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An update on cardiac implantable electronic devices for the general physician

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Abstract

Cardiac electronic device implantation is a common and important intervention for patients with tachy- and bradyarrhythmia. An increasing number of patients are receiving more complex devices such as cardiac resynchronisation therapy or devices with a defibrillation function. Over the last 5 years, two new models of cardiac device have emerged, subcutaneous defibrillators and leadless pacemakers. With an ageing population and data demonstrating 2000 per 100,000 of the population aged over 75 years have a cardiac device, it is essential that the general physician remains updated on the common pacemaker indications and available therapies.

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Cardiac electronic device implantation

A cardiac device system will comprise a pulse generator which is traditionally placed in a pre-pectoral pocket inferior to the clavicle on the non-dominant side where possible. Transvenous access is gained via the Seldinger technique into either the axillary or subclavian vein or via cutdown to the cephalic vein. A ventricular lead is most commonly placed in the right ventricular (RV) apex and a second lead can be placed into the right atrium (RA) as required (Figure 1). Following satisfactory checks, the leads are fixed into position. The mechanism of fixation depends on the lead chosen: active fixation leads use a screw helix which is deployed into the myocardium, while passive fixation leads use a barbed tip to passively tether the trabeculated myocardium. Fibrosis at the lead tip occurs over 4–6 weeks following implantation which permits more durable lead fixation. It is during this time that the leads are vulnerable to becoming dislodged. Most implantable devices have battery longevity of approximately 8–10 years although this depends on the complexity and frequency of therapy being delivered, as well as the make and model of the device.

Complications

The majority of studies report an overall risk of any complication occurring at 5–6%.[1] Early complications include haematoma, infection, haemo/pneumothorax, lead displacement and cardiac tamponade. The occurrence of pneumothorax is reportedly < 2% and the majority resolve spontaneously. Late complications commonly involve either lead displacement or disruption with 2.4% of leads requiring revision at 4 weeks.[2] Pulse generator box change or lead revision confers higher risk (haematoma 1%, infection 1%, lead damage 1–2%). Defibrillator implantation carries a higher risk of haematoma and tamponade given the heavier construction of the lead and pulse generator. Patients receiving cardiac resynchronisation therapy (CRT) have an additional risk of failure to place left ventricular (LV) lead, and LV lead displacement of 3–5%.[3]

Pacemaker nomenclature

There is a 5 letter pacing code to describe the fundamental device setup (Figure 2). Common modalities the general physician may encounter include VVIRO and DDDRO. VVIRO reflects pacing and sensing occurring within the ventricle, pacing is inhibited when an intrinsic beat occurs and the pacemaker can offer rate response to exercise. VVIRO modality is implanted for those where sensing and pacing the atria is not required (as in atrial fibrillation). DDDRO implies both the RA and RV are paced and sensed, the pacemaker can be inhibited and triggered by events sensed in both cardiac chambers and can deliver rate response. DDDRO is commonly preferred in atrioventricular block.

Reversible causes

It is worth considering whether an underlying cause for a bradyarrythmia is present before proceeding directly to permanent pacemaker implantation. A study of 277 patients presenting to an emergency department with bradyarrhythmia

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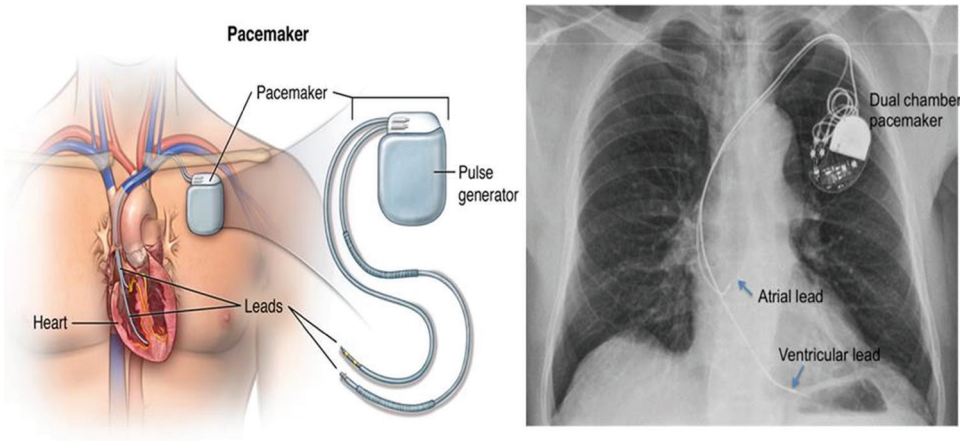


