

Cardiac pacemakers

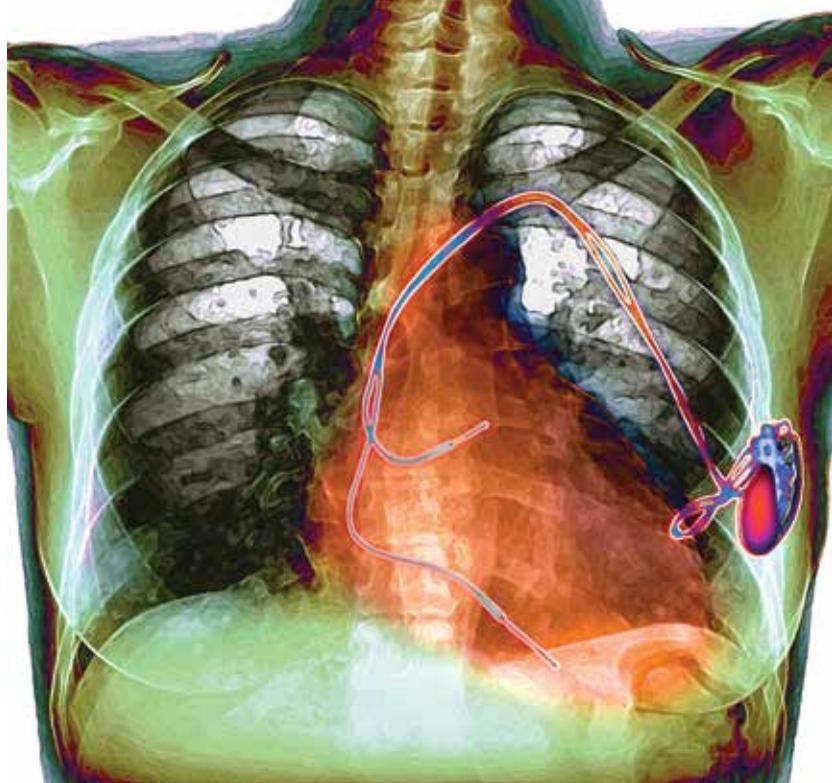
Keeping the beat

GERRY KAYE MB ChB, MD, FRCP(UK), FRACP, FCSANZ

Implantable pacemakers are the treatment of choice for patients with symptomatic bradycardia or high-grade atrioventricular block, allowing them to live near-normal lives. Technological advances include demand and rate-responsive pacing, which have improved battery longevity and exercise tolerance, and remote monitoring, which allows devices to be monitored in the patient's own environment.

Key points

- Pacemakers are the treatment of choice for patients with symptomatic bradycardia or heart block.
- Patients with pacemakers can live a near-normal life.
- Battery life of pacemakers is approximately eight to 10 years.
- Patients with any late swelling or persistent pain over the pacing generator require urgent referral to the local pacing unit.



Since the first human pacemaker implant in 1958, advances in electronic circuitry, miniaturisation and batteries have revolutionised pacemaker technology (Figure 1). Pacemakers are used primarily for the treatment of pathological bradycardia and/or heart block. There has been a steady increase in pacemaker implantation rates from the 1970s, and almost 16,000 were implanted in Australia in 2013.¹ Worldwide the rate of pacemaker implantation varies widely: 565 per million population in Australia, 518 in the UK, 767 in the USA and 927 in Germany.¹

Indications

Indications for a pacemaker are summarised in Box 1.² The most common indication is symptomatic bradycardia, which occurs in conditions such as:

- sinus node dysfunction (or sick sinus syndrome)
- atrioventricular block (AV; or heart block)
- atrial fibrillation (AF) with a slow and fast ventricular response (the tachy-brady syndrome).

Pacemakers are also indicated in some patients with heart failure and broad QRS complexes on the surface ECG who have no requirement for bradycardia support. This is referred to as cardiac resynchronisation therapy or biventricular pacing. As these patients are also at high risk of sudden death, a defibrillator is often required. These devices were discussed in more detail in recent articles in *Cardiology Today*.^{3,4} They will not be discussed further here.

