



PRACTICE

CLINICAL UPDATES

Bradyarrhythmias and pacemakers

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What you need to know

- Suspect bradyarrhythmia in patients with symptoms of fainting, shortness of breath, chest pain, or lethargy, and a heart rate <60 beats/min on examination
- Request 12-lead electrocardiography (ECG) to detect conduction abnormalities such as atrioventricular block or sinus node disease, and blood tests to exclude thyroid disorder and electrolyte imbalance
- Referral to a cardiologist is usually needed to evaluate heart function and for management
- Pacemaker implantation is indicated when symptoms can be attributed to bradycardia or atrioventricular block, or in asymptomatic patients with type 2 second-degree heart block or complete heart block
- Complex devices such as biventricular pacemakers and implantable cardioverter defibrillators are being considered in patients with a pacing indication and left ventricular impairment to prevent worsening left ventricular function or sudden cardiac death

Bradyarrhythmias are heart rhythms with a rate of <60 beats/min, commonly due to conduction abnormalities in the heart such as sinus node disease or atrioventricular block.¹ There is increased risk of sudden cardiac death due to slowing or stopping of the heart, and of falls, especially in older people, due to fainting.

Worldwide over one million pacemakers are implanted annually for bradyarrhythmias.^{2,3} With an ageing population this is expected to increase.⁴ New technologies such as leadless pacemakers are being introduced, which general physicians will increasingly encounter. This update will help physicians identify and manage bradyarrhythmias appropriately and will familiarise them with an increasingly complex array of pacemakers.

Sources and selection criteria

We used the 2016 European Society of Cardiology (ESC) heart failure guidelines,²⁹ the 2013 ESC¹³ and 2012 American College of Cardiology Foundation (ACCF)/American Heart Association (AHA)/Heart Rhythm Society (HRS) pacing guidelines¹⁶ and the 2014 National Institute for Health and Care Excellence (NICE) guidelines³⁰ in drawing up the recommendations in this article. We also searched EMBASE and PubMed for additional resources on bradyarrhythmia.

How do patients present?

Patients may present with lethargy, shortness of breath, chest pain, dizziness and fainting as a consequence of reduction in cardiac output, and cardiac and cerebral hypoperfusion. Symptoms such as syncope are episodic and often infrequent. Approximately 20-40% of patients report an episode of syncope with bradycardia, and this may recur in 10-15% of patients.^{5,6} Case reports have also documented patients who remained asymptomatic and whose diagnosis was incidental.

What are the causes?

Bradyarrhythmias are more common in older people due to degeneration and fibrosis of the conducting system.^{7,8} It is however important to identify and treat reversible causes such as hypothyroidism⁹ and drug therapy.^{10,11} Comorbidities such as hypertension and ischaemic heart disease are also recognised risk factors.¹² Cardiac ischaemia, particularly of the right coronary circulation, can cause sinus bradycardia and sustained complete heart block.

Rarely, young people with congenital atrioventricular block¹³ or inflammatory cardiomyopathies such as sarcoid, can present with bradyarrhythmia.¹⁴ **Box 1** lists common and rare causes.

Box 1: Causes of bradyarrhythmias**Common causes**

- Degeneration and fibrosis
- Ischaemic heart disease
- Hypertension
- Thyroid dysfunction
- Atrioventricular blocking drugs (β blockers, calcium channel blockers, digoxin)

Rarer causes

- Inflammatory cardiac diseases (such as sarcoidosis)
- Infectious diseases (such as Lyme disease, acute rheumatic fever, infectious mononucleosis)
- Cardiomyopathies (Leiden dilated cardiomyopathy, amyloidosis)
- Neurological diseases (myotonic dystrophy, limb girdle muscular dystrophy)
- Catheter ablation

How is it diagnosed?

