branch, every impulse from the atria is conducted to the ventricles. Thus, unlike the AV blocks, bundle branch blocks do not cause a change in heart rhythm (Houghton and Gray, 2014). To understand how they affect the appearance of the 12-lead ECG, we need to think about conduction through the ventricles, and how this creates the normal pattern of QRS complexes in the precordial leads.

R-wave progression

In the normal conduction system, the electrical impulse is rapidly conducted to every part of both ventricles by the bundle branches and extensive network of Purkinje fibres. This results in efficient distribution of the electrical impulse, and a narrow QRS complex (Klabunde, 2012). It also creates a particular pattern of QRS complexes that we associate with a normal 12-lead ECG. Let's look at this normal pattern in the precordial leads.

Figure 11 shows leads V1 to V6 from a normal 12-lead ECG. You can see that V1 has a small initial upwards deflection (an R-wave) followed by a deep downward deflection (an S-wave). As we move across the precordium towards V6, the R-wave grows in size, usually reaching its greatest height by V5. In contrast, the S-wave gradually diminishes in size. This normal pattern is referred to as 'R-wave progression'. The 'transition' point at which the R-wave exceeds the S-wave is usually between V3 and V4 (Garcia, 2015).

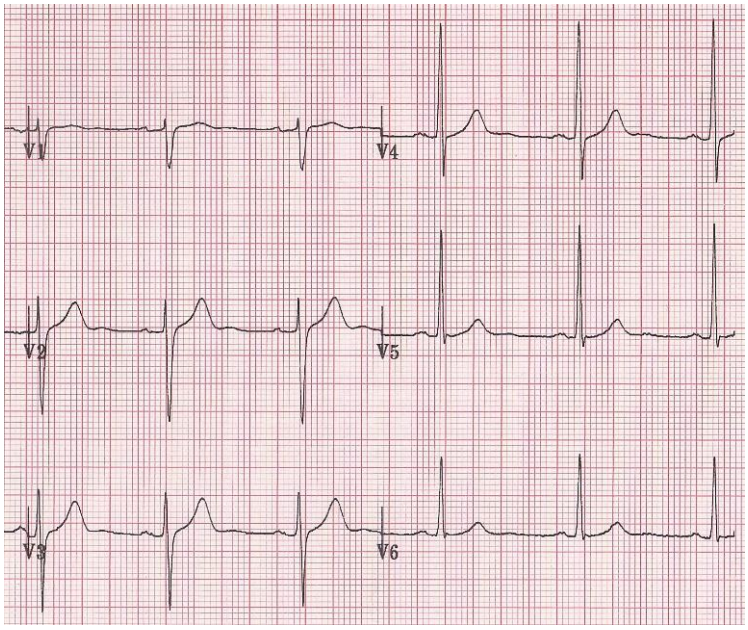

Figure 11. Normal R-wave progression

To understand why the QRS complexes change this way, we need to consider several important concepts. Firstly, an electrical wavefront moving towards a lead creates a positive ECG deflection, while one moving away creates a negative one. Secondly, although the depolarisation wave travels through the heart in many directions, the largest wavefront will dominate the ECG, and dictate the orientation of the ECG complexes. The left ventricle has much thicker walls than the right, and therefore generates more electricity. The predominant direction of the depolarisation wave is therefore towards the left, away from V1 and towards V6, as shown in *figure 12* (Hampton, 2013).