Sinus node dysfunction

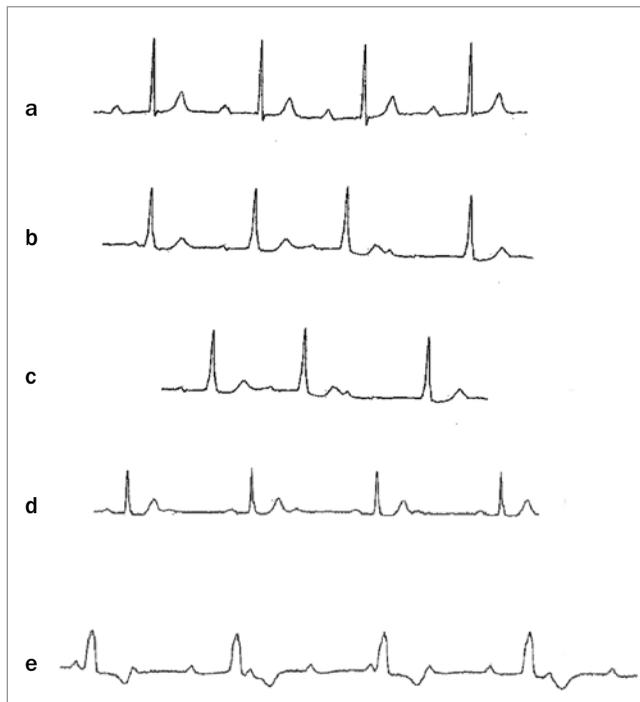
Sinus node dysfunction, or sick sinus syndrome, is an abnormality in the generation of the action potential by the sinus node. It is characterised by severe sinus bradycardia, sinus pauses or arrest (often longer than 3 seconds), often alternating with atrial tachyarrhythmias (paroxysms of AF and/or flutter). The incidence

CARDIOLOGY TODAY 2015; 5(4): 11-16

Dr Kaye is Associate Professor of Cardiology at the University of Queensland Medical School and an Interventional Electrophysiologist in the Department of Cardiology, Princess Alexandra Hospital, Brisbane, Qld.



Figure 1. Evolution of pacemakers, from the first early pacemakers (top right) moving anticlockwise to a smaller modern system (bottom right).



Figures 2a to e. ECG rhythm strips showing different types of heart block.
 a. First-degree heart block with prolongation of the PR interval.
 b. Mobitz type I block, or the Wenckebach phenomenon, showing progressive prolongation of the PR interval followed by a nonconducted P wave (a dropped beat).
 c. Second-degree atrioventricular Mobitz type II block. There is a constant PR interval and sudden failure of conduction to the ventricles after the third P wave, with no following QRS complex, resulting in a dropped beat.
 d. Second-degree 2:1 atrioventricular block with twice as many P waves as QRS complexes.
 e. Complete heart block, with a slow regular ventricular rhythm at 40 beats per minutes and a faster P wave rate that has no relation to the ventricular rate.

1. Indications for a pacemaker²

- Symptomatic bradycardia
- Acquired high-grade atrioventricular block, with or without symptoms (e.g. Mobitz type II, second-degree block or third-degree block)
- Atrial fibrillation with high-degree atrioventricular block or pauses of 5 seconds or longer
- Symptomatic poor left ventricular function after failure of medical treatment

increases exponentially with age, with a mean age at diagnosis of 68 years, and the two sexes equally affected. Sinus pauses of more than 5 seconds with presyncope or syncope usually require pacing. If atrial arrhythmias predominate then antiarrhythmic medication is usually required, which may exacerbate any bradycardia. A pacemaker is often then indicated to prevent severe symptomatic bradycardia.⁵⁻⁷

Atrioventricular block

AV block, or heart block, accounts for about 40 to 60% of patients requiring a pacemaker.⁸ It is categorised as first-, second- or third-degree (complete), as follows.

