

Cardiac Arrhythmias – Part I

Bradycardia

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Abstract

Symptomatic bradycardia is a common reason for presentation to Emergency Departments in the UK. Nevertheless, the acute management of bradycardia remains a cause for unnecessary anxiety and confusion among admitting physicians. This article reviews the aetiology, electrocardiographic appearances and management of the common defects of cardiac conduction which may manifest clinically as bradycardia. Particular attention is paid to bradycardia in the context of acute myocardial infarction and the role of temporary cardiac pacing.

Keywords

Bradycardia, conduction disturbance, temporary pacing, myocardial infarction

Key Points

1. The atrioventricular node is normally the only electrical communication between atria and ventricles.
2. Bradyarrhythmias are usually caused by disturbed electrical impulse formation in the sinoatrial node and/or disturbed conduction of the formed impulse to the ventricles.
3. A clinically important bradycardia may be defined as an inappropriate slowing of heart rate resulting in presyncope, syncope, haemodynamic compromise or cardiac arrest.
4. Asymptomatic bradycardias do not usually require emergency intervention.
5. Treatment of the stable patient with bradycardia is based on elimination of reversible causes of the bradycardia and assessment of the likelihood of clinically significant disease of the cardiac conduction system requiring pacemaker implantation.
6. In the context of acute myocardial infarction, temporary cardiac pacing is usually indicated in the presence of infraHisian AV block.
7. In the context of an acute ST segment elevation myocardial infarction, thrombolysis should not be delayed to facilitate transvenous insertion of a temporary pacing lead.

Introduction

Disturbances of cardiac rhythm may be broadly divided into bradyarrhythmias and tachyarrhythmias, where the normal adult heart rate is arbitrarily defined as between 60 and 100 beats per minute. This article is the first in a series of three to address the presentation, diagnosis and immediate management of the patient presenting to an

Emergency Department with an abnormality of cardiac rhythm, and will focus specifically on bradycardia. The second and third articles will address narrow complex tachycardias and broad complex tachycardias respectively.

The Cardiac Conduction System

An understanding of the anatomy and physiology of cardiac conduction is essential to the diagnosis and management of all arrhythmias. In the structurally normal heart, the sinoatrial node (SAN) is the origin of the cardiac impulse.¹ Normally located in the right atrium, near the junction with the superior vena cava, the myocytes of the SAN are capable of spontaneous diastolic depolarisation, normally at a rate greater than myocytes elsewhere in the heart. Once the cardiac impulse exits the SAN, it traverses and depolarises the right atrium, spreading to the left atrium via Bachmann's bundle in the upper interatrial septum and via a connection near the coronary sinus in the lower interatrial septum. Depolarisation of the atrial cell membranes is followed by mechanical contraction of the atria.

The atrioventricular node (AVN) is a complex structure, located in the lower interatrial septum, anterior to the mouth of the coronary sinus and near the attachment of the septal leaflet of the tricuspid valve to the interatrial septum.² In the structurally normal heart, the AVN is the only electrical communication between the atria and ventricles. In terms of understanding the electrophysiological function of the AVN, it serves as an important delay to impulse propagation from the atria to the rapidly conducting His-Purkinje network.³ This delay allows atrial contraction to maximise end-diastolic ventricular filling, by which time the electrical impulse has escaped the AVN to propagate down the His bundle, the right and left bundle branches and depolarise the left and right ventricular myocardium. This is followed by ventricular systole.

Disturbance of Sinoatrial Node Function

The SAN is subject to physiological and pathological influences. Physical inactivity, training and sleep may be accompanied by an asymptomatic,

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physiological bradycardia, with heart rates as low as 30 beats per minute being recorded in healthy, elite athletes.⁴

Pathological SAN dysfunction may be due to intrinsic or extrinsic causes (Table I). The most common clinically encountered causes of bradycardia secondary to SAN dysfunction include idiopathic fibrotic degeneration and ischaemia. Because the SAN is richly innervated by the autonomic nervous system,⁵ situations associated with an increase in vagal tone may result in a symptomatic bradycardia, for example, carotid sinus hypersensitivity. Systemic disorders including hypothermia and hypothyroidism are other well recognised, extrinsic causes of pathological bradycardia.

