How to read an electrocardiogram (ECG). Part 2: Abnormalities of electrical conduction

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Introduction

This is the second in a series of articles that aim to help readers to understand and interpret recordings of the surface ECG. The first article introduced the basic principles of the ECG including the electrophysiology of the heart and the features of a normal ECG (1). This one describes some of the common abnormalities of electrical conduction which can be seen on the ECG.

Electrical conduction and its abnormalities

Contraction of the heart muscle occurs in response to electrical depolarisation – the rapid spread of electrical activity throughout the myocardium which is facilitated by specialised conduction tissue. This process normally begins with spontaneous depolarisation of cells in the **sinus node**, situated in the right atrium (RA), then proceeds quickly through the atria to the **atrioventricular node**, and then down the bundle of His to the left and right bundle branches and into the ventricular myocardium. See figure 1.

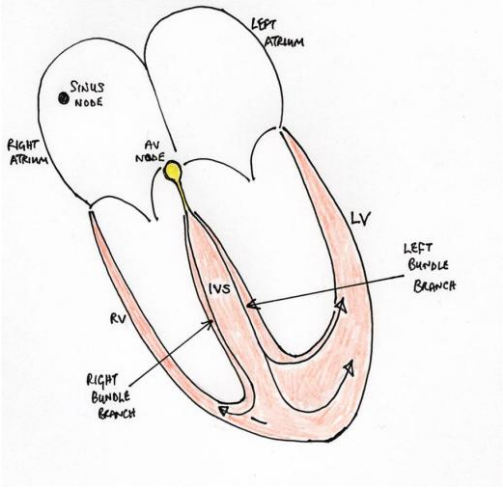

Figure 1. The normal conduction system of the heart.

This process of electrical conduction can be disrupted at any point throughout the specialised conduction system, but most commonly occurs in the sinus node itself, the atrioventricular (AV) node or in the left and right bundle branches. Conduction can be disrupted by a variety of disease processes including ischaemia or infarction, infiltrative disorders or even age-related degeneration.

Depending upon the site and severity of a conduction abnormality, the clinical consequences may vary from no noticeable effect at all (asymptomatic) through to sudden death from asystole. Commonly however patients with conduction system disease present with symptoms such as exercise intolerance or dyspnoea, transient episodes of impaired consciousness (presyncope) or complete loss of consciousness (syncope). Therefore it is important to be able to recognise conduction defects on ECG when investigating patients who present with these symptoms.

Sinus node disease

One of the characteristic features of specialised cardiac conduction tissue is its tendency to depolarise spontaneously. Under normal circumstances, the rate of this spontaneous depolarisation is fastest in the sinus node and hence the rate and rhythm of the heart is normally dictated by this phenomenon – **sinus rhythm**. The rate of depolarisation is influenced by the autonomic nervous system, speeding up under the influence of sympathetic drive during periods of exertion or stress and slowing down during periods of rest or sleep.

With increasing age it is common for the sinus node to lose some of its ability to depolarise spontaneously or to respond to autonomic nerve influence. So sinus rates may be slower than required, or perhaps may cease altogether. This may result in failure of the heart rate to rise adequately with exercise (chronotropic incompetence) leading to exercise intolerance and dyspnoea, and in severe cases may cause transient incapacity or collapse.

Sinus node disease can be manifested on the ECG as sinus bradycardia, sinus pauses, or even prolonged sinus arrest and transient asystole.

Sinus bradycardia may cause fatigue or even heart failure, and significant pauses in sinus node activity (sinus arrest) can lead to transiently impaired consciousness – presyncope if partial, syncope if complete. See figures 2 and 3.

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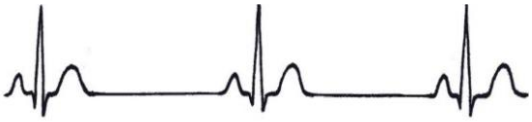

Figure 2. Sinus bradycardia.