- First-degree AV block is defined as a PR interval of more than 200 ms (five small squares on the ECG) (Figure 2a). Pacing is rarely required.
- Second-degree AV block comprises:
 - Mobitz type I block (also known as the Wenckebach phenomenon), in which there is a gradual prolongation of the PR interval in sinus rhythm, culminating in a dropped beat (Figure 2b). Pacing is not usually required.
 - Mobitz type II block, in which there is no lengthening of the PR interval in sinus rhythm but a sudden dropped beat – i.e. no conducted QRS complex (Figures 2c and d). Pacing is mandatory because of the unpredictable risk of sudden ventricular standstill.
- Third-degree (complete) AV block is characterised by a slow regular ventricular response of about 30 to 40 beats per minute, with no conduction of the sinus beats to the ventricles (Figure 2e). There is no relation between the P and QRS waves; atrial and ventricular contraction are independent. Complete heart block may be congenital or acquired. Patients with acquired irreversible heart block require pacing.

Tachy-brady syndrome

Some patients with permanent AF have an intermittently slow heart rate (Figure 3). Patients who have pauses of 5 seconds or longer, even if asymptomatic, and those who have pauses longer than 3 seconds with symptoms of presyncope or syncope should receive a pacemaker. Frequently, drug treatment is also required



Figure 3. ECG showing a long pause during atrial fibrillation.

to slow rapid ventricular rates during AF, which will exacerbate any tendency to bradycardia. A combination of rate-slowing medications and a pacemaker is common.

Pacemaker technology

Types of pacemakers: single or dual chamber

All pacemakers initially used a single pacing lead placed in the right ventricle. This type of pacemaker ignored any contribution of the atria. They are referred to in the pacemaker shorthand nomenclature system as VVI (ventricular pacing, ventricular sensing, inhibitory) pacemakers (see Table).

Dual chamber pacemakers (known as DDD systems) were developed to mimic the normal conduction of the heart. They pace both the atria and the ventricles (Figure 4). In Australia, most pacemakers are DDD systems.¹ DDD pacemakers are usually implanted in sinus rhythm in patients with AV block or sinus node dysfunction, particularly if they are symptomatic.

Pacemaker components

All pacemakers consist of a battery and electronic circuitry hermetically sealed in a sterile metal case. Connected to this are wires or pacing leads, which are floated into the heart via the great thoracic veins to allow conduction of an electrical impulse to capture and stimulate the heart.

Battery

The battery is usually made of a lithium-iodine compound. On average, a pacemaker battery will last eight to 10 years, depending on how often the device paces and how many complex functions are enabled. Battery depletion is determined externally by interrogating the device electronically. Batteries rarely deplete suddenly, and often a warning of at least one year is given. Battery replacement involves day surgery (see below).

Leads

Pacemaker leads consist of an inner metallic core through which electrical energy is delivered to the heart. This core is insulated from body fluids by an inert external synthetic coat made of polyurethane, silicone or a newer copolymer of polyurethane and silicone.

Leads are of two types: passive or active fixation (Figures 5a

	Chamber paced	Chamber sensed	Pacemaker mode	Sensor
Ventricle	V	V	I*	R
Atrium	A	A	I	R
Dual chamber	D	D	D	R
Dual chamber – mode switched†	D	D	I	R

Abbreviations: A = atrial; D = dual chamber; I = inhibited; R = rate response; V = ventricular.

* In inhibited (I) mode, the pacemaker does not deliver a pacing signal when the patient's intrinsic rhythm is adequate. It starts pacing when the intrinsic heart rate falls below a predetermined level (also termed demand pacing).

† Dual chamber – mode switched pacemakers use a software-based system to detect the onset of atrial fibrillation and switch the pacemaker automatically to restore stable ventricular pacing. In contrast, the development of atrial fibrillation caused older dual chamber pacemakers to pace rapidly and erratically in the ventricle.