Clinical

Take a detailed history of the presenting symptom and associated cardiac symptoms, such as fainting or syncope, shortness of breath, and chest pain. Ask about the duration and frequency of symptoms and any exacerbating or relieving factors. These symptoms may be caused by bradyarrhythmia but may also suggest coexistent ischaemia or left ventricular impairment. Sudden onset syncope with no prodrome or syncope in the supine position raises the suspicion of bradyarrhythmia.¹⁵ A history of known cardiac disease in the patient or family history of inherited diseases or premature sudden cardiac death (<50 years old) increases the likelihood that the syncopal episode is cardiac in nature.¹⁵ Review the patient's medications as β blockers and calcium channel blockers may cause bradycardia.

On examination, a pulse rate <60 beats/min is generally defined as bradycardic, although a rate of <40-50 beats/min more likely suggests an abnormality, and the rate can be as low as 20 beats/min. Record the blood pressure and respiratory rate. If the patient seems confused or drowsy, you may assess the level of consciousness using the Glasgow coma scale.

Investigations

Blood tests to exclude thyroid disorders and electrolyte imbalance are recommended. In all patients with suspected bradyarrhythmia, request conventional 12-lead electrocardiography (ECG). If the patient is clinically stable this can be performed in primary care. [Table 1](#) lists findings on ECG that correlate with the type of bradyarrhythmia. [Figure 2](#) depicts ECG findings in atrioventricular block. As bradyarrhythmias can be episodic the 12-lead ECG can be normal.

What additional investigations may be required?

In patients with episodic symptoms with or without ECG features of conduction disease, offer ambulatory Holter monitoring to detect an arrhythmia and correlate with symptoms.¹⁷ It is appropriate to refer the patient to secondary care for this. The period of ambulatory monitoring is decided by the frequency of symptoms: if symptoms are at least daily, a 24 hour recording may suffice, and if symptoms are at least weekly a seven day recording is appropriate. If symptoms are less frequent an event recorder can be used. Implantable cardiac

monitors are sometimes required to investigate infrequent episodes of syncope.¹⁸ A basic electrophysiology study can be considered if a bradyarrhythmia is suspected but not identified with ambulatory monitoring.¹³

An echocardiogram is recommended once a bradyarrhythmia is diagnosed, particularly in the presence of symptoms, to assess left ventricular ejection fraction as this will influence choice of pacemaker.

As per expert consensus, cardiac magnetic resonance imaging is advised in patients <55 years old to exclude cardiomyopathy.¹⁹

When to refer?

All patients with symptoms and a bradyarrhythmia diagnosed on ECG will require referral to cardiology for further management. Patients who are clinically stable could be considered for outpatient cardiology referral. [Box 3](#) outlines red flags for immediate referral to secondary care or emergency services.

Box 3: Red flags for immediate referral to secondary care

- Patient clinically unstable and shows signs of haemodynamic compromise, including hypotension, impaired consciousness, or chest pain
- Ongoing pre-syncope or syncope associated with any bradyarrhythmia
- Syncope with following features¹⁸:
 - Abnormal electrocardiogram
 - Heart failure
 - New or unexplained breathlessness
 - Syncope on exertion
 - Syncope without prodrome in a patient >65 years old
 - Family history of inherited arrhythmia or sudden cardiac death
- Persistent (that is, continuous, and not intermittent) type 2 second-degree heart block or complete heart block whether symptomatic or not (as per authors' opinion)

How is it managed?

Correct any reversible causes such as thyroid dysfunction, atrioventricular nodal blocking drugs, and electrolyte imbalance.^{13 16} When a patient presents with chest pain or an ischaemic ECG pattern, once ischaemia is treated, further observation on a coronary care unit is advisable until bradyarrhythmia resolves. Patients with haemodynamic compromise may have metabolic acidosis, acute renal failure, and hyperkalaemia due to hypoperfusion. Although metabolic derangement can cause arrhythmia, it is critical to not delay treatment of the bradycardia while addressing the metabolic derangement.

Currently there is no long term medical therapy for bradyarrhythmias, although isoprenaline or adrenaline is sometimes given in high dependency environments to increase heart rate for patients in extremis while arranging pacing. If bradyarrhythmia persists and the patient has associated symptoms, pacemaker implantation must be discussed with the patient.^{13 16} Pacemakers provide a lifelong treatment for the bradyarrhythmia and associated symptoms and prevent the risk of sudden cardiac death.