Figure 1 Demonstration of standard dual chamber pacemaker design and chest X-ray showing lead placement

Chamber/s Paced	Chamber/s Sensed	Response to Sensed Event	Programmability	Multisite Pacing
0= None	**0**= None	**0**=None	**0**=None	0=None
A= Atrium	**A**= Atrium	**T**= Triggered	**R**= Rate modulation	A= Atrium
V= Ventricle	**V**= Ventricle	**I**= Inhibited		V= Ventricle
D= Dual (Both)	**D**= Dual (Both)	**D**= Dual (Both)		D=Dual (both)

Figure 2 Summary of pacemaker nomenclature

identified a reversible cause in 45% of patients. These included medication effects, electrolyte or metabolic disturbances (hypo/hyperkalaemia, hypothyroidism), hypothermia, myocardial infarction, and recreational drug intoxication.[4] Often a period of stabilisation with initiation of rate stimulating medications and/or temporary pacing can be used until the underlying cause has been resolved.

Indications for brady pacing

There is no defined heart rate below which pacing is always indicated. Many patients may be asymptomatic despite profound bradycardia, while others can be symptomatic with a significantly prolonged PR interval alone.[5] It is primarily the presence of symptoms or prognostically significant conduction disturbance that guides pacemaker implantation.

The majority who present do so with presyncope, syncope or reduced exercise capacity.[6] Others may have more insidious symptoms such as lethargy or apathy. Capture of the electrocardiogram is critical in identifying the underlying pathology. This can be obtained with a resting 12 lead ECG, on ambulatory monitoring or, in the case of those with infrequent symptoms, an event recorder or implantable loop recorder.

Sinoatrial node disease

Conditions in which the sinoatrial (SA) node has impaired impulse generation or propagation are commonly seen in the seventh and eighth decade of life due to fibrotic change within and surrounding the SA node. Inflammation, infiltration and ischaemia are also common aetiologies.[7] SA disease may manifest with chronotropic incompetence (defined as failure to achieve 80% of expected maximum heart rate on exercise testing), sinus arrest and sinus pauses. Pauses may occur spontaneously or on reversion of atrial tachyarrhythmia to sinus rhythm (the tachy-brady syndrome). There is no evidence that cardiac pacing improves survival in these patients regardless of symptoms so pacemaker implantation is for symptom relief.

Atrioventricular node disease

The atrioventricular (AV) node is also prone to fibrotic or ischaemic insult resulting in intermittent or permanent AV block. This is understood by its three grades of increasing severity: first, second and third degree AV block. First degree AV block (PR interval > 200 ms) and Mobitz type I second degree AV block (Wenkeback) do not infer prognostic limitation and therefore pacing in these conditions is reserved for those with strong symptom-ECG correlation (particularly in the elderly population where conduction abnormalities are common). Mobitz type II, second degree AV block and third degree AV block are associated with poorer patient outcomes in terms of recurrent syncope and cardiac decompensation, therefore pacing is generally indicated in these patients regardless of symptoms.[8]