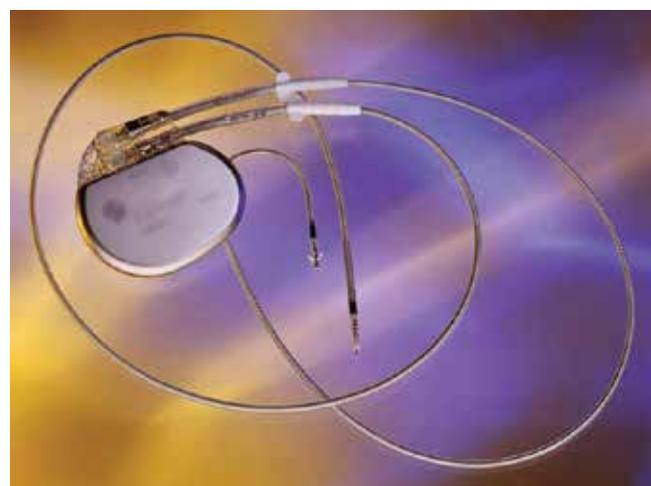
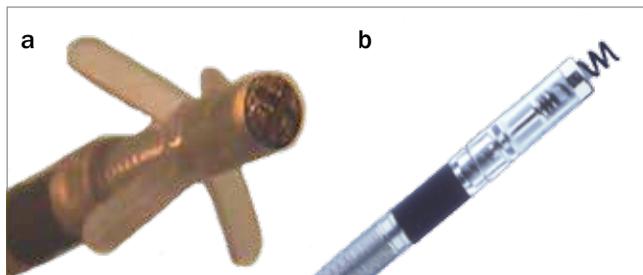


Figure 4. A modern dual chamber pacemaker with two leads inserted into the clear plastic 'header' of the device and held securely in place by small set screws. The pre-shaped J-shaped lead is placed in the atrium, with the second lead in the right ventricle.

IMAGE © 2015 MEDTRONIC. REPRODUCED WITH PERMISSION OF MEDTRONIC AUSTRALASIA PTY LTD.



Figures 5a and b. Pacemaker lead tips. a (left). A passive lead with plastic tines that self-lock into the muscular trabeculations within the right ventricular endocardium, designed to be placed at the right ventricular apex or high right atrium. b (right). An active fix lead with a mechanism that advances the screw to actively fix to the heart, which can be placed almost at any position within the heart.

and b). Passive leads have tines at the tip, similar to an arrow head, which are designed to anchor the tip to the endocardial muscular trabeculations. Their advantages are ease of implantation and a low risk of endocardial trauma. Their disadvantage is they are designed for placement only in specific parts of the heart: the apex of the ventricle or the atrial appendage.

Active fixation leads have a screw mechanism that allows the lead tip to be effectively positioned anywhere within the right side of the heart. Although there is an increased risk of the screw perforating the myocardium, this rarely causes serious adverse outcomes. In Australia, approximately 80% of leads are active fixation.

In the case of both passive and active fixation leads, the lead tip eventually becomes fibrosed to the myocardium making late displacement impossible. The lead body is highly resilient, flexing and twisting to accommodate daily cardiac contractions. Lead failure is rare (fewer than 0.05% fracture per year), and longevity is around 10 to 15 years. However, fractures do occur, and lead integrity can be assessed noninvasively by interrogating the pacemaker. Modern devices are able to check lead function on a daily basis and even to attempt automatic reprogramming to maintain patient safety. A broken lead needs to be replaced or, uncommonly, removed.

Demand pacing and pacemaker programming

Modern pacemakers have the ability to sense spontaneous cardiac depolarisations and inhibit pacemaker output when not required, thereby extending battery life. This is termed demand or inhibited pacing, designated by the abbreviation I (e.g. VVI mode).

Programming enables many functions to be altered to individualise pacing function after the device has been implanted; the pacing rate and the power output can be altered for both atrial and ventricular channels. Pacemakers are programmed non-invasively, which often requires only 5 to 10 minutes.

The endocardial signal or internal ECG can also be recorded. In some pacemakers, patients can externally trigger the pacemaker to store these signals when they experience symptoms, allowing it to act as a cardiomemo device.