Complete heart block remains the most common indication for pacing, followed by sinus node disease.² For atrioventricular block, the suggested treatment varies with the degree of block. [Table 1](#) lists indications for pacing based on the type of heart block and presence of symptoms. The 2013 ESC and 2012 ACCF/AHA/HRS guidelines recommend pacing on prognostic

grounds irrespective of symptoms for type 2 second-degree atrioventricular block and complete heart block.^{13 16} For type 1 second-degree heart block, pacing is recommended when patients are symptomatic.¹⁶ Otherwise the condition can be considered benign in asymptomatic patients. Many physicians also offer pacemaker implantation to older people with asymptomatic type 1 second-degree heart block as there is greater risk of progression to complete heart block.

In contrast, pacing for sinus node disease is mostly for the alleviation of symptoms. Symptoms associated with a sinus rate <40 beats/min, sinus pauses >3 seconds, or chronotropic incompetence (the inability of the heart rate to increase with exertion) are conventional indications for pacing.^{13 16} Chronotropic incompetence can be difficult to determine, but in general it is unlikely if the heart rate rises to >100 beats/min on an ambulatory monitor or a treadmill test. Although sinus node disease is considered as a “safe rhythm” (meaning that it will not cause death by stopping), falls in older people are not completely safe. Current guidance is to offer pacemaker implantation in those with sinus pauses of >6 seconds even without symptoms.^{13 20}

With any pacemaker, a rate-response function can be activated to allow the pacemaker to increase the heart rate during exercise or activity. This function is typically used in patients with chronotropic incompetence.

What complications to watch for?

Once a pacemaker is implanted, at least annual follow-up is advised to check functioning of the leads, battery life, and any symptoms. This is usually performed in a device clinic run by physiologists with physicians available to provide input. Remote monitoring can be arranged which allows devices to download information to the pacing clinic over the internet, reducing the need for follow-up in person.

Table 2 describes the different pacemaker types, their indications, and their associated complications. Patients with a high pacing burden (more frequent pacing), as often seen in type 2 second degree heart block and complete heart block, can develop left ventricular dysfunction. Review new symptoms of shortness of breath, reduced exercise tolerance, orthopnoea, or cough and offer referral to a cardiologist for further evaluation. A transthoracic echocardiogram to assess left ventricular systolic function is often required. Upgrading the existing pacemaker to a biventricular pacemaker will allow improvement in left ventricular function by restoring synchrony. Other causes of left ventricular dysfunction such as ischaemic heart disease should also be considered.

What are recent advances in pacemakers?

Although most pacing devices implanted are conventional single or dual chamber pacemakers, the indications for more complex devices are broadening.

Biventricular pacing

A high burden of pacing can result in progressive impairment of left ventricular function in some patients, particularly those who already have some left ventricular impairment²¹ or are hospitalised for congestive heart failure.^{22 23}

As right ventricular pacing does not utilise the His-Purkinje system, the resulting delay in activation of the left ventricle, particularly the lateral wall, causes dyssynchrony in a manner similar to left bundle branch block (see supplemental video 1 online). Implanting a second lead to the left ventricle (**fig 3**) can

ameliorate the impact of right ventricular pacing in those who are likely to pace the ventricles a lot.²⁴

Although biventricular pacing seems to be helpful for patients with pre-existing left ventricular systolic dysfunction,²¹ the evidence is limited and conflicting for patients without pre-existing left ventricular dysfunction.²⁵ The unpublished findings from the randomised BIOPACE trial (1810 patients) show that, in the absence of left ventricular impairment, empiric biventricular pacing did not improve outcomes compared with right ventricular pacing in patients with underlying atrioventricular block.²⁵

The recent ESC heart failure guidelines²⁴ now recommend a biventricular device rather than a conventional pacemaker in patients with left ventricular impairment and probable high burden of pacing regardless of symptoms of heart failure. The guidelines also advise that, if patients with conventional pacemakers and high pacing burden develop newly documented left ventricular impairment, their device should be upgraded to a biventricular device.^{16 24}