Figure 3. A short sinus pause, followed by the return of sinus rhythm.

If the sinus node fails to depolarise however, the AV node is usually that part of the conduction tissue with the next fastest rate of spontaneous depolarisation and will usually take over and dictate the cardiac rhythm itself – hence the so-called **'nodal rhythm'**. It is because of this nodal 'escape rhythm' that isolated sinus node disease does not usually lead directly to death, even though it may cause significant symptoms including syncope. See figure 4.

Figure 4. A short sinus pause, followed by an escape beat arising from the AV node; note that the QRS complex is of normal morphology because although spontaneous depolarisation commences in the AV node on this occasion, subsequent conduction through the ventricles occurs in the normal way.

Atrioventricular (AV) block

Increasing age, along with other common conditions such as ischaemia or myocardial infarction, can lead to damage to the AV node or the more distal conduction tissues such as the bundle of His and the right and left bundle branches.

Damage to these structures can lead to delay or even complete failure of conduction to the ventricles – this phenomenon is called **AV block**. Depending upon the site and severity of damage to the conduction system, there are varying degrees of AV block and these result in different patterns of abnormality recognisable on the ECG.

In the mildest form of AV block, **first degree AV block**, the AV node simply conducts more slowly than usual, but with no failure of conduction. During normal sinus rhythm the wave of electrical depolarisation spreading from the atria is slightly delayed in the AV node, which is manifested on the ECG as the PR interval. In first degree AV block the delay in the AV node is simply longer than normal, which in turn leads to prolongation of the PR interval on the ECG. This long PR interval (> 200 ms) is the hallmark of first degree AV block.

As disease in the AV node progresses, there comes a point when conduction through the AV node is not only delayed but may fail completely. Often a pattern of gradually increasing PR intervals with each successive heartbeat is seen until finally one P wave fails to conduct to the ventricles at all - after which the AV node conduction recovers and the whole sequence starts over again. This is called **Mobitz type 1** AV block (also known as Wenckebach block) and is the mildest form of **second degree AV block**. See figure 5.

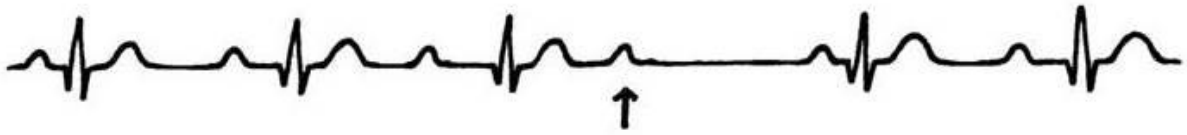

Figure 5. Mobitz Type 1 second degree AV block (also known as Wenckebach block); note that the PR interval increases with each beat until eventually there is a non-conducted P wave (arrowed) and then this pattern repeats itself.

With more severe disease of the AV node, conduction may suddenly fail even without any prior prolongation of the PR interval. This may occur with variable frequency. Sometimes it occurs just once, before normal conduction then resumes, or perhaps after alternate P waves (so-called 2 to 1 AV block). Sometimes there may be several non-conducted P waves in succession before one is then conducted normally – hence this can be 3 to 1, 4 to 1, or 5 to 1 block depending on the pattern. These are all examples of **Mobitz type 2** AV block, which is a more severe form of **second degree AV block** (see figure 6).

Figure 6. Mobitz Type 2 second degree AV block; on this occasion only 1 P wave in 3 is conducted to the ventricles, but note that each conducted beat has a normal, and constant, PR interval.

Mobitz type 2 is distinguished from Mobitz type 1 AV block by the fact that there is no prolongation of the PR interval in type 2. In other words there is complete non-conduction of the affected P waves, and those P waves that *are* conducted to the ventricles do so with a normal PR interval.

The most severe form of AV block is characterised by complete failure of any conduction from atria to ventricles – this is called complete or **third degree AV block**. The site of such AV block may be within the AV node or in the more distal conduction tissues - but the hallmark of this condition on the ECG is the complete lack of a consistent relationship between P waves and QRS complexes.