Cardiac sensors

Demand pacemakers were initially able to pace the ventricles only at a constant rate. This resulted in a physiological mismatch, leading to shortness of breath or exercise-induced tiredness. Modern pacemakers have sensors capable of detecting changes in patients' exercise demands and responding appropriately. This is termed rate-responsive pacing. Pacemakers with this capacity are designated by the letter 'R' (Table). For example, a VVIR pacemaker paces and senses in the ventricle and responds to exercise. A DDDR system paces and senses in the atria and ventricles and responds to exercise.

Many different sensors are effective in responding to patients' daily demands. For example, a commonly used sensor detects changes in the frequency of body vibrations and is generally used in patients with sinus node dysfunction or AF with a slow ventricular rate.

Optimal pacing mode

If the primary disorder is sinus node failure, and AV nodal function is intact, then atrial pacing is preferred. In patients with complete heart block, no difference in mortality has been found between single chamber (VVI) and dual chamber (DDD) pacing, but the incidence of AF is less with DDD pacing. For patients with complete heart block, a rate-responsive ventricular demand (VVIR) pacemaker may be as effective as DDD pacing for those who are very elderly, but DDDR pacing is recommended for younger patients and for elderly patients with good exercise capacity.

Remote monitoring

A recent development is the ability to interrogate pacemakers remotely via either a cellular network or telephone landline. Data are transmitted to a central server, returned to the host country and downloaded locally on a daily basis. Many parameters can be interrogated, including battery longevity, lead integrity and complex functions such as the frequency of arrhythmias (e.g. AF). The devices cannot be programmed remotely because of safety concerns. However, recent studies have shown a life-saving benefit from remote monitoring, and Australia recently introduced a Medicare rebate to reflect this.⁸

External interference

All modern pacemakers are well shielded, and considerable interference is required to alter or damage a pacemaker. In normal daily life, exposure to high-level electromagnetic fields is unlikely. Equipment capable of generating a high-density current, such as arc-welding units, should be avoided. Airport and department store security systems are safe. If a pacemaker is overwhelmed by external interference then additional safety features switch off the sensing circuits, and the device paces continuously until the interference is resolved. Rarely, the device will sense extrinsic electronic noise and interpret it as an underlying QRS complex,

2. Websites with useful information about cardiac arrhythmias

- **Arrhythmia Alliance** (www.heartrhythmcharity.org.uk)
- **Arrhythmia Alliance Australia** (www.aa-international.org/au)
- **American Heart Rhythm Society** (www.hrsonline.org)

which may cause inhibition of pacing output and failure to pace, resulting in presyncope or syncope.

Effect on quality of life

- **Daily activities.** There are very few restrictions on daily activity after pacemaker implantation. Strenuous exertion, such as swimming, is best avoided for one month after implantation. Normal sexual function and exercise are allowed once the device has 'bedded in', about two weeks after implantation, and their recommendation depends mainly on the underlying cardiac pathology. The use of microwave ovens is permitted providing the patient does not stand very close to the oven.
- **Driving.** Patients who have had blackouts and an identifiable cause treated by pacemaker implantation are permitted to drive two weeks after the procedure for a standard vehicle licence, and one month after for a commercial licence.⁹
- **Flying.** Patients are advised not to fly for a minimum of 72 hours after pacemaker implantation. Travellers should inform security staff if they have a pacemaker as it can trigger metal detectors. Pacemaker implantation precludes piloting a commercial aeroplane. Noncommercial pilots must have their licenses assessed by the National Aviation Authority.
- **Pregnancy.** Women of child-bearing age rarely need a pacemaker. However, pregnancy is not contraindicated in those who do, and its advisability relates only to the underlying cardiac condition.
- **General surgery.** Diathermy should be kept at least 10 cm away from the pacing generator, and an ECG should be recorded throughout a general surgical operation. If close diathermy cannot be avoided then the pacemaker function can be protected by placing a strong magnet over the generator. As soon as the magnet is withdrawn, the pacemaker reverts to its normal function.
- **Telephones.** Mobile or cellular phones can interfere with pacemaker function, and it is recommended that the phone be held to the ear opposite the pacemaker.
- **MRI scanning.** In recent years, devices have been developed that are 'MRI conditional', meaning that certain MRI scans can be performed, in some cases even cardiac MRI. Patients are informed whether they can have an MRI scan with their particular pacemaker.
- **TENS machines.** It is not advised to use a transcutaneous