Leadless pacemakers

Leadless pacemakers are a fairly recent development designed to overcome complications with conventional transvenous pacing, such as device erosion, device generator infection, and lead failures (**table 2**).^{26 27} These devices consist of a miniaturised pacing system, made up of an encased battery and electrodes that are introduced via the femoral vein and are attached to the right ventricle with a screw-in mechanism or tines (see supplemental video 2 online and **figs 4** and **5**). Prospective non-randomised multicentre studies have shown these devices to have promising safety and efficacy profiles,^{28 29} but they have not yet been directly compared with conventional pacemakers. These devices currently provide only right ventricular pacing and do not allow atrial sensing or pacing as seen with conventional pacemakers. They are therefore reserved for patients with atrial fibrillation and bradycardia. **Box 4** describes expected advances in pacemaker technology.

Box 4: What new developments in heart pacemakers can we expect?

Battery-less pacemakers—These harvest mechanical energy from cardiac contractions to power the pacemaker, which eliminates the need for replacing batteries. These technologies are in prototype, and one was presented at ESC Congress in 2014.³⁰ Current pacemakers require procedures to change the generator, with associated risks

His bundle pacing—This allows pacing through the intrinsic conduction system (by pacing the His bundle itself), thereby overcoming the risk of ventricular dyssynchrony. The His Optimised Pacing Evaluated for Heart Failure (HOPE-HF) trial is evaluating the effect of continuous His pacing versus no pacing or back-up pacing on exercise capacity in patients with left ventricular impairment and narrow or broad QRS complexes with typical right bundle branch block³¹

Leadless pacemakers that can sense the P wave and then pace the ventricle following this, and dual chamber leadless pacemakers (which allow an implant in both atria and ventricles which can communicate to achieve dual chamber pacing)

How patients were involved in the creation of this article

Three patients who have a pacemaker implanted reviewed the manuscript. Two patients particularly felt that awareness about new technologies is important to those in general practice to ensure continuity of their care was maintained. One patient felt it was important that patients understand the reason for pacemaker implantation and how their physician chooses a certain type of pacemaker. They also asked to discuss biventricular pacing and to highlight the rate-response function in pacemakers that can be fine tuned to the patient's activity. In response, we discuss the indications of and types of pacemakers in more detail and have added information on the rate-response function of pacemakers.

Education into practice

- Can you describe typical features on presentation and on ECG that would lead you to suspect bradyarrhythmia in a patient?
- Think about how you would discuss pacing as a treatment with a patient
- Have you had patients with a pacemaker in-situ develop symptoms of heart failure? How would you assess and manage them?

Additional educational resources**For healthcare providers**

- European Society of Cardiology. Cardiac pacing and cardiac resynchronization therapy: ESC clinical practice guidelines. www.escardio.org/Guidelines/Clinical-Practice-Guidelines/Cardiac-Pacing-and-Cardiac-Resynchronization-Therapy
 - Provides detailed evidence on indications for pacing and when complex devices should be considered
- Ponikowski P, Voors AA, Anker SD, et al. 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. *Eur Heart J* 2016;37:2129–200. <https://academic.oup.com/eurheartj/article/37/27/2129/1748921/2016-ESC-Guidelines-for-the-diagnosis-and>
 - Provides details on the indication for biventricular pacemakers

For patients*

- Arrhythmia Alliance. Treatments: Pacemaker. www.heartrhythmalliance.org/aa/uk/pacemaker
- Syncope Trust And Reflex anoxic Seizures (STARS) www.heartrhythmalliance.org/stars/uk/
- NHS choices. Pacemaker implantation. www.nhs.uk/conditions/PacemakerImplantation/Pages/Introduction.aspx

*These sites provide detailed information on pacemakers, including indication, procedures, and life after

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Tables

Table 1 | Common bradyarrhythmias and their electrocardiographic (ECG) features, risk factors, symptoms, and management