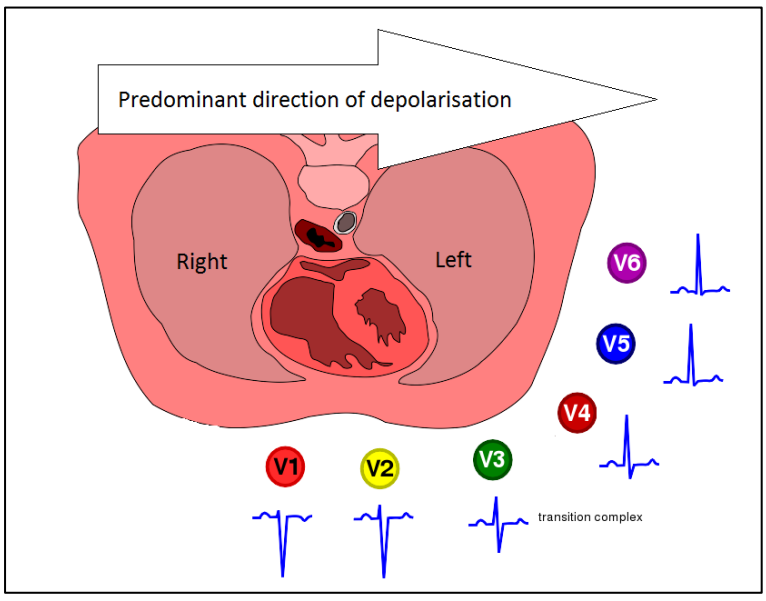

Figure 12. R-wave progression in context.

Abnormal ventricular depolarisation

In bundle branch blocks, only one ventricle is depolarised normally. The other ventricle has to wait for the electrical impulse to spread across from the other side of the heart, through the muscle cells. This is much slower than depolarisation via a normal conduction system, and hence the QRS complex widens. This abnormal depolarisation also changes the normal pattern of QRS complexes on the ECG, most obviously in the precordial leads (Bennett, 2013).

Figure 13 shows a typical example of right-bundle branch block (RBBB). The first thing to note is the width of the QRS complex. This is around 140 milliseconds, or three and a half small squares. In QRS width, small differences are important so this is abnormally wide. If we look at the precordial leads, we can see that the R-wave progression is also abnormal, with a very tall R-wave in V1 and V2. The QRS complex in these leads has an RSR pattern, which is sometimes described as M-shaped, or like rabbit ears (Aehlert, 2011). The QRS goes upward (R-wave) then drops back (S-wave) before climbing again (another R-wave). If we look across at the left-sided precordial leads, and particularly at V6, we see a wide S-wave following the R-wave. Both the RSR in V1 and/or V2, and the wide S-wave in V6, are characteristic features of RBBB (Surawicz et al, 2009). The final thing to note is the T- wave inversion in the right precordials (V1 to V3). T-wave inversion, and ST-segment abnormalities, are also common features of bundle branch blocks (Garcia, 2015).

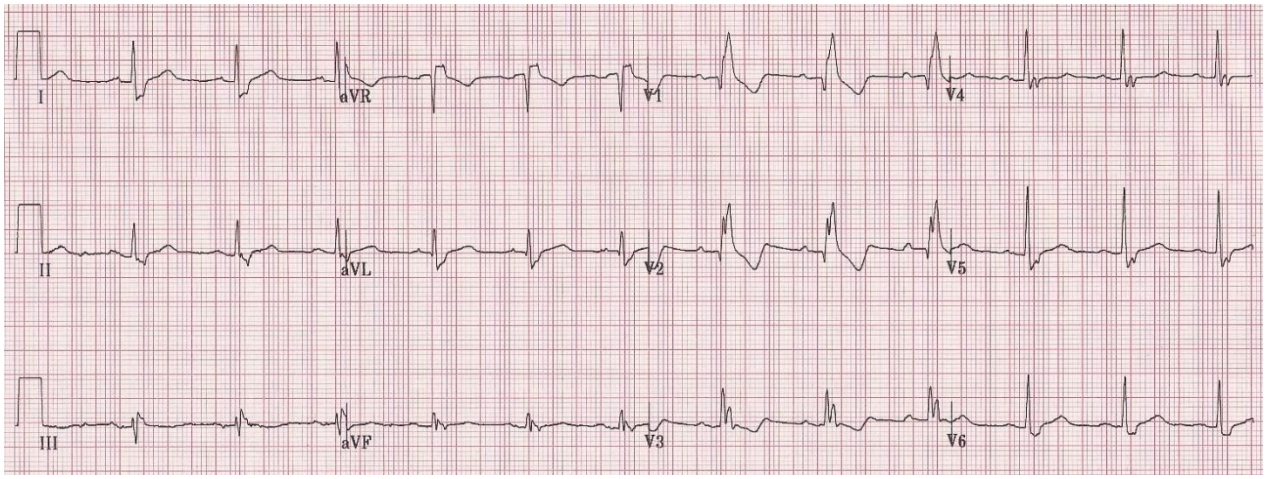

Figure 13. Right-bundle branch block. Note the RSR in V1/V2, and wide S-wave in V6. The QRS is broad.