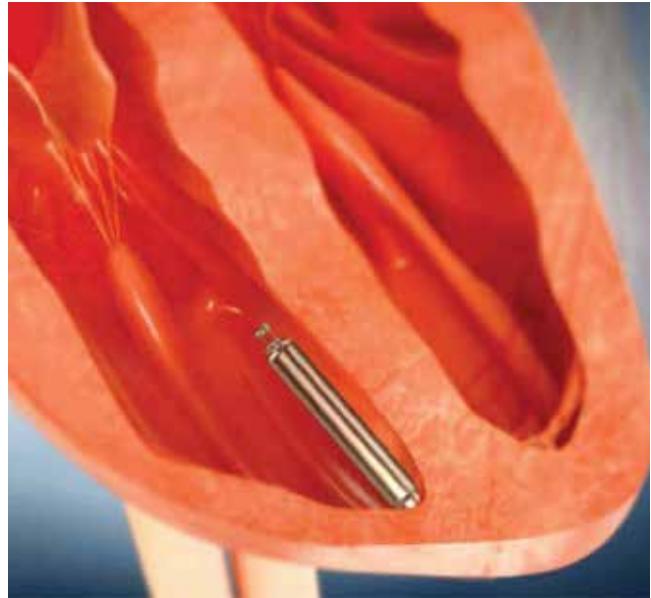


Figure 6. A leadless single chamber (ventricular) pacemaker. The generator containing the battery and all the necessary pacing components are miniaturised and implanted directly into the right ventricular wall via the femoral vein.

IMAGE PROVIDED COURTESY OF ST JUDE MEDICAL INC.

electrical nerve stimulation (TENS) machine close to a pacing generator.

- **End of life.** Pacemakers should be allowed to continue functioning in patients at the end of life, unlike implantable cardioverter defibrillators (ICDs), which may need to be deactivated (with patient or carer consent) to avoid distressing shocks in the last hours. In addition, pacemakers do not need to be deactivated after a patient has died, but they must be removed before cremation as incineration can cause an explosion. (ICDs should be deactivated before removal.)

Pacemaker implantation

Pacemakers are inserted under local anaesthesia with light sedation in a sterile environment.^{10,11} Patients are treated with antibiotics immediately before. By convention, pacemakers are inserted in the left side of the body under the clavicle, but they can also be inserted on the right side. The procedure takes 30 to 60 minutes. The pacing leads are positioned in the heart using x-ray fluoroscopic imaging. Patients are mobilised within a few hours and allowed home the following day. Some centres implant pacemakers as a day procedure.

Complications

After pacemaker implantation, bruising around the wound is common as is some discomfort, all usually short lived. Patients are encouraged to keep the left shoulder mobile to a degree as prolonged immobility can lead to frozen shoulder.

Acute complications of pacemaker implantation include:

- pneumothorax (1% risk)

- haemothorax (rare)
- death (rare)
- cardiac perforation (has been described and can, very rarely, lead to cardiac tamponade)
- lead displacement (with modern leads this is uncommon; it occurs more often with atrial than with ventricular leads)
- acute infection (rare).

Chronic complications include:

- late infection (serious but uncommon); an infected pacemaker site mandates removal of both generator and leads
- pacemaker erosion (rare and always associated with infection)
- lead fracture (uncommon); a broken lead needs replacement or, rarely, removal.

Pacemaker and lead removal

An absolute indication to remove a lead is infection. Removing a chronically implanted lead carries a small mortality of 1 to 3%. It involves the use of a laser-based or mechanical extraction device and is performed only in specialised centres.

