accurately measure and assess patients’ pulse/heart rate. This is a main component of the Resuscitation Council UK’s (2006) systematic airway, breathing, circulation, disability, exposure (ABCDE) approach to assessing patients who are critically ill.

Sinus bradycardia has the same electrocardiogram characteristics as sinus rhythm (normal ECG rhythm), except that the rate is less than 60 beats/min (Jevon, 2009). Figs 1, 2 and 3 show sinus rhythm, sinus bradycardia and AV block.

CAUSES OF BRADYCARDIA

The vagus (parasympathetic) nerve has an important role in controlling heart rate. Continuous vagal activity (vagal tone) acts as an important role in controlling heart rate.

Continuous vagal activity (vagal tone) acts as an important role in controlling heart rate. The vagus (parasympathetic) nerve has an important role in controlling heart rate. Continuous vagal activity (vagal tone) acts as a brake on the heart: when vagal activity increases, the heart rate slows, and when vagal activity diminishes, the heart rate increases (Jevon, 2009).

During sleep, vagal activity increases, sometimes resulting in a normal bradycardia. Stimulation of the vagus nerve, for example, during tracheal suction, can also result in bradycardia.

The sympathetic nerve is involved in the “fight or flight” response and has the effect of increasing heart rate. Beta-adrenoceptor antagonists (beta-blockers) such as atenolol and bisoprolol, shield the heart from excessive sympathetic activity, which can result in bradycardia (often a desired effect). When patients present with a slow heart rate, it is prudent to check their medication history to see whether or not these medicines have been prescribed.

The heart rate is ultimately controlled by the cardiac centre in the medulla oblongata. Damage to this as a result of hypoxia or cerebral insult, such as a stroke, can lead to bradycardia.

There are many bradycardia related cardiac arrhythmias including sinus bradycardia and AV block. AV block is when the conduction of the cardiac impulse through the AV junction is delayed or blocked. There are varying degrees of AV block: first, second and third (complete).

Other causes of bradycardia include:
- Myocardial infarction;
- Hypothermia;
- Hypoxia;
- Hypothyroidism;
- Hypovolaemia;
- Raised intracranial pressure (Jevon, 2009; Gwinnutt, 2006; Wyatt et al, 2006).

MANAGING PATIENTS WITH BRADYCARDIA

- Assess patients following the ABCDE approach to ascertain whether they are critically ill. Ensure appropriate senior help is called if necessary, following early warning score (EWS) escalation protocols;
- Ensure patients have a clear airway and are breathing adequately;
- If patients are critically ill, start prescribed emergency oxygen (see part 2 of this series, Jevon, 2010);
- Monitor vital signs and complete the EWS chart following local protocols. It is important to adjust the frequency of EWS observations as appropriate;
- Try to identify the cause of the bradycardia. In particular, check medication history. If patients have been prescribed a beta-adrenoceptor antagonist seek medical advice. It may be necessary to withhold the drug until they are medically reviewed;
- If patients are hypotensive or feel light-headed, it is important to lie them flat;
- Start ECG monitoring if appropriate and, if possible, record a single lead ECG strip from the monitor; this will help the clinician to accurately interpret the ECG rhythm;
- Prepare for intravenous cannulation to administer drugs such as atropine, if indicated;
- Record a 12 lead ECG; this will help to establish the correct interpretation of the ECG rhythm (Nolan et al, 2005);
- If necessary, assist medical staff with interventions such as administering atropine, pacing to raise the heart rate and monitoring the effect on pulse rate (Jevon, 2009).