Bradyarrhythmia	ECG features	Risk factors	Symptoms	Management
Sinus node disease	Sinus pauses, sinus bradycardia, chronotropic incompetence	Rate controlling or anti-arrhythmic drugs Age Ischaemic heart disease Infiltrative cardiac disease	Lethargy, breathlessness, dizziness, syncope	Pacemaker implantation if symptomatic pauses >3 sec or without symptoms if >6 sec sinus pauses ^{13 16}
Atrioventricular node disease:				
1st degree heart block	PR interval >200 ms	As for sinus node disease	No symptoms	Pacemaker implantation if PR interval >300 ms and symptomatic with dizziness or syncope (presumed intermittent 2nd degree heart block Mobitz type 2 or complete heart block) ^{13 16}
2nd degree heart block, Mobitz type 1 (Wenckebach)	Progressive PR interval lengthening followed by non-conducted P wave	As for sinus node disease <i>plus</i> Young person with high vagal tone or athlete	Rarely symptomatic in young people, but suggestive of significant conduction disease in elderly people	Pacemaker implantation if symptoms of dizziness or syncope ^{13 16}
2nd degree heart block, Mobitz type 2	Fixed relationship between p wave and QRS, but only every 2nd or 3rd p wave is conducted	As for sinus node disease <i>plus</i> Myocardial infarction	Lethargy, dizziness, syncope, shortness of breath	Pacemaker implantation irrespective of symptoms ^{13 16}
3rd degree heart block (complete heart block)	P wave and QRS dissociation	As for 2nd degree heart block	As for 2nd degree heart block	Pacemaker implantation irrespective of symptoms ^{13 16}
Atrial fibrillation:				
Bradycardia	Slow ventricular rate	As for 2nd degree heart block	As for 2nd degree heart block	Pacemaker implantation if symptoms of dizziness or syncope ¹³
Atrioventricular block (complete heart block)	Regular ventricular rhythm	As for 2nd degree heart block	As for 2nd degree heart block	Pacemaker implantation irrespective of symptoms ¹³

Table 2| Pacemaker types, their indication and the associated long term complications.

Pacemaker type	Indication ^{13 16}	Long term complications
Single chamber PPM (one lead):		
Lead in RV position	Atrial fibrillation <i>plus</i> Bradycardia <i>or</i> Atrioventricular block (No atrial lead required as atrial fibrillation)	Device infection Lead failure Subclavian vein or superior vena cava occlusion Pacemaker syndrome LV impairment (with RV pacing burden >40%)
Lead in RA position	Previously indicated in sinus node disease, but this pacemaker type no longer recommended	As above
Dual chamber PPM (two leads, in RA and RV)	Sinus node disease 1st degree heart block (PR interval >300 ms) with symptoms 2nd degree heart block, Mobitz type 1, with symptoms 2nd degree heart block, Mobitz type 2 Complete heart block	As above
Biventricular pacemaker with no atrial lead (two leads, in RV and LV)	Atrial fibrillation with impaired LV and likely high RV pacing burden <i>plus</i> Bradycardia <i>or</i> Complete heart block	Device infection Lead failure Lead displacement Diaphragmatic or phrenic nerve capture Subclavian vein or superior vena cava occlusion
Biventricular pacemaker with atrial lead (three leads, in RA, RV, and LV)	Impaired LV and likely high RV pacing burden <i>plus</i> Sinus node disease <i>or</i> 1st degree heart block (PR >300 ms) with symptoms <i>or</i> 2nd degree heart block, Mobitz type 1, with symptoms <i>or</i> 2nd degree heart block, Mobitz type 2 <i>or</i> Complete heart block	As above
Leadless pacemakers (no leads, device itself in RV)	Atrial fibrillation <i>plus</i> Bradycardia <i>or</i> Complete heart block (No atrial lead required as atrial fibrillation)	Device infection Dislodgement Perforation Pacemaker syndrome

PPM = permanent pacemaker; RV = right ventricle; RA = right atrium; LV = left ventricle.