One may wonder, if there is absolutely no conduction possible from atria to ventricles, why ventricular asystole does not occur, but the reason is that usually an **escape rhythm** takes over. This is the heart's safety mechanism. If no signals reach the AV node from the atria, the AV node itself may spontaneously depolarise (as may happen in sinus node disease, described above), and in this way the ventricles may continue to contract and to preserve life. If the AV takes over the heart rhythm in this way, the ventricles will still depolarise normally via the His bundle and the right and left bundle branches and the QRS complex on the ECG will be normal. However, if the AV node fails to depolarise the patient has to rely on some more distal conduction tissue taking over this escape rhythm (usually slower and less reliable than an AV nodal escape rhythm). In this case conduction through the ventricles may be abnormal and give rise to a broad QRS complex. Whatever the level of the escape rhythm though, there is never any relationship between the P waves and QRS complexes in third degree block – a phenomenon known as **AV dissociation**. Note also that in third degree AV block the P wave rate is always faster than the QRS rate (see figure 7).

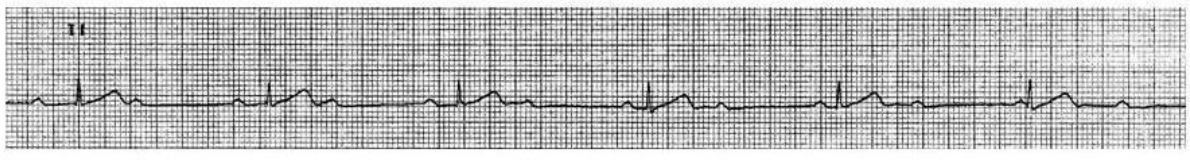

Figure 7. Third degree (also known as 'complete') AV block; note that there is no consistent relationship between the P waves and the QRS complexes – this is known as AV dissociation.

Bundle branch block

Sometimes disease of the conduction system is confined to either the **left** or the **right bundle branches** and this results in a characteristic pattern of the QRS complexes on a 12-lead ECG.

Keeping in mind the normal specialised conduction system (see figure 1) it can be appreciated that if, for example, conduction down the left bundle branch is blocked, then the wave of electrical depolarisation has to travel first down the right bundle branch and can then only reach the left ventricular myocardium via non-specialised conduction tissue. In other words the left ventricle is depolarised via an abnormal route, which takes longer than usual and therefore results in prolongation of the QRS complex on the ECG. This abnormal sequence of electrical depolarisation of the ventricles, with the right ventricle depolarised before the left (instead of the two together as normal) is what causes the characteristic ECG pattern known as **left bundle branch block** (LBBB). See figure 8.