Pacemaker follow up

Until the advent of remote monitoring, pacemakers required regular review every six to 12 months to assess battery life, lead integrity and the pacemaker pocket and to determine any changes in device programming. With the advent of remote monitoring, devices can be assessed daily, reducing the need for patients to return regularly to a specific pacing clinic. It is likely that in the future patients will be seen annually for wound review and clinical assessment. As remote monitoring detects abnormalities that would otherwise have been detected only at a routine follow up, it is likely that such detections will trigger earlier clinic review. At present, devices cannot be reprogrammed remotely, only interrogated

Role of the GP

Patients with symptomatic bradycardia need exclusion of noncardiac causes (e.g. hypothyroidism) and then cardiological assessment. Shortly after pacemaker implantation, the GP will be asked to assess the wound. At any time in the lifetime of a pacemaker if there is wound swelling, redness or a change in overlying skin colour (particularly a blue discolouration) then the patient should be referred back to the pacing unit for urgent assessment to prevent loss of wound integrity, which would mandate device removal. Under no circumstances should an attempt be made to drain any fluid by needle aspiration. Also, the development of symptoms such as increasing shortness of breath, progressive tiredness, presyncope or syncope should trigger re-referral of the patient to the pacing unit, even if they occur years after implantation.

Websites with useful information about cardiac arrhythmias are listed in Box 2.

The future

The Achilles heel of pacing is the intracardiac lead, which can break over time. Pacemakers without intracardiac leads are currently in clinical trials. These devices are sufficiently miniaturised to allow direct implantation into the endocardium of the right ventricle (Figure 6). Currently, only single chamber systems are available, but leadless dual chambers are in development. In the future, it is likely that leadless pacing will be the standard of care. **CT**

References

1. Mond HG, Crozier I. The Australian and New Zealand cardiac pacemaker and implantable cardioverter-defibrillator survey: calendar year 2013. *Heart Lung Circ* 2015; 24: 291-297.
2. Brignole M, Auricchio A, Baron-Esquivias B, et al. 2013 Guidelines on cardiac pacing and cardiac resynchronization therapy. *Eur Heart J* 2013; 34: 2281-2329.
3. Kaye G. Implantable defibrillators: preventing sudden cardiac death. *Cardiol Today* 2015; 5(3): 6-10.
4. Brazzale AG, Atherton JJ. New therapies in chronic heart failure. *Cardiol Today* 2015; 5(3): 18-21.
5. Adan V, Crown LA. Diagnosis and treatment of sick sinus syndrome. *Am Fam Physician* 2003; 67: 1725-1732.
6. Benditt DG, Sakaguchi S, Goldstein MA, et al. Sinus node dysfunction: pathophysiology, clinical features, evaluation, and treatment. In: Zipes DP, Jalife J, eds. *Cardiac electrophysiology: from cell to bedside*. 2nd ed. Philadelphia: WB Saunders; 1995. pp. 1215-1247.
7. Mangrum JM, DiMarco JP. The evaluation and management of bradycardia. *N Engl J Med* 2000; 342: 703-709.
8. Hindricks G, Taborsky M, Glikson M, et al. Implant based multi-parameter telemonitoring of patients with heart failure (In-Time): a randomised controlled trial. *Lancet* 2014; 384: 583-590.
9. Austroads, National Transport Commission Australia. Assessing fitness to drive for commercial and private vehicle drivers. Medical standards for licensing and clinical management guidelines: a resource for health professionals in Australia. 4th ed. Sydney: Austroads; 2012 (updated 2014). Available online at: <https://www.onlinepublications.austroads.com.au/downloads/AP-G56-13> (accessed October 2015).
10. Rajappan K. Permanent pacemaker implantation technique: part I: arrhythmias. *Heart* 2009; 95: 259-264.
11. Rajappan K. Permanent pacemaker implantation technique: part II. *Heart* 2009; 95: 334-342.

Further reading

Kaye G, Furniss S, Lemery R. *Fast facts: cardiac arrhythmias*. 2nd ed. Oxford, UK: Health Press; 2013.

COMPETING INTERESTS: Dr Kaye has received research grants from Medtronic and speaker fees from Biotronik, Medtronic and Pfizer.

Don't miss

Read more about pacemakers in GP Emergency Medicine on page 28.