Figures

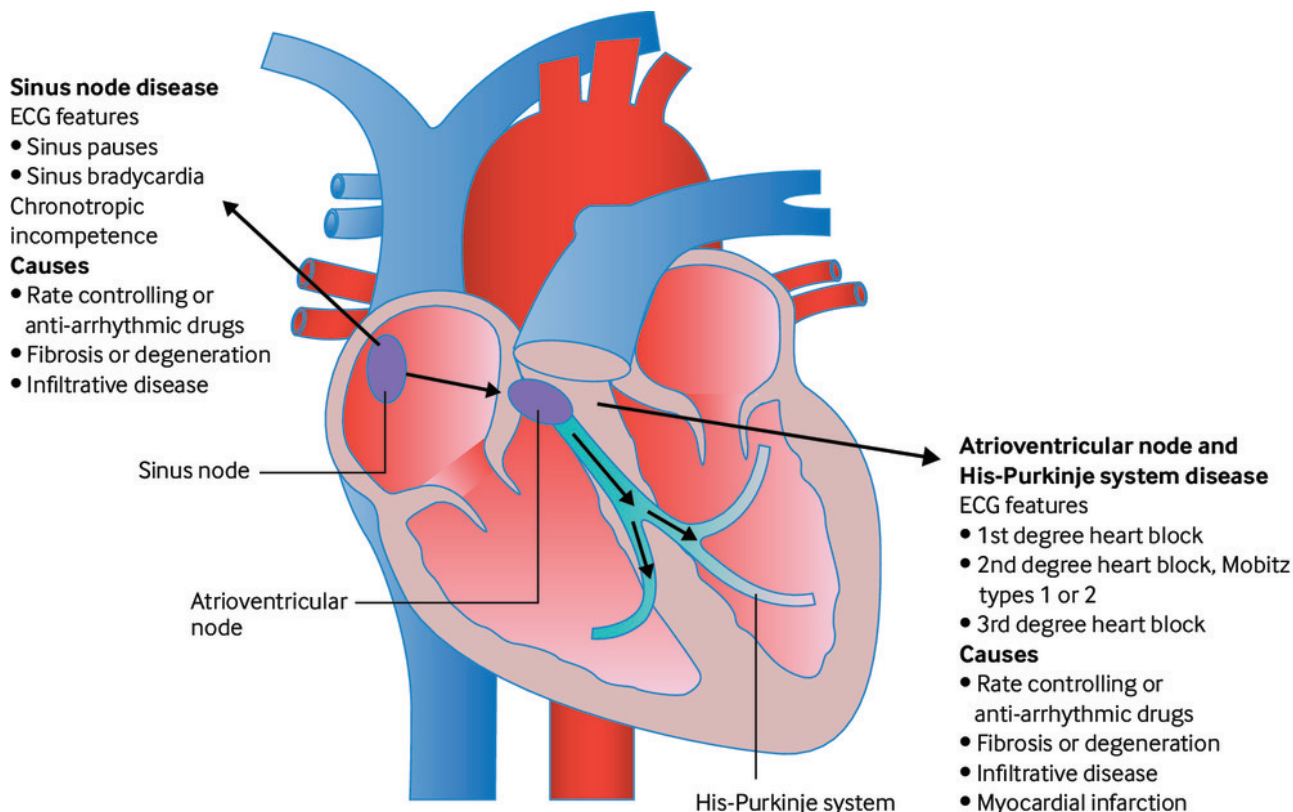


Fig 1 Position of sinus and atrioventricular nodes in the heart, with ECG features and causes of sinus node disease, first degree heart block, and atrioventricular node disease are described.



Fig 2 12-lead electrocardiograms showing features of (A) 3rd degree heart block (complete heart block where P waves are dissociated from the QRS complex) and a junctional escape (narrow QRS complexes); (B) type 2 second-degree atrioventricular block (Mobitz 2), where there is a fixed relationship between P wave and QRS, but only every third P wave is conducted); (C) type 1 second-degree heart block (Wencheback or Mobitz 1), where P-R interval lengthens progressively until a P wave is not conducted and the whole cycle starts again