Bundle branch block

Bundle branch block (BBB) implies degradation of conduction fibres below AV node level and is seen on the ECG by QRS width > 120 ms and typical left BBB or right BBB patterns. General physicians may encounter the terms bifascicular and trifasicular block. Bifascicular block is defined as RBBB with either left anterior hemiblock (left axis deviation) or left posterior hemiblock on ECG. Trifasicular block is commonly described as prolonged PR interval + RBBB + left anterior

or posterior hemiblock. LBBB with left anterior hemiblock is suggestive of LV dysfunction. Half of patients symptomatic with syncope and BBB on ECG progress to AV block over 5 years.[9, 10] However asymptomatic patients with bifascicular or trifascicular block have an annual incidence of AV block of approximately 1% and no increased mortality. Therefore, empirically pacing these patients is not recommended and regular follow up is not usually required. Sudden cardiac death was observed in a third of patients with syncope, BBB and reduced ejection fraction ≤ 35% (EF).[11] In patients with BBB and heart failure, EF < 35%, or previous myocardial infarction, biventricular pacing with consideration of additional defibrillator modality is recommended.

Unexplained falls and syncope

This area is extensive and cannot be covered in detail here. It is best managed as per national guidance with involvement of falls and syncope specialist services as required. Taking a detailed history is vital and can help the physician select appropriate further investigations.[12] The SAFE-PACE trial demonstrated a 70% reduction in falls (but not syncope) in 80 patients with carotid sinus hypersensitivity who were fitted with a dual chamber pacemaker, highlighting the value of specialist falls and syncope input in this heterogenous group of patients.[13] Pacing is not indicated for patients with unexplained syncope without evidence of conduction disturbance on cardiac monitoring.[7] In cases where symptoms are infrequent, loop recorders can be implanted. These small devices are inserted under local anaesthetic and have a battery life of 2–3 years. In the event of symptoms, patients or relatives can activate the device to store recent cardiac activity. Patients should present to their local cardiac physiology department thereafter to have the trace downloaded and analysed.

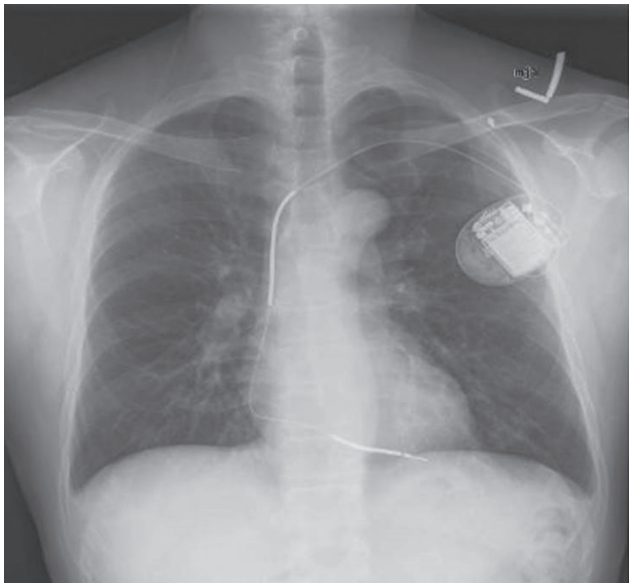
Implantable devices for tachyarrhythmia

An internal cardiac defibrillator (ICD) is larger than a standard pacemaker and although it can deliver bradycardia pacing, its primary function is to prevent sudden cardiac death. ICDs may be dual or single chamber and can be identified on chest X-ray by the one or two thick shock coils (Figure 3). If abnormal electrical activity triggers the ICD's pre-set algorithms, it will deliver therapy attempting to restore sinus rhythm. Anti-tachycardia pacing (ATP) is the first therapy usually delivered and relies on pacing the RV faster than the underlying arrhythmia to terminate the abnormal electrical circuit. Internal cardioversion takes place if ATP fails or VF is detected. Interestingly, ATP may not always be noticed by the patient and it is only at routine device follow up that therapies are seen to have delivered.

