SYMPTOMATIC BRADYCARDIA
1: PHYSIOLOGY AND CAUSES

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ABSTRACT Watson, D. (2008) Symptomatic bradycardia 1: physiology and causes. Nursing Times; 104: 38, 26–27. This is the first in a two-part unit on managing bradycardia. It discusses the physiology of the condition and examines its main causes. It also explores assessment through interpretation of ECGs or rhythm strips.

INTRODUCTION
The current prevalence of heart disease may mean practitioners frequently care for patients with symptomatic bradycardia. While many are persistently bradycardic and totally asymptomatic, this article aims to address the management of patients who are symptomatic or show adverse signs.

ANATOMY AND PHYSIOLOGY
Several key areas of the heart are identified when reviewing conduction of the electrical impulses that generate contraction of the myocardium. The main areas are the sinoatrial node (SA), atrioventricular node (AV), the atrioventricular bundle of His left and right bundles, and finally the Purkinje fibres (Sinnatambay, 2006).

The SA node is the main pacemaker of the heart in most patients. It is located in the wall of the right atrium, just below the superior vena cava. Normally it generates electrical impulse at a rate of 60–100 beats per minute (bpm) (Resuscitation Council UK, 2006). It should be noted, however, that any cells within the cardiac muscle can potentially initiate an electrical impulse. The impulse generated from the SA node travels to the AV node, which is located in the right atrium, on the interatrial septum (Sinnatambay, 2006). This is identified on the ECG as the P wave. The AV node usually has the ability to generate a rate of 50–60bpm in normal patients. The impulse then travels from the AV node to the AV bundle – this can be found along the inferior border of the interventricular septum. The AV bundle plays an important role in conduction as the atria and ventricles are separated by a fibrous band. This is the only means in the normal heart that facilitates conduction of the contractile impulse from the atrium to the ventricle. This divides into the left and right bundles. The right bundle runs along the septum, surfacing on the right side of the sternum and then continues to the Purkinje fibres. The left bundle separates when it reaches the septal endocardium of the left ventricle and spreads out over the ventricle (Sinnatambay, 2006).

The conduction system depends on a good blood supply to maintain normal function. The SA node in about 60% of patients receives blood from the SA nodal artery, a derivative of the right coronary artery. In the other 40% the supply comes from the circumflex artery (a derivative of the left coronary artery) (Sinnatambay, 2006).

CAUSES OF BRADYCARDIA
Although the textbook definition of bradycardia is a pulse rate below 60bpm in adults, most patients are asymptomatic with the higher bradycardic rates. This is because ventricular filling increases in those who are able, allowing compensation for the reduced rate. In the long term this increased ventricular filling can over-extend the ventricles (Woodrow, 2001).

Practitioners should treat the patient and not the ECG – patients must be assessed thoroughly and the ECG taken into account in context. Rang and Dale (2007) proposed that basic phenomena underpin cardiac arrhythmias. These are delayed repolarisation (recovery of the resting potential of the cells); re-entry impulses; ectopic pacemaker activity; and heart block. The remainder of this article focuses on bradycardia and heart block.

There are many potential causes of bradycardia. Some are directly cardiac-related, while others have a consequential effect on the cardiac muscle or conduction. These include:
- Ischaemic heart disease;
- Digoxin;
- Hypoxia;
- Metabolic disturbances;
- Myocardial infarction;
- Beta-blockers;
- Cardiomyopathy.

Sinus bradycardia is usually left untreated unless patients are symptomatic. In deciding on possible treatment it is useful to establish what the rhythm is, as it could be first or second-degree heart block, complete heart block or simply bradycardia.

First-degree heart block
This manifests as a sustained prolongation of the P-R interval when looking at the rhythm strip. The normal interval (time from onset of P wave to start of QRS complex) is up to five small boxes (0.2 seconds); any longer is classed as prolonged. This is due to delayed atrioventricular conduction.

However, every impulse is still conducted, so every P wave (representing atrial contraction) is followed by a QRS complex (representing ventricular contraction). First-degree heart block is usually left untreated as most patients are asymptomatic (Mangrum, 2000). It is good practice to compare current ECG with previous ECGs.