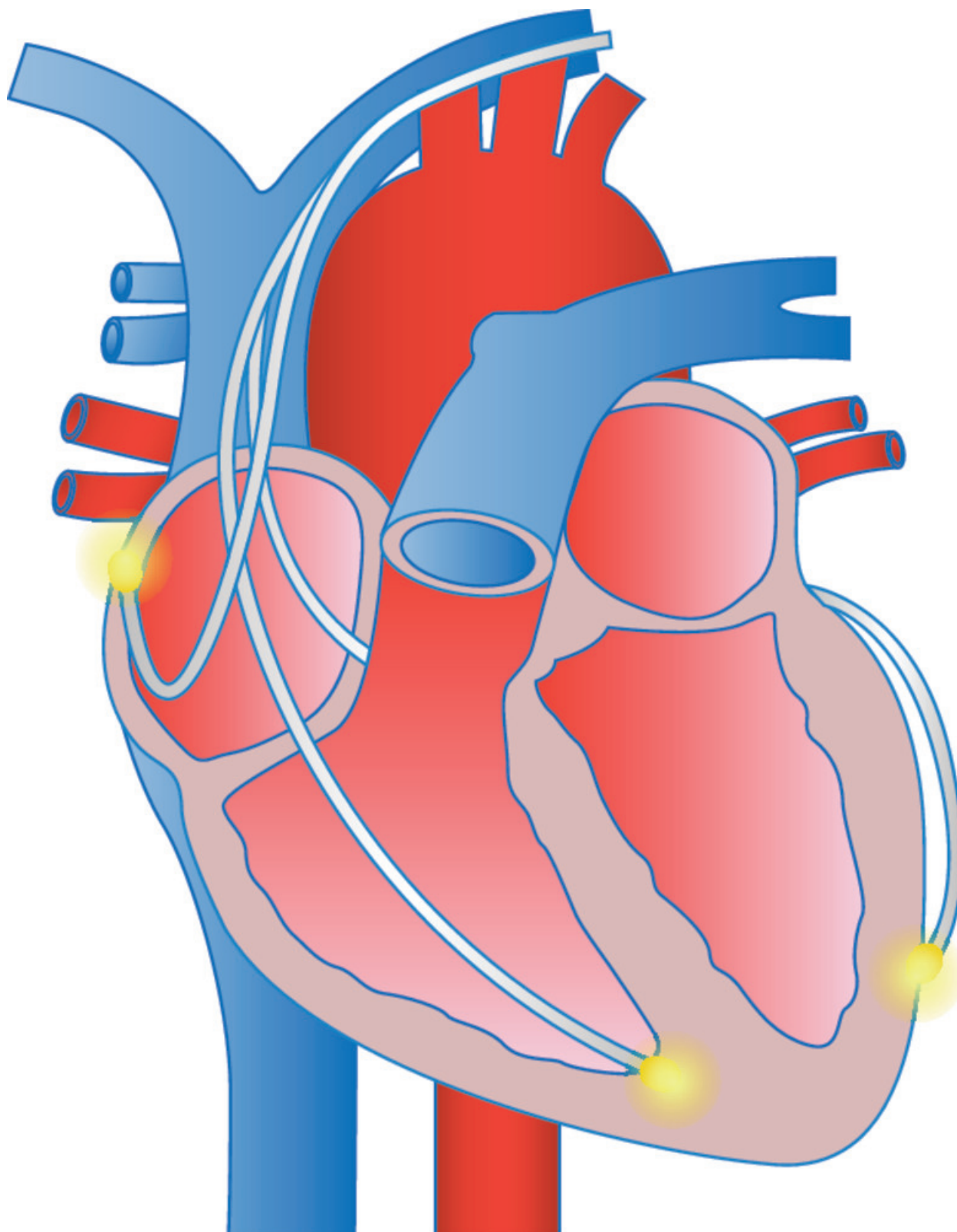


Fig 3 Biventricular pacing. Three leads enter the heart through the superior vena cava. One lead paces the right atrium, another paces the right ventricle, and a third passes into the coronary sinus (an opening of the cardiac venous system into the right atrium), where it passes inside the cardiac veins epicardially to run along the atrioventricular groove and then along small veins to the lateral wall of the left ventricle. This allows stimulation of both sides of the left ventricle simultaneously to minimise dyssynchrony.