Some of the main examples of patients who may be offered an ICD include:

- Primary Prevention: commonly implanted in those with persistent severe LV systolic dysfunction (EF < 35%) despite optimal guideline directed heart failure therapy. Rare indications are the familial cardiac conditions inferring increased risk of sudden cardiac death, e.g. hypertrophic cardiomyopathy, long QT syndrome, Brugada syndrome, etc.
- Secondary Prevention: those with symptomatic ventricular arrhythmia or survivors of out of hospital cardiac arrest regardless of EF.

Figure 3 Chest X-ray showing lead placement demonstrating the thicker shock coils in this single lead ICD



The ICD relies on optimal programming to correctly categorise arrhythmia for individual patients; in some cases inappropriate activation or inaction may occur. This is usually either because a fast atrial-driven heart rate is being incorrectly categorised as ventricular arrhythmia or the ventricular rate is too slow to trigger the treatment algorithm. Inappropriate device function may be addressed by re-programming of the device by cardiac physiology services. Patients may be provided with remote home monitoring equipment which sends any device activity to the local pacing hub over the internet allowing for subsequent follow-up and treatment. Specialised cardiac physiology services often comprise an annual device check combined with cardiologist review, and 6 monthly remote monitoring review by a physiologist. The general physician should be familiar with which hospitals in their area have cardiac physiologists specialising in ICD management, and how to refer patients in and out of hours to this service.

Patients presenting with an ICD shock should be screened for causes of ventricular arrhythmia : infection, ischaemia, heart failure and electrolyte/metabolic abnormalities. As an ICD should serve primarily as a safety net and activation of the device is painful and frightening for patients, further endeavour may be made to suppress subsequent ventricular arrhythmia either through medical therapy or electrophysiology study and ablation. Sustained ventricular arrhythmias and shocks in the hospital setting should prompt urgent cardiology review and device interrogation by a specialist cardiac physiologist. The general physician should focus on reducing patient pain and anxiety with appropriate analgesia and sedation. They can

	NYHA Class			
QRS Interval	**I**	**II**	**III**	**IV**
< 120 ms	ICD if there is a high risk of sudden death			ICD and CRT not clinically indicated
120–149 ms without LBBB	ICD	ICD	ICD	CRT-P
120–149 ms with LBBB	ICD	CRT-D	CRT-P or CRT-D	CRT-P
> 150 ms with or without LBBB	CRT-D	CRT-D	CRT-P or CRT-D	CRT-P

Table 1 National Institute of Health and Care Excellent Guidance on treatment options with ICD or CRT for people who have heart failure with left ventricular dysfunction (EF <35%) according to NYHA class and QRS duration[17]

inhibit further shock therapy by leaving a magnet on top of the ICD. This will not alter the pacemaker component of the ICD but will inhibit the delivery of shock therapy. This allows for the physician to identify and treat the underlying arrhythmia, bearing in mind the cause of the shocks may not always be ventricular in origin.

ICDs can deliver brady pacing; however, this may not be its primary function. In some cases, it may have been programmed to commence pacing therapy at slower heart rates than with a traditional pacing device. As such, the general physician may encounter a patient with an ICD in situ with a resting heart rate below 50 bpm, and should not automatically infer malfunction of the device.

Subcutaneous ICD

The most common complication arising from transvenous ICD implantation is lead failure.[2] Subcutaneous ICDs (S-ICDs) offer the benefit of implantation without requiring any venous or myocardial contact and a reduction in the complication rates at implantation.[14, 15] These can be ideal in younger patients needing an ICD for primary prevention, or those with difficult venous access. The pulse generator is implanted in the left axillary line with a single lead tunnelled superiorly along the parasternal line. As the voltage delivered is higher in S-ICDs, and transmits with activation of surrounding skeletal muscle, therapy is more painful for patients and ATP is not possible. S-ICDs are not appropriate for patients requiring regular pacing or CRT. S-ICDs are more susceptible to interference from muscular activity and oversensing the T wave which can give rise to inappropriate shocks. Patients with poor electrical tracings or high interference would not be put forward for S-ICD.

