ABSTRACT

Objective: To review the diagnosis and management of cardiac arrhythmias in a two-part presentation.

Data sources: Articles and published peer-review abstracts on tachycardias and bradycardias.

Summary of review: Bradycardias are caused by a failure of the sinus node to generate normal impulses or due to a defect in cardiac conduction that in turn causes a delay or failure of impulse propagation. During sleep, the heart rate may decrease to 30 beats per minute (bpm) with episodes of sinoatrial block, junctional rhythms and first and second-degree atrioventricular block that occur often enough (particularly in trained athletes) to be considered normal variants. However, treatment is required if symptoms of dizziness, confusion, fatigue, Stokes-Adams attacks or heart failure occur.

Sinus node dysfunction or ‘sick sinus syndrome’ is usually caused by intrinsic nodal disease and may present with episodes of tachycardia and bradycardia (tachycardia-bradycardia syndrome). Treatment usually requires a permanent pacemaker. Atrioventricular (AV) conduction disturbances are characterised by a delay or failure of the atrial impulse to be conducted through the AV conducting system. If the escape rhythm is unstable the patient also requires a pacemaker.

In the critically ill patient tachycardias are more often encountered than bradycardias. However, the intensivist should be familiar and skilled in the management of complete heart block and asystole, correcting the underlying defect (drug toxicity, hyperkalaemia, etc), while using catecholamines, atropine, aminophylline or a temporary pacemaker for initial resuscitation.

Conclusions: Bradycardias are uncommon in the critically ill patient and often are caused by an underlying disorder (e.g. hyperkalaemia, calcium channel blocker toxicity, beta-adrenergic receptor blocker toxicity, etc). However, post cardiac bypass and acute myocardial infarction may cause cardiac conduction defects that may require urgent resuscitation with a temporary pacemaker. (Critical Care and Resuscitation 2002; 4: 54-60)

Key words: Critical illness, sinoatrial block, sinus bradycardia, sinus arrest, sick sinus syndrome syndrome, atrioventricular block, complete heart block, idioventricular rhythm, asystole
infarction and cardiac arrest, acting as a competitive inhibitor of $P_1$ receptors (i.e. adenosine $A_1$ and $A_2$ receptors) to inhibit the adenine (released from ischaemic cardiac cells) mediated reduction in cardiac conduction.

**Sinus bradycardia.** Sinus bradycardia is defined as a sinus rhythm of less than 60 beats per minute (bpm). It is associated with the physiological and pathological conditions listed in Table 1. It rarely requires treat-ment. Patients with acute myocardial infarction and sinus bradycardia have a lower mortality compared with all patients with myocardial infarction, which may be due to the protective effect of sinus bradycardia on the compromised myocardium.

If the bradycardia is chronic and severe (e.g. less than 40 beats per minute) and associated with hypotension and reduced cerebral and cardiac perfusion or unstable escape rhythms (e.g. multifocal ectopics, ventricular tachycardia), a pacemaker is usually required (although long term oral propantheline bromide 7.5 - 15.0 mg, 6- to 8-hourly, has also been used).

**Sinoatrial block.** In sinoatrial (SA) block the sinus impulse is blocked within the SA junction (i.e. the junction between the SA node and the surrounding atrial myocardium). First-degree SA exit block describes a prolonged conduction time from the SA node to the surrounding atrial tissue. It requires special techniques for its diagnosis as it cannot be diagnosed from the standard surface ECG.

Second-degree SA exit block is diagnosed in a patient in sinus rhythm when there is an abrupt absence of a P wave and QRS complex with the next P wave occurring at double the normal PP interval. This disorder may be caused by excessive vagal stimulation, digoxin, quinidine, disease of the SA node (e.g. in association with pericarditis) and ‘sick sinus syndrome’.

Third-degree SA exit block is characterised by a lack of atrial activity and cannot be distinguished from sinus arrest on the surface ECG. An escape rhythm (usually nodal rhythm) determines the heart rate.

**Sinus arrest.** In sinus arrest no impulse is generated from the sinus node (Figure 1) and an escape rhythm determines the rhythm of the heart. This disorder may be caused by a severe increase in vagal tone.