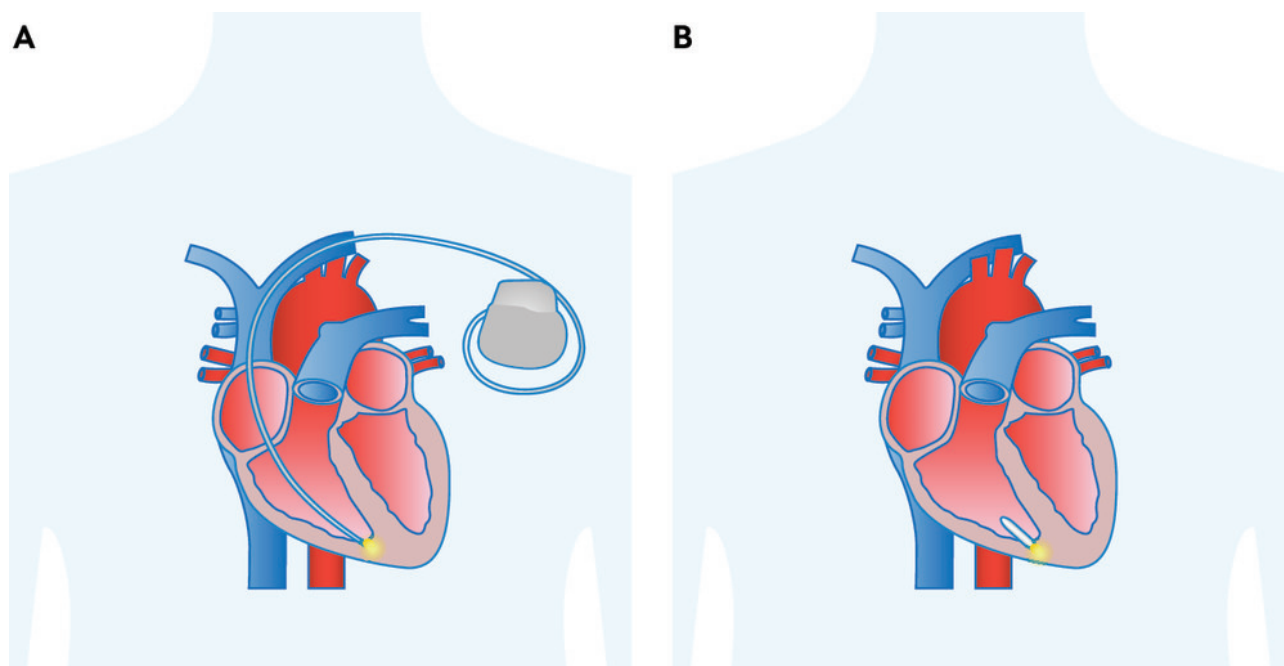


Fig 4 Conventional and leadless pacemakers. A conventional pacemaker sits under the skin near the collarbone, and the lead reaches the heart through the superior vena cava (A); whereas a leadless pacemaker simply sits in the right ventricle (B)

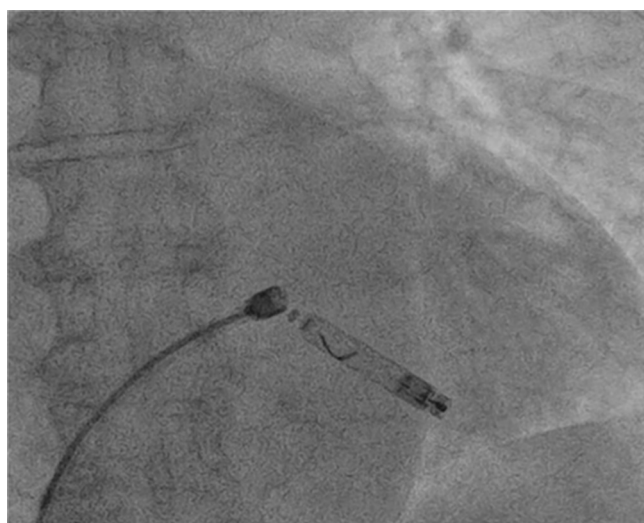


Fig 5 X ray image of a catheter inserted from the groin releasing a leadless pacemaker into the right ventricle. This was taken from the first implant of a leadless pacemaker in the UK, performed at St Bartholomew's Hospital on 13 January 2014