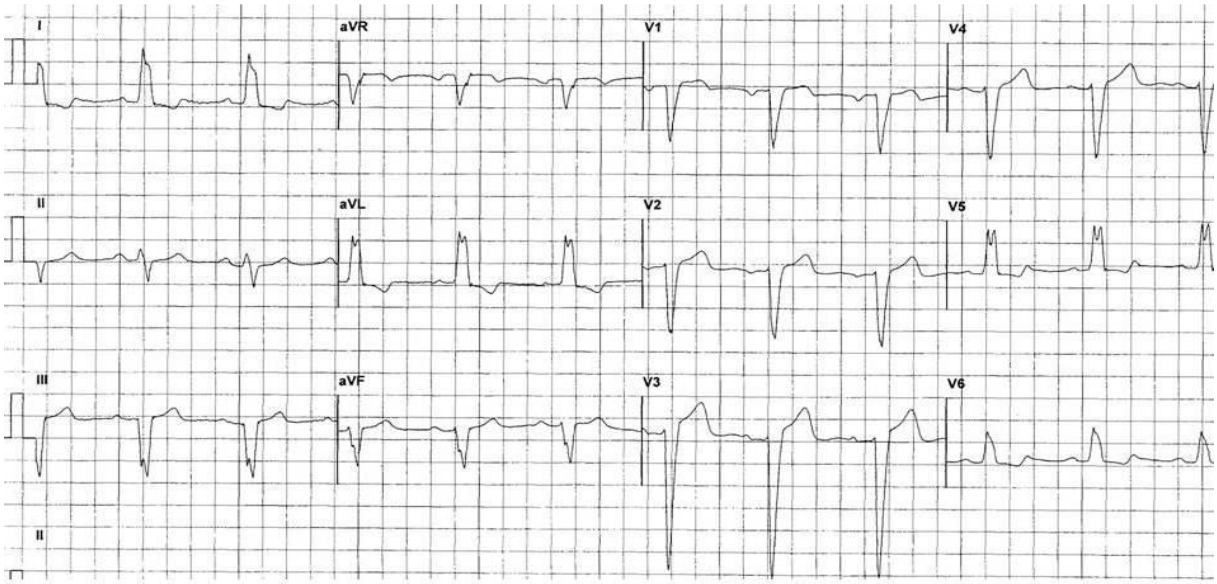

Figure 8. Sinus rhythm with Left Bundle Branch Block (LBBB).

Conversely, if conduction down the right bundle branch is blocked, then the wave of electrical depolarisation has to travel first down the left bundle branch before it can then reach the right ventricle. In this case the right ventricle is depolarised later and via an abnormal route, again taking longer than usual and resulting in a characteristic broadened QRS complex pattern known as **right bundle branch block** (RBBB). See figure 9.

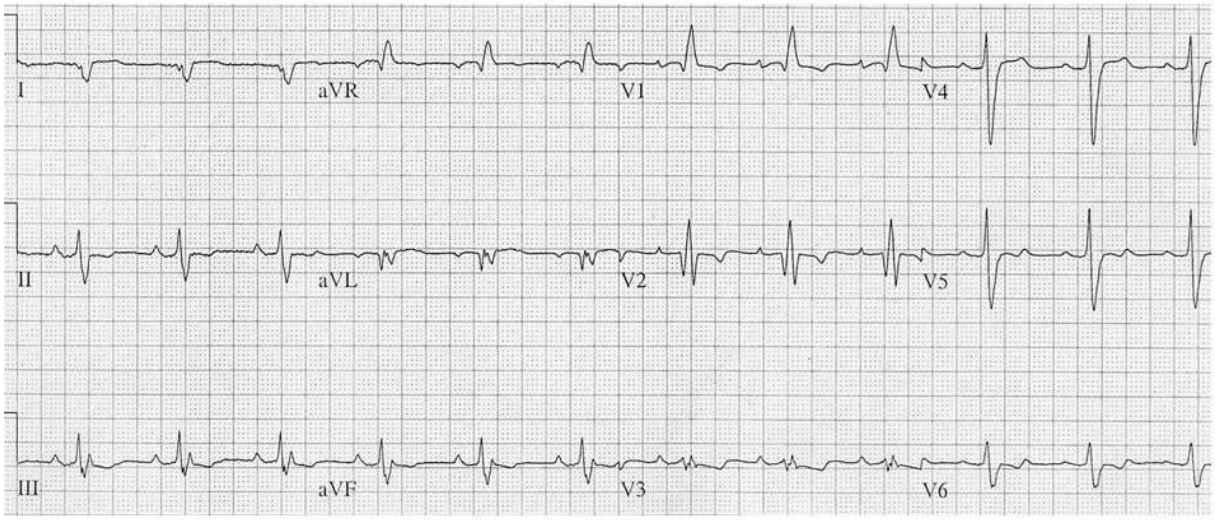

Figure 9. Sinus rhythm with Right Bundle Branch Block (RBBB).

References

1. Price D. How to read an Electrocardiogram (ECG). Part One: Basic principles of the ECG. The normal ECG. Southern Sudan Medical Journal 2010; 3(2) 26-28