Figure 14 shows left-bundle branch block (LBBB). The QRS is again wide, and in this example is over 170 milliseconds (more than four small squares). Superficially, the R-wave progression appears more normal than in RBBB. We start out with a small R-wave and deep S-wave in V1 as you would expect, however there is no growth of the R-wave as we move through V2 and V3, and the S-waves remain deep and wide. There is a sudden transition to a positive QRS in the left sided precordial leads. The upright QRS complexes in V5 and V6 are short, stumpy, and notched. These are typical findings in LBBB (Kumar et al, 2013). If you look at the QRS in V6 you can see that the QRS is also monophasic – it is all R-wave, with no preceding q wave. The upright QRS complexes in the left precordials also have inverted T-waves. These are also common findings (Garcia, 2015). *Box 2* compares the principal features of left and right bundle branch block.

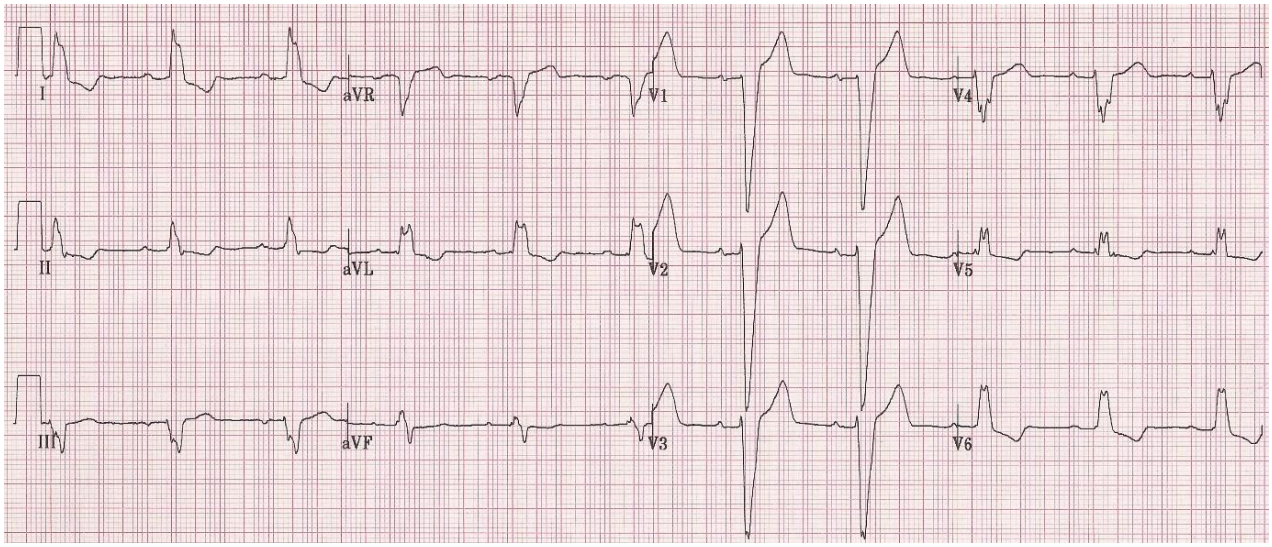

Figure 14. Left-bundle branch block. Note the deep S-waves in V1-V3, and notched, monophasic R-wave in V6. The QRS is broad.