There are four principle electrocardiographic manifestations of SAN dysfunction: sinus bradycardia, sinus arrest, sinus node exit block and tachycardia-bradycardia syndrome. Sinus bradycardia is arbitrarily defined as a heart rate of less than 60 beats per minute. The clinical context of sinus bradycardia is important: if accompanied by chronotropic incompetence i.e. failure to elevate the heart rate to achieve 85% of the age-predicted maximum heart rate at peak exercise, it is likely to indicate disease of the SAN.⁶ Sinus arrest implies failure of impulse production within the SAN whereas sinus node exit block implies failure of impulse conduction to the atrial myocardium from within the node after impulse production. Tachycardia-bradycardia syndrome describes the association of sinus bradyarrhythmia with atrial tachyarrhythmia in a symptomatic patient. Importantly, profound sinus bradycardia may be associated with the emergence of an atrial or AVN escape rhythm which would normally be suppressed by the more rapidly depolarising SAN.

Disturbance of Atrioventricular Conduction

The normal propagation of a cardiac impulse which has successfully exited the SAN may be interrupted in the atrium, AVN, Bundle of His or left and right bundle branches. Dysfunction of any of these components may lead to a disturbance of atrioventricular conduction with resultant bradycardia (figure 1).

Figure 1. Schematic representation of anatomical level of conduction disturbance. Abbreviations: atrioventricular AV, atrioventricular block AVB, left bundle branch block LBBB, right bundle branch block RBBB. Third degree AV block can occur within the AV node (supraHisian), within the Bundle of His (intraHisian) or below the bundle of His (infraHisian) i.e. simultaneous left and right bundle branch block

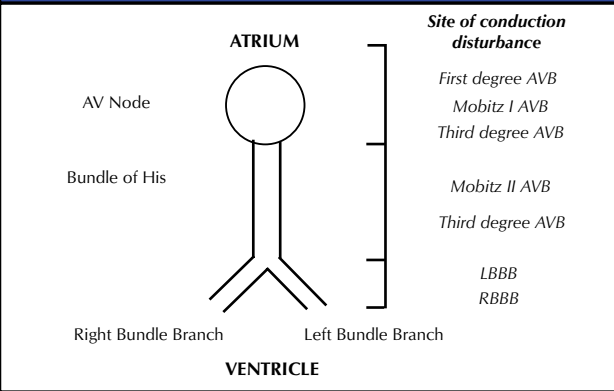


Table I. Causes of and appropriate investigations for bradycardia	
Intrinsic	
Cause	Investigation
Idiopathic degeneration (Lev's disease and Lenegre's disease)	Diagnosis of exclusion
Aortic stenosis	Transthoracic echocardiogram (TTE)
Myocardial ischaemia/infarction	Troponin I/T, CK, TTE
Infiltration: Sarcoidosis Amyloidosis	CXR, serum ACE, TTE immunoglobulins, urine dipstick, urinary Bence-Jones protein, target organ biopsy, TTE
Haemochromatosis	LFT, serum ferritin, iron TIBC, blood glucose, TTE, cardiac MR
Malignancy	CXR, TTE, transoesophageal echocardiogram (TOE)
Fabry's disease	Alpha-galactosidase A activity, TTE, cardiac MR
Cardiomyopathy	TTE Other investigations guided by likely aetiology
Infective endocarditis	Blood cultures, inflammatory markers, urine dipstick, TTE, TOE
Rheumatoid disease, systemic lupus erythematosus	FBC, ESR, CRP, Rheumatoid factor, ANA, C3, C4, dsDNA
Congenital heart disease	CXR, TTE, maternal lupus serology
Extrinsic	
Medication ((beta adrenergic blockade, calcium channel antagonists, digoxin, antiarrhythmic drugs, lithium, carbamazepine, clonidine, paclitaxel, H2 antagonists)	Drug levels where appropriate
Raised intracranial pressure	Fundoscopy, CT Brain
Hypothermia	Core temperature
Hypothyroidism	Thyroid function test
Myocarditis eg lyme carditis, Chagas disease	TTE, isolation of infectious agent – viral, bacterial, fungal, rickettsial, spirochaetal, helminthic, protozoal
Obstructive jaundice	LFT, liver ultrasound, CT abdomen, ERCP
Iatrogenic (post cardiac surgery)	
Autonomic syndromes (carotid sinus hypersensitivity, coughing, micturition)	ECG during carotid sinus massage, tilt table testing
Electrolyte disturbance (hyperkalaemia, hypokalaemia)	Urea and electrolytes
Obstructive sleep apnoea	