**Sinus node dysfunction**

Sinus node dysfunction or ‘sick sinus syndrome’ can be caused by intrinsic disease (fibrous tissue replacement of the SA node) or extrinsic causes (e.g. calcium channel blockers, digoxin, beta-adrenergic receptor blockers, hypothyroidism, excessive vagal tone, jaundice). Permanent injury from infarction (unlike the atrioventricular node) is an uncommon cause for SA node dysfunction. The tachycardia-bradycardia syndrome is a common manifestation of SA node dysfunction where episodes of paroxysmal atrial fibrillation, flutter or tachycardia are followed by severe sinus bradycardia (Figure 2), sinoatrial block or sinus arrest, resulting in

---

**Table 1. Causes of bradycardia**

<table>
<thead>
<tr>
<th>Physiological</th>
<th>Pathological</th>
</tr>
</thead>
<tbody>
<tr>
<td>Convalescence from infections</td>
<td>Excess vagal stimulation</td>
</tr>
<tr>
<td>Athletes</td>
<td>Intraoperative stimulation (e.g. cervical dilation, eye, testicular and carotid surgery)</td>
</tr>
<tr>
<td>Sleep</td>
<td>Orbital pressure</td>
</tr>
<tr>
<td></td>
<td>High intracranial pressure</td>
</tr>
<tr>
<td></td>
<td>Hypothermia</td>
</tr>
<tr>
<td></td>
<td>Obstructive jaundice</td>
</tr>
<tr>
<td></td>
<td>Myxoedema</td>
</tr>
<tr>
<td></td>
<td>Sleep apnoea syndrome</td>
</tr>
<tr>
<td></td>
<td>Infections</td>
</tr>
<tr>
<td></td>
<td>Typhoid fever, brucellosis</td>
</tr>
<tr>
<td></td>
<td>Chagas’ disease (trypanosomiasis)</td>
</tr>
<tr>
<td></td>
<td>Infiltrative or collagen diseases</td>
</tr>
<tr>
<td></td>
<td>Systemic lupus erythematosis, scleroderma</td>
</tr>
<tr>
<td></td>
<td>Rheumatoid arthritis</td>
</tr>
<tr>
<td></td>
<td>Myotonic dystrophy</td>
</tr>
<tr>
<td></td>
<td>Carotid sinus hypersensitivity</td>
</tr>
<tr>
<td></td>
<td>Drugs</td>
</tr>
<tr>
<td></td>
<td>Beta-blockers</td>
</tr>
<tr>
<td></td>
<td>Digoxin</td>
</tr>
<tr>
<td></td>
<td>Calcium-blockers (e.g. verapamil, nimodipine)</td>
</tr>
<tr>
<td></td>
<td>Tamoxifen</td>
</tr>
<tr>
<td></td>
<td>Antiarrhythmic agents</td>
</tr>
<tr>
<td></td>
<td>Hyperkalaemia, hypermagnesaemia</td>
</tr>
</tbody>
</table>

**Sinoatrial node disorders**

- Sick sinus syndrome
- Pericarditis
- SA node trauma

**Atrioventricular node disorders**

- Lev’s disease
- Lenegre’s disease
- Myocardial infarction or ischaemia (usually inferior)
- Acute rheumatic fever, infectious mononucleosis, Lyme disease
- Endocarditis, myocarditis
- AV node trauma
symptoms of low cardiac output (e.g. dizziness, confusion, fatigue, Stokes-Adams attacks, heart failure). The diagnosis of the ‘sick sinus syndrome’ is made if there is less than a 25% increase in heart rate with intra-venous atropine (the normal response is an increase in heart rate by 50 - 60%).