	Right bundle branch block	**Left bundle branch block**
QRS width	> 120 milliseconds	> 120 milliseconds
V1 / V2	RSR pattern T-waves often inverted	Deep S-waves
V6	Wide S-wave	Tall, monophasic R-wave T-waves often inverted

Box 2. Comparison of left- and right-bundle branch blocks

Some people find the mnemonic "William Marrow" helpful in remembering bundle branch blocks. William has two Ls in the middle, and represents LBBB. The W is the wide, deep S-wave in V1, and the M is the stumpy and notched R-wave in V6. Marrow, with its two Rs in the middle, is RBBB. The M is the RSR in V1, and the W at the end is the wide S-wave in V6 (Houghton and Gray 2014).

Causes and treatment

The left-bundle branch is a much more extensive structure than the right, and receives a blood supply from both the left and right coronary arteries (Josephson, 2010). Although LBBB may be caused by age related degeneration of the conduction system, more often it results from disease processes affecting the left ventricle. These include hypertension, valvular heart disease, cardiomyopathy, myocarditis and coronary heart disease (Kumar et al, 2013). Any patient presenting with previously unknown LBBB should therefore be evaluated for an underlying cause. Unlike AV blocks, bundle branch blocks do not cause bradycardia or asystole, so do not generally require emergency treatment. There are, however, a number of clinical scenarios in which LBBB influences treatment. The first of these is suspected MI.

The ECG is an important tool in the assessment of patients presenting with a history of chest pain. Abnormalities in the T-waves and ST-segments are suggestive of ischaemia, and are diagnostic in ST

elevation MI (Steg et al, 2012). These changes may be masked by the ST and T-wave abnormalities that normally occur in LBBB (Bennett, 2013). In a patient with chest pain and LBBB, it is often unclear whether ECG changes are due to ischaemia, or simply the result of conduction block, and in clinical practice less than half of these patients are found to have an occluded coronary artery (Jain et al, 2011; Kontos et al, 2011). Various criteria to differentiate normal LBBB from ischaemic LBBB have been suggested, of which the Sgarbossa is the most widely evaluated (Sgarbossa et al, 1996). Unfortunately, although this criterion has a high specificity, its sensitivity is low, meaning that patients who are in fact having an infarct may be missed (Tabas et al, 2008). As a result, current guidelines do not suggest its use, and instead recommend prompt percutaneous coronary intervention (PCI) in any patient who presents with new LBBB and a history consistent with acute MI (Steg et al, 2012).

The second situation in which LBBB has particular clinical relevance is heart failure. Delayed depolarisation of the left ventricle results in mechanical dyssynchrony. In other words, the right ventricle contracts before the left. This reduces the efficiency of the heart, and lowers cardiac output (Kumar et al, 2013). In people with well-preserved ventricular function, this has limited significance. In patients with heart failure and poor ejection fraction, it can cause a critical decline in pump function, and increased symptoms (O'Donovan, 2009). People with heart failure and reduced ejection fraction, complicated by LBBB, often derive symptomatic benefit from cardiac resynchronisation therapy. In this treatment, a biventricular pacemaker is implanted so that both ventricles can be paced simultaneously (National Institute for Health and Care Excellence, 2014).

The right-bundle branch is a long, slender structure, with a blood supply from the left coronary artery only. It is more prone to damage than the left bundle branch, and thus RBBB is a more common finding on the ECG (Josephson, 2010). Pathological causes of RBBB include coronary heart disease, congenital heart disease (especially Ebstein's anomaly and atrial septal defect), and pulmonary embolus (Houghton and Gray, 2014). Unlike LBBB, RBBB is also found in otherwise normal hearts, in which case it is often considered a benign finding (Bennett, 2013). A recent study has suggested that this is not always the case. Bussink et al (2013) demonstrated a 30% increase in mortality in people with RBBB but no known heart disease at initial evaluation. The increase in mortality was attributed to the later development of cardiovascular disease, suggesting that RBBB may be an early marker of disease in some individuals.

Incomplete bundle branch block

In incomplete bundle branch block, conduction down one bundle branch is delayed but not completely blocked (Houghton and Gray, 2014). This results in the characteristic features of RBBB or LBBB, but the QRS complex remains relatively narrow at between 100 and 120 milliseconds (Surawicz et al, 2009). Houghton and Gray (2014) suggest that the causes of incomplete bundle branch block are the same as those of complete bundle branch block. Bussink et al (2013), however, suggest that incomplete right bundle branch block is usually benign. It was not associated with increased mortality in their study.