First degree atrioventricular block

The PR interval on the 12 lead ECG represents the time taken from the onset of atrial depolarisation to the onset of ventricular depolarisation. Consequently, prolongation of the PR interval beyond 200ms i.e. first degree AV block, may arise as a result of delayed conduction anywhere between SAN and ventricle, but usually occurs at the level of the AVN. Every P wave is followed by a QRS complex in first degree AV block. Importantly, in the absence of another conduction disturbance, first degree AV block does not by itself result

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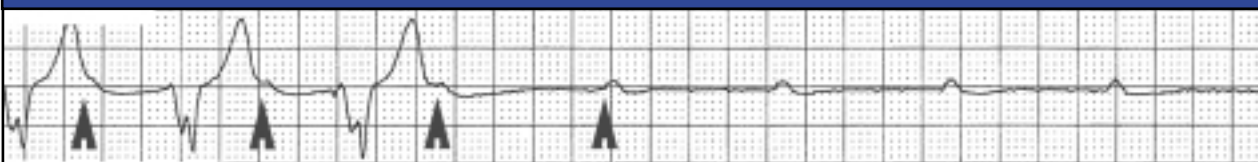
Figure II. Mobitz Type II AV Block with 2:1 atrioventricular conduction. There are two P waves for every QRS complex. The PR interval of the conducted beats is fixed. Paper speed is 25mm/s



Figure III. SupraHisian complete AV Block. There is no relationship between the P waves and QRS complexes. The atrial rate is faster than the ventricular rate. The QRS complex is narrow. This patient has congenital complete AV block secondary to maternal systemic lupus erythematosus. Paper speed is 25mm/s.



Figure IV. InfraHisian complete AV block. The arrowheads show P waves inscribed in the T waves of the first three QRS complexes, which are broad. This unstable, slow, broad complex escape rhythm fails, revealing normal sinus node function with complete loss of AV conduction. Paper speed is 25mm/s



in bradycardia, and the patient is usually asymptomatic unless there is marked atrioventricular asynchrony as a result of the conduction delay.

Second degree atrioventricular block

Intermittent atrioventricular conduction is synonymous with second degree AV block, and is usually accompanied by intermittent bradycardia. The nomenclature of second degree AV block is often a source of confusion, for which reason an understanding of the electrophysiological disturbance is more important than remembering a classification based solely on the surface ECG and which can only be applied when at least two consecutive beats are conducted.

Mobitz type I or Wenkebach AV block describes progressive prolongation of the PR interval with subsequent failure of ventricular capture following a P wave. When the QRS complex is of normal duration, the level of block is almost always proximal to the His bundle, within the AV node.

Mobitz type II AV block (figure II) describes atrioventricular conduction block in which an atrial impulse fails to conduct to the ventricle with no preceding change in the PR interval. It is almost always due to conduction disturbance in the infranodal tissues i.e. the His-Purkinje system.

Third degree AV block

Complete failure of conduction of impulses from atrium to ventricle defines third degree AV block. The patient is dependent on an escape rhythm, the origin of which defines the rate and reliability. With third degree AV block located in the AVN, the escape rhythm usually originates from within the His-Purkinje system, resulting in a relatively narrow complex QRS morphology, at a rate

of 40–55 bpm (figure III). When the disturbance is infra-nodal, the escape rhythm is ventricular in origin, broad complex and unreliable (figure IV). Because of the severity of the conduction disturbance, infranodal third degree AV block is frequently accompanied by symptomatic bradycardia.

Acute Management of Clinically Important Bradycardia

A clinically important bradycardia may be defined as an inappropriate slowing of heart rate resulting in presyncope, syncope, haemodynamic compromise or cardiac arrest. Asymptomatic bradycardias, other than complete heart block, do not usually require emergency intervention.