Second-degree heart block
This manifests differently on the rhythm strip. Unlike first-degree heart block, not every impulse is conducted down through the ventricles. On the rhythm strip, this is displayed as a P wave without a reciprocal QRS complex. In clinical practice second-degree heart block has various terms

LEARNING OBJECTIVES
1. Be able to discuss the conduction system of the heart.
2. Know how to differentiate between various types of heart block.
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cells within the ventricles will attempt to generate impulses. Earlier, any cells within the myocardium can therefore no warning that conduction has occurred. This is due to failure of the atrioventricular node conduction pathway. Frequently, this pattern then begins again. Usually this is only observed and left untreated if patients are asymptomatic (RCUK, 2006).

However, unlike type 1, in type 2 heart block, there is no gradual prolongation of the PR interval. There is a constant PR interval, and therefore no warning that conduction may be inhibited, resulting in the absence of a QRS complex as failure to conduct the P wave occurs. This unpredictability makes the rhythm higher risk as it carries the risk of ventricular standstill or asystole and is an indication for pacing.

Complete heart block

Third-degree heart block (also known as complete heart block) demonstrates no recognisable relationship between the P waves and the QRS complex. This means there is no coordinated activity between the atrium and the ventricles. As identified earlier, any cells within the myocardium can generate impulses. In complete heart block, cells within the ventricles will attempt to generate an impulse due to the failure of the P wave to conduct down through the normal channels. A rate of 30–40bpm may be present (RCUK, 2006).

IDENTIFYING RHYTHM

Inexperienced practitioners can find rhythm strips or ECGs difficult to interpret. However, most rhythms can be identified by following a step-wise approach to interpretation. The first step, as taught in advanced life support training, is to identify if there is any electrical activity present (RCUK, 2006). An absence could be asystole. However, a pulse check should be made and all connections and the gain on the ECG machine should be maximised. No electrical activity without any palpable pulse is asystole and immediate resuscitation should be commenced.

The second step is to identify the QRS or ventricular rate. There are a variety of ways to calculate this. Normal ECG paper is calibrated in mm, with five large boxes accounting for one second. A rapid way to count the rate in a regular rhythm is to count the number of large squares between two consecutive QRS complexes; this is then divided by 300, giving the number of beats per minute. However, this is not as accurate in rhythms identified as irregular, so it is better to count the number of small boxes between two consecutive QRS complexes and divide this by 1,500.

Step three is to ascertain if the rhythm is regular or irregular. Initially this may appear easy. However, the faster the heart rate, the more difficult this can become to identify. One method of identifying the regularity of a strip is to mark an R-R interval on a piece of paper and superimpose this on other R-R intervals throughout the rhythm strip; if it is regular these should match up. In strips or ECGs identified as irregular, analysis should establish if these are regularly irregular, such as in second-degree heart block type 1, or irregularly irregular, as in atrial fibrillation.

Step four involves identifying if the QRS complex width is normal (less than three small squares or 0.12 seconds) or prolonged (greater than three small squares or 0.12 seconds). If the QRS width is less than three small squares, the rhythm is fractionally irregular. In most cases, originates from above the ventricle. If the width is greater than three small squares, the rhythm originates from somewhere in the ventricles or some form of bundle branch block may be present.

Step five considers whether atrial activity is present. This is identified by the presence of P waves, and is not always possible to identify. Atrial fibrillation results in the absence of P waves on the rhythm strip. Furthermore, the faster the rate, the more difficult it is to identify P waves as they sometimes become obliterated by QRS and T waves. Wherever possible, a 12-lead ECG should be obtained as this frequently allows identification of P waves if present in at least one lead.

The final step is to relate any atrial activity present with the ventricular activity. This step allows practitioners to identify what type of heart block is present (if any). The aim is to see if every P wave is followed by a QRS complex. If this is the case it can be assumed the normal conduction pathway from the atrium to the ventricles is operational, and atrial contraction is stimulated by atrial depolarisation. If not every P wave is followed by a QRS complex, investigation is required into the P-R interval to identify the presence of heart block and allow classification of its type.

Nurses’ priority is to treat the patient not the ECG. Initial management should involve ABCDE (airway, breathing, circulation, disability, exposure) assessment (Watson, 2006) with supplemental oxygen and IV access as required, with early referral for help or expert advice. Part 2 of this unit, to be published in next week’s issue, explores the signs of bradycardia and its management.

KEY REFERENCES


The full reference list for this unit is available in Portfolio Pages on nursingtimes.net