Conclusion

Both congenital and acquired disease can cause abnormalities in cardiac conduction, leading to conduction blocks. In some cases, they also occur in the absence of identifiable disease. Block may occur in the sinus node, AV junction or bundle branches, resulting in a range of ECG abnormalities. Careful evaluation of both heart rhythm, and QRS morphology across the precordium, is necessary for their identification. Some blocks, such as first-degree AV block, are relatively benign, while others have the potential to cause severe haemodynamic compromise or cardiac arrest. Systematic clinical assessment, and appropriate monitoring, are therefore just as important as skills in ECG interpretation. Nurses must also have a working knowledge of the emergency treatment of bradycardia to care for these patients safely. For practitioners working in autonomous roles, recognising the relationship between conduction blocks and heart disease is also important from a screening standpoint.

This article concludes our review of the first step in ECG interpretation; the evaluation of rate, rhythm and intervals. We will be returning to bundle branch blocks in two months' time, when we discuss the cardiac axis and hemiblocks. Next month, we turn our attention to the next step in our system of interpretation; evaluating the ECG for signs of pre-excitation.

Key points

- Conduction blocks can occur in all parts of the conduction system, causing a variety of ECG changes. They may be congenital, acquired, idiopathic, or physiological. Age related degeneration of the conduction system, and coronary heart disease, are common causes.

- Sinoatrial block occurs when the electrical impulse is unable to leave the sinus node. It results in a pause on the ECG, and is often found in conjunction with other features of sick sinus syndrome. A permanent pacemaker is required in symptomatic patients.

- Atrioventricular (AV) blocks occur when the impulse is delayed or blocked in the AV node, bundle of His or bundle branches. It may be first-, second- or third-degree in nature, depending on whether the electrical impulse is delayed, intermittently blocked, or always blocked.

- First-degree, and Mobitz type-I AV blocks may be benign, especially in younger people and at night. In older people, they are more often pathological, and in the acutely unwell person can rapidly progress to higher degrees of block.

- Mobitz type-II and third degree AV block are associated with bradycardia and asystole, and may cause haemodynamic compromise or cardiac arrest. Systematic patient assessment is essential, followed by emergency treatment if necessary. This may include atropine and/or temporary pacing. In the longer term, permanent pacing is often required.

- Bundle branch blocks do not affect heart rate or rhythm, but are often markers of underlying heart disease, especially in the case of left bundle branch block (LBBB). LBBB may also indicate acute myocardial infarction, requiring primary percutaneous coronary intervention. In heart failure, LBBB may cause mechanical dyssynchrony. This can be treated with resynchronisation therapy.

References

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

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

Bussink BE, Holst AG, Jespersen L, Deckers JW, Jensen GB, Prescott E (2013) Right bundle branch block: prevalence, risk factors, and outcome in the general population: results from the Copenhagen City Heart Study, *European Heart Journal*, 34, 138–146

Cheng S, Keyes MJ, Larson MG, McCabe EL, Newton-Cheh C, Levy D, Benjamin EJ, Vasan RS, Wang TJ (2009) Long-term Outcomes in Individuals With Prolonged PR Interval or First-Degree atrioventricular Block, *JAMA*, 301(24), 2571-2577.

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

Ewy GA (2014) Sick sinus syndrome: synopsis, *J Am Coll Cardiol,* 64, 539-40.

Fogoros RN (2007) *Antiarrhythmic drugs, a practical guide*, 2nd edition, Oxford, Blackwell Publishing.

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

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

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

Hreybe H & Saba S (2009) Location of Acute Myocardial Infarction and Associated Arrhythmias and Outcome, *Clinical Cardiology*, 32(5), 274-277.