Immediate assessment – emergency

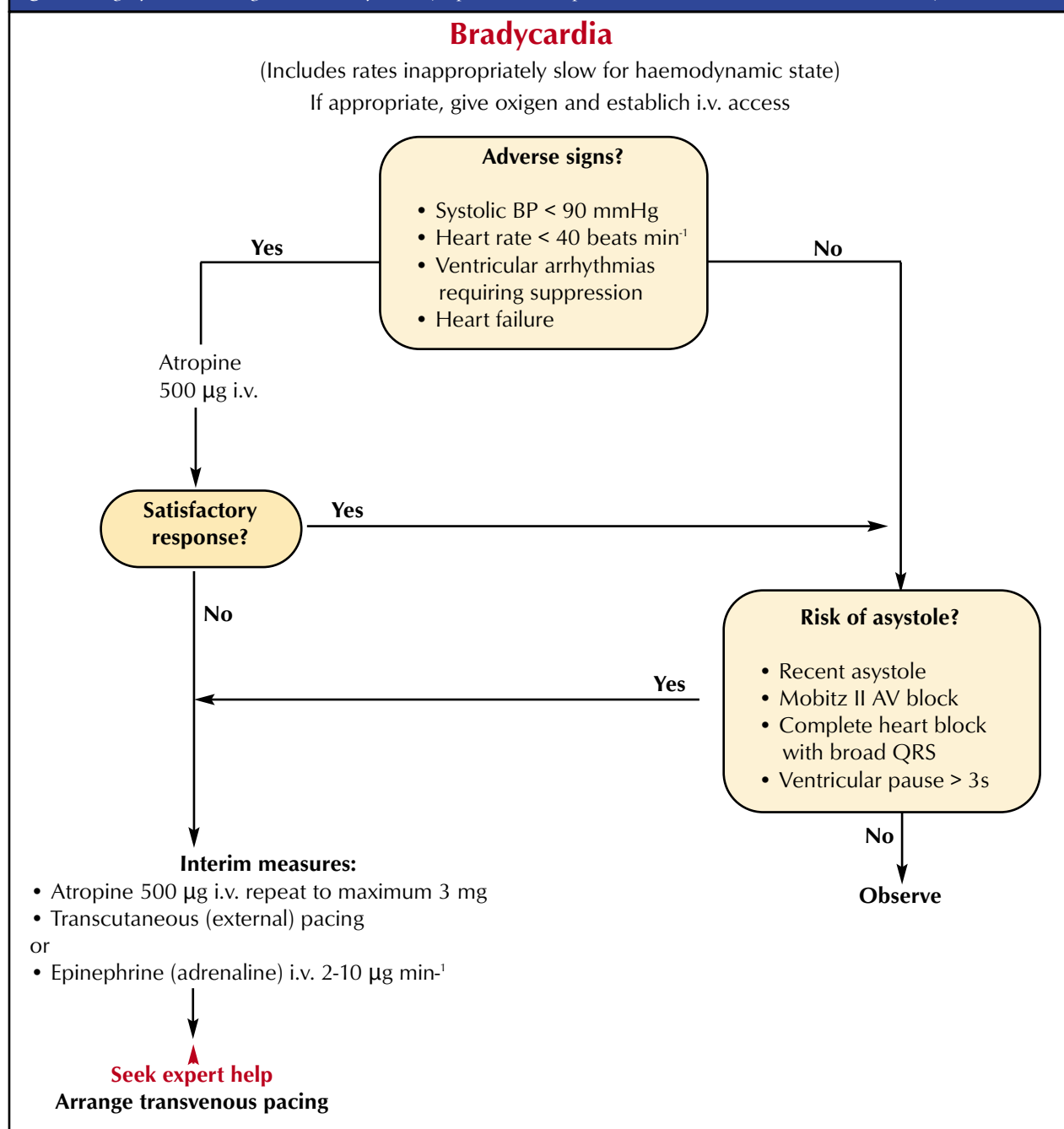
As with any medical emergency, the immediate management demands assessment of the airway, breathing, circulation and establishment of intravenous access. An ECG monitor should be attached to permit real time observation of the cardiac rhythm. For a patient with asystole or pulseless bradycardia, the UK Resuscitation Council guidelines should be followed (figure V).