Treatment for this disorder, if symptomatic, usually requires a pacemaker (an atrial pacemaker is preferred as it reduces the incidence of atrial fibrillation, thromboembolism and pacemaker syndrome\textsuperscript{10}), although initial management with theophylline 200 - 400 mg/day may be of benefit.\textsuperscript{11}

### Atrioventricular conduction disturbances

#### Atrioventricular block

This is characterised by a delay or failure in conduction of the atrial impulse through the AV conducting system. The conduction may be inhibited in either the AV node or bundle of His; inhibition of the impulse below the bifurcation of the bundle of His causes bundle branch or fascicular blocks with AV conduction maintained unless all three fascicles are affected simultaneously. There are three grades of atrioventricular block:
1. **First-degree block.** This is defined as a PR interval greater than 0.20 s (Figure 3). The PR interval represents the time taken for the impulse to travel from the SA node to the AV node (usually 0.04 s) plus the time taken for the impulse to travel through the AV node, the bundle of His, the bundle branches and the Purkinje system. Thus prolongation of the PR interval may represent a delay in any stage along the conducting pathway. However, if the duration of the QRS complex is normal, a prolonged PR interval is almost always caused by a delay within the AV node. If the QRS duration is prolonged the delay may be present in any of the levels previously mentioned. A delay within the His-Purkinje system is always accompanied by a prolonged QRS although it can occur with a normal PR interval.

2. **Second-degree block.** This is characterised by an intermittent failure of AV conduction producing intermittent absence of a QRS complex initiated from the atrial impulse. The number of impulses which are not conducted may be expressed as a conduction ratio; for example, 2:1 block if every second atrial impulse is not conducted (Figure 4) or 3:2 block if every third impulse is not conducted. The His bundle electrocardiogram (where the His bundle ‘H’ deflection can be identified) has enabled second-degree blocks to be
divided into prolongation of the AH interval (e.g. AV nodal conduction is slowed) and prolongation of the HV interval (i.e. infranodal block). There are two standard ECG types of second-degree block:

a) Mobitz I (Wenckebach) block. This is characterised by a progressive prolongation of the PR interval and decrease in the RR interval (due to progressive decrease in PR increments) until there is non-conduction of the P wave (Figure 5). The sinus rate (i.e. PP interval) remains constant. The block results in a rest period, which facilitates the recovery of the conducting tissue, allowing the sequence to begin again. The block is almost always associated with a prolongation of the AH interval and a normal QRS duration (in the absence of a bundle branch block), and has been reported in 6% of asymptomatic medical students without apparent heart disease. It is often found as a transient abnormality in patients with acute inferior myocardial infarction or with drug toxicity (e.g. digoxin, beta-adrenergic receptor blockers, calcium channel antagonists)

b) Mobitz II block. This is characterised by a constant PR interval with a periodic nonconducted P wave (Figure 4). It is usually associated with QRS widening and a likelihood of progressing to complete heart block. The block is characteristically associated with prolongation of the HV interval.

3. Third-degree block. This is characterised by a complete block of the atrial conduction through the AV conduction system (Figure 6). If the block is in the AV node the escape rhythm is usually in the His bundle, with a stable rate of 40 - 60 bpm, a QRS complex of normal duration and is often haemodynamically stable. The rhythm is usually under vagal influence (i.e. alters with exercise and atropine). However, if the conduction defect is either an intra- or infra- His block, the ventricles are activated by a His-Purkinje system escape pacemaker which has a lower intrinsic rate (i.e. 25 - 45 bpm, a wide QRS complex (idioventricular rhythm) and is usually haemodynamically unstable (and therefore requires a pacemaker) as it may lead to asystole.

The clinical features of complete heart block include cannon ‘a’ waves, varying intensity of first heart sound, and syncopal (i.e. Stokes-Adams) attacks. Third-degree block may be caused by sclerodegenerative disease of the cardiac conducting tissue (i.e. Lenegre’s disease), fibro calcareous process of the cardiac skeleton (associated with aortic stenosis, and known as Lev’s disease), intracardiac surgery, ischaemic heart disease, tumours, digoxin toxicity, hyperkalaemia and congenital heart disease. The ECG reveals AV dissociation and a QRS complex which is often wide and bizarre.

**Bundle branch blocks.** While left bundle branch block (LBBB) is often observed in patients with cardiac disease, both LBBB and right bundle branch block are asymptomatic and almost always present as an unexpected ECG finding.

**AV dissociation.** This describes the condition where the atria and ventricles beat independently (i.e. the P wave bears no relationship to the QRS) and characteristically occurs with complete heart block. However, it can also occur in severe sinus bradycardia when the escape and sinus rhythm are similar, where the P waves occur just before, during or just after the QRS complex (isorhythmic AV dissociation), or when a junctional or ventricular pacemaker is enhanced and competes with the normal sinus rhythm in ventricular tachycardia, accelerated junctional or ventricular rhythms (interference AV dissociation).