Jain S, Ting HT, Bell M, Bjerke CM, Lennon RJ, Gersh BJ, Rihal CS, Prasad A (2011) Utility of Left Bundle Branch Block as a Diagnostic Criterion for Acute Myocardial Infarction*, Am J Cardiol*, 107, 1111–1116

Jensen PN, Gronroos NN, Chen LY, Folsom AR, deFilippi C, Heckbert SR, Alonso A (2014) Incidence of and Risk Factors for Sick Sinus Syndrome in the General Population*, J Am Coll Cardiol,* 64, 531–8.

Josephson L (2010) Hypertrophies and Intraventricular Conduction Defects: Causes, Presentation, and Significance, *Dimens Crit Care Nurs*, 29(6), 259-275.

Jowett NI & Thompson DR (2007) *Comprehensive coronary care*, 4th edition, London: Bailliere Tindall.

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

Kontos MC, Aziz HA, Chau VQ, Roberts CS, Ornato JP, Vetrovec GW (2011) Outcomes in patients with chronicity of left bundle branch block with possible acute myocardial infarction, *Am Heart J,* 161, 698-704.

Kumar V, Venkataraman R, Aljaroudi W, Osorio J, Heo J, Iskandrian AE, Hage FG (2013) Implications of Left Bundle Branch Block in Patient Treatment, *Am J Cardiol*, 111, 291-300.

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

National Institute for Health and Care Excellence (2014) Implantable cardioverter defibrillators and cardiac resynchronisation therapy for arrhythmias and heart failure (review of TA95 and TA120). NICE technology appraisal guidance 314. https://www.nice.org.uk/guidance/ta314 (Accessed 29/12/2015).

McNaughton A (2013) Temporary pacing: clinical indications and techniques, *British Journal of Cardiac Nursing*, 8(6). 289-292.

Monfredi O, Dobrzynski H, Mondal T, Boyett MR & Morris GM (2010) The Anatomy and Physiology of the Sinoatrial Node—A Contemporary Review, *PACE,* 33, 1392–1406.

Mymin D, Mathewson FA, Tate RB, Manfreda J (1986) The natural history of primary first-degree atrioventricular heart block. *N Engl J Med*. 315(19), 1183-1187.

O'Donovan K (2009) Cardiac resynchronization therapy: Biventricular pacing for heart failure, *British Journal of Cardiac Nursing,* 4(9), 406-414.

Packard JM, Graettinger JS, Graybiel A. (1954) Analysis of the electrocardiograms obtained from 1000 young healthy aviators; ten year follow-up. *Circulation*. 10(3), 384-400.

Pitcher D, Nolan J (2015) *Peri-arrest arrhythmias.* Resuscitation Council UK, London. http://tinyurl.com/ogeh2jt (accessed 11 December 2015)

Sampson M (2016) Understanding the ECG. Part 3: Arrhythmias, *British Journal of Cardiac Nursing* **11**(1):

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

Sgarbossa EB, Pinski SL, Barbagelata A, Underwood DA, Gates KB, Topol EJ, Califf RM, Wagner GS (1996) Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle branch block. GUSTO-1 (Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries) Investigators, *N Engl J Med,* 334, 481-487.

Sharma S, Sandler B, Cristopoulos C, Saraf S, Markides V & Gorog DA (2012) Temporary transvenous pacing: endangered skill, *Emerg Med J*, 29, 926-927.

Steg PG, James SK, Atar D, et al (2012) ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: the Task Force on the management of ST-segment elevation acute myocardial infarction of the European Society of Cardiology (ESC), *Eur Heart J,* 33, 2569–619.

Surawicz B, Childers R, Deal BJ, Gettes LS (2009) AHA/ACCF/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram. Part III: Intraventricular Conduction Disturbances A Scientific Statement From the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society Endorsed by the International Society for Computerized Electrocardiology, *J Am Coll Cardiol*, 53(11), 976-981.

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

Tabas JA, Rodriguez RM, Seligman HK, Goldschlager NF (2008) Electrocardiographic criteria for detecting acute myocardial infarction in patients with left bundle branch block: a meta-analysis. *Ann Emerg Med*, 52, 329 –336.

Tunaoglu FS, Yildirim A & Vurali (2010) Isolated congenital heart block, *Tex Heart Inst J*, 37(5), 579-583.

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