History

Evaluation of the stable patient with confirmed or suspected bradycardia should begin with a thorough, targeted history, with a view to establishing the potential contribution of the aetiologies detailed in table I. Of particular importance is to recognise a bradyarrhythmia as a harbinger of myocardial ischaemia, although it is unusual to see a bradyarrhythmia as the only electrocardiographic manifestation of acute myocardial ischaemia.

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Figure V. Algorithm for management of bradycardia. (Reproduced with permission from the Resuscitation Council UK)



Examination

The type and aetiology of most bradycardias may be gleaned by the astute physician from a thorough physical examination.⁷

Just as the P wave and QRS complex of the electrocardiogram identify the relationship of atrial and ventricular function, so too do the A and V waves of the jugular venous pulse. For example, two-to-one atrioventricular block is recognized by two A waves for every one carotid pulsation.

The physical signs of complete atrioventricular block in the jugular venous pulse were first described by William Stokes in 1846.⁷ He noted intermittent cannon A waves

(atrial contraction against a closed tricuspid valve, coincident with ventricular escape beat) with smaller independent A waves at a rate faster than the ventricular bradycardia identified by the slow carotid pulse. Wenckebach also described his phenomenon solely by analysis of the jugular venous pulse.

The auscultatory correlate of complete atrioventricular block is variability in intensity of the first heart sound – loud when the PR interval is short (i.e. the mitral and tricuspid leaflets are maximally open) and soft when the PR interval is long (i.e. the mitral and tricuspid leaflets are in or near their closed position). Persistent but not fixed splitting of the second heart sound may be heard with

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right bundle branch block because of delayed pulmonary valve closure. Reversed splitting of the second heart sound, with pulmonary component preceding aortic, may be heard in left bundle branch block or right ventricular pacing (delayed aortic valve closure).

Investigation

The single most important investigation in the immediate evaluation of the bradycardic patient is the 12 lead surface electrocardiogram. In order to obtain the maximum diagnostic yield from the ECG, the following is a suggested approach to interpretation:

- What is the ventricular rate during bradycardia i.e. rate of QRS complexes, and is it regular or irregular?
- Is there evidence of myocardial ischaemia?
- Are there visible P waves? If so, what are their rate and relationship to the QRS complexes eg. 1:1, 2:1, 3:1, 4:1 etc or a variable relationship? Do they precede or follow the QRS complex i.e. antegrade (atrioventricular) conduction or retrograde (ventriculoatrial) conduction?
- What is the QRS morphology? Is it narrow (<120ms) or broad (>120ms)? Is there bundle branch block?
- If there are no visible P waves, and the ventricular rate is irregular, the diagnosis is likely to be atrial fibrillation with a slow ventricular response rate and the AVN intact.
- If there are no visible P waves, the ventricular rate is slow and regular, and the QRS duration is <120ms, this is likely to be atrial fibrillation with a nodal/His escape rhythm because of complete AV block. If the QRS duration is >120ms, this is likely to represent a ventricular escape rhythm.

Other electrocardiographic clues to the aetiology of the bradycardia may include the acute ST segment or T wave changes of myocardial ischaemia. An irregular baseline may be seen in the shivering, hypothermic patient. Low voltage QRS complexes and bradycardia may be seen in hypothyroidism. A commonly used definition of low voltage requires the QRS amplitude to be less than 0.5mV in each of the limb leads and less than 1.0mV in each of the precordial leads.⁸

Other investigations which may be relevant in the assessment of a patient with bradycardia are summarised in Table I.

Treatment

Treatment of bradycardia in the stable patient begins with elimination of reversible causes. Should bradycardia persist, the likelihood of clinically significant disease of the cardiac conduction system which is likely to require implantation of a permanent pacemaker is assessed, for example Mobitz II AV block or complete AV block. Although certain indications for pacing are unambiguous, for example, complete AV block, others require considerable expertise and judgement, in addition to familiarity with the published guidelines and the potential complications likely to arise in a particular patient. The

decision to implant a permanent pacemaker is therefore best made by a cardiologist in conjunction with the physician responsible for the patient's care. A summary of the ACC/AHA/NASPE 2002 guidelines on permanent pacing is available.⁹

Bradyarrhythmia and Myocardial Infarction

Ischaemic injury to the myocardium can result in a disturbance of function at any level of the cardiac conduction system. In the majority of individuals, the sinoatrial node and the atrioventricular node receive their blood supply from the right coronary artery, while in a minority, the circumflex artery supplies these structures. The left anterior descending coronary artery supplies the interventricular septum and consequently the His bundle and left and right bundle branches.¹⁰

Acute myocardial infarction is complicated by a disturbance of conduction in approximately 16% of cases in the thrombolytic era.¹¹ The most common disturbance is narrow complex AV nodal block, followed by RBBB, bifascicular block, LBBB and broad complex AV block in descending order of frequency.