**Parasystole.** Parasystole is defined as the simultaneous activity of two (rarely more) independent impulse forming centres, one of which is protected from the other by an entrance block. An extrasystole has a consistent coupling interval with the preceding beat, the parasystolic beat having coupling intervals, a constant shortest inter-ectopic interval (although it may exhibit Wenckebach exit block and therefore vary by 0.04 - 0.12 s) and the frequent appearance of fusion beats. Parasystole is usually associated with heart disease (e.g. cardiomyopathy, ischaemic heart disease or hypertensive heart disease), although the arrhythmia is benign and usually does not require treatment. Ventricular parasystole usually has a rate of 30 - 40 bpm (i.e. the inherent rate of the ventricle), whereas atrial parasystole usually has a rate of 45 - 55 bpm. Atrial parasystole occurs in cardiac transplant patients as both the donor and the recipient SA nodes (the former has the faster rate of the two as it is not under parasympathetic control) generate independent P waves.

The treatment of a bradycardia depends upon the severity of symptoms, the correlation between symptoms when bradycardia occurs and the presence of reversible causes.

For symptomatic patients management with a permanent pacemaker depends upon the correlation between the bradycardia and symptoms and the presence (or otherwise) of reversible causative factors. Patients who have potentially reversible factors may be treated with a temporary pacemaker during management of the underlying disorders.

If a patient is asymptomatic then the generally acceptable indications for a permanent pacemaker are:

a) a third-degree AV block with a documented asystolic period lasting 3 s or more, or an escape rhythm less than 40 bpm while the patient is awake,
b) a third-degree AV block or second-degree AV Mobitz II block in patients with chronic bifascicular and trifascicular block, and
c) a congenital third-degree AV block with a wide QRS escape rhythm, ventricular dysfunction or bradycardia inappropriate for age.

CARDIAC PACING

Cardiac pacing involves the periodic delivery of low electrical energies by a pulse generator, through bipolar or unipolar pacing electrodes, to the heart to initiate and maintain cardiac rhythm. Bipolar pacing electrodes allow the electric current to travel down a wire to the distal electrode, depolarise the myocardium and return to the pacemaker via the proximal electrode. These electrodes are commonly used for temporary pacing.

Unipolar pacing electrodes allow the electric current to travel down a wire to the electrode, depolarise the myocardium, and to return to the pacemaker via body fluids. These electrodes are often used for permanent pacing, although the skin electrode needed for the returned current may cause muscle twitching, and sensing of skeletal muscle potentials may lead to pacing artifact.

Both the ventricular and atrial myocardium may be sensed and paced by a pacing electrode, delivering the impulse directly to the:

a) endocardium via a pacing lead which is inserted transvenously under fluoroscopic control, using a rigid or balloon-tipped pacing catheter,

b) epicardium via a pacing lead which is fixed at the time of cardiac surgery,

c) chest wall (i.e. transthoracic) via pads which are applied to the chest wall providing temporary cardiac pacing during an emergency, and
d) oesophagus (i.e. transeosophageal) via an oesophageal pacing lead providing temporary pacing.

Transvenous pacing

A semi-rigid or balloon-tipped pacing catheter may be inserted percutaneously into the internal or external jugular or subclavian vein and positioned into the right ventricle under fluoroscopic control. When the electrode is correctly positioned, the stimulation threshold or capture (i.e. minimum current required to consistently pace the heart) is measured. This should ideally be less than 1 mA, and certainly less than 2 mA with a pulse duration of 0.5 ms. The pacemaker output is then normally set at about 5 mA. The stability of the pacing electrode is tested by asking the patient to breathe deeply and cough.

If a temporary pacemaker is inserted, the generator remains beside the patient. If a permanent pacemaker is inserted, an implantable pulse generator is placed subcutaneously in the abdominal wall, chest wall or axilla. Implantable pulse generators are sealed devices weighing from 30 - 130 g. They are powered by a lithium battery and, depending on the model, may last to last 2 - 15 years and be transcutaneously (i.e. noninvasively) programmable.15

REFERENCES