First degree AV block in the context of myocardial ischaemia is rarely of haemodynamic significance and does not require specific treatment. In the absence of haemodynamic compromise, and in the presence of a normal QRS duration, the prescription of a beta-blocker should not be delayed by the presence of first degree AV block.

Mobitz type I AV block occurs in about 10 percent of patients admitted to CCU with acute myocardial infarction, and is nine times more common than Mobitz type II AV block in that context.¹² Unless the patient is symptomatic and the QRS complex is broad, specific treatment is not usually necessary. Atropine 300 to 600 microgrammes is appropriate for symptomatic bradycardia in this population. Temporary pacing is not indicated unless atropine is ineffective in relieving symptoms or haemodynamic impairment.

The incidence of Mobitz type II AV block in myocardial infarction is less than 1 percent. As discussed earlier however, its presence implies increased likelihood of block distal to the AV node and carries a significant risk of progression to complete AV block. In the context of an acute myocardial infarction, Mobitz type II AV block should be treated by temporary transvenous pacing in VVI mode at a demand rate of 50 to 60 bpm.

In the thrombolytic era, third degree or complete AV block occurs in approximately 4% of acute myocardial infarctions that reach hospital.¹³ When associated with right coronary territory ischaemia, the level of block is usually confined to the AV node, the resultant escape rhythm is often stable and of normal QRS duration and will recover spontaneously within days. In that setting, temporary pacing can usually be avoided. Anterior infarction, secondary to proximal occlusion of the left anterior descending coronary artery and with resultant interruption of the His-Purkinje system within the interventricular system, may result in sudden, complete

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AV block with an unstable, slow, broad complex ventricular escape rhythm. Insertion of a temporary transvenous pacemaker is warranted here but this complication indicates a worsened prognosis.

Temporary Transvenous Cardiac Pacing

Careful consideration should be given before insertion of a temporary transvenous pacing lead. Approximately one third of temporary pacing procedures are complicated by one or more of the following: lead displacement, systemic infection, local infection, inadvertent arterial puncture, pneumothorax or haemopericardium.¹⁴ Approximately one quarter of patients in this series who required permanent pacemaker implantation had this procedure delayed because of a complication arising from the placement of a temporary pacing lead. Unsurprisingly, the immediate complication rate is reduced in the hands of experienced operators. In the thrombolytic and primary percutaneous coronary intervention era, the incidence of rhythm disturbance necessitating insertion of a temporary pacing wire is decreasing,¹³ which itself has important training and service-delivery implications for district general hospitals without on call cardiology services.¹⁵

The unequivocal indications for insertion of a temporary pacing wire in the acute setting are summarised in Table II. In essence, patients who may need to go on to have a permanent pacemaker implanted should only have a temporary wire inserted if the permanent system cannot be implanted quickly enough, if they have had a syncopal

Table II. Indications for temporary cardiac pacing in the acute setting

Indications for temporary pacing in the acute setting
Asystole
Symptomatic bradycardia unresponsive to atropine
Any 2 nd degree or 3 rd degree AV block with haemodynamic compromise or syncope at rest
Mobitz type II AV block in the context of myocardial ischaemia
Ventricular tachycardia secondary to bradycardia

episode, if they are haemodynamically compromised by bradycardia or have ventricular tachycardia as a result of the bradycardia and therefore require overdrive pacing.¹⁶

Summary

Acute management of bradycardia is dependent on the anatomical level of disturbance of function of the cardiac conduction system, the presence or absence of a reversible precipitant and the likelihood of future prognostically significant bradycardia. Temporary cardiac pacing is indicated as a bridge to permanent pacing in the context of syncope secondary to conduction disturbance, haemodynamic compromise or ventricular tachycardia secondary to bradycardia where permanent pacing cannot be offered as the initial treatment.

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