Cardiac resynchronisation therapy

CRT is considered in patients with impaired LV systolic function, EF < 35% and interventricular conduction delay on the ECG (QRS duration > 120 ms). Alongside the conventional RV and RA leads, a third lead can be placed via the venous system of the coronary sinus to the surface of the LV. This aims for synchronised electrical and hence mechanical activation of both ventricles. This can increase cardiac output and studies have shown reduced all-cause mortality of 22% and reduced admission rate of 37%.[16]

Due to the multifaceted nature of cardiac dyssynchrony, the degree of symptomatic benefit patients derive from CRT varies. There is evidence that patients with broader QRS with typical LBBB pattern derive most benefit, and those with RBBB or non-specific bundle branch patterns derive the least benefit from resynchronisation.[17]

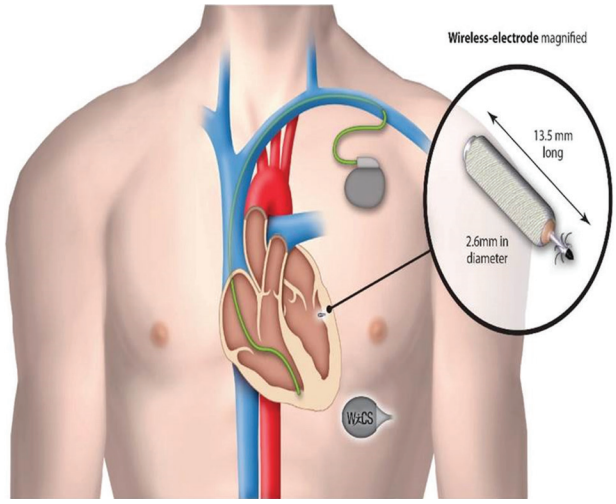
The prognosis of heart failure is poor with a 1 year mortality of 20% for those presenting to hospital with heart failure.[18] The main mode of death for those in NYHA (New York Heart Association) class II-III heart failure is ventricular arrhythmia. CRT with defibrillator function (CRT-D) is recommended for patients with impaired EF, broad QRS and reasonable functional status. For those in an advanced NYHA category or other major comorbidity such as renal impairment, malignancy or pulmonary disease, the defibrillator function is not recommended; rather CRT-P may be preferred. Table 1 summarises the National institute for Health and Care Excellent (NICE) guidance for ICD and CRT in patients with impaired EF < 35%.[17]

Leadless pacemakers

These 2–4cm devices are deployed into the RV via femoral venous access (Figure 4). They have a battery life of 3–5 years typically and are suitable for patients in whom standard lead access is untenable or non-desirable. These devices currently have limited programming functionality and can only deliver RV VVI pacing. Longevity trials are still ongoing[19] and questions remain regarding the safe removal of these devices, potential migration and deploying multiple devices over a patient's lifespan.

There is ongoing research into leadless CRT devices utilising an endothelialised LV electrode.[20] This grain-sized electrode is placed within the left ventricular endocardium and activated by a subcutaneously implanted pisoelectric ultrasound transmitter to enable synchronous biventricular contraction.

Figure 4 Representation of leadless LV pacemaker utilising standard RV pacemaker and a subcutaneous transmitter



Patients nearing the end of life

Given the prognostic impact of defibrillator devices as well as the pain and distress which occur on shock delivery, discussion about device deactivation should begin prior to device implantation and be reviewed whenever there is a serious deterioration in the patient's health. Often it is the non-cardiac pathologies such as malignancy, stroke or progressive organ failure that become more relevant to a patient's prognosis and as such it may also be the non-cardiologist that addresses end-of-life concerns and reviews the defibrillator.

Patients with capacity can refuse continued medical treatment and this includes cardiac pacemakers and defibrillators.[21] Deactivating the defibrillation function alone would allow pacing to continue but no therapeutic shocks would be delivered in the event of ventricular arrhythmia. It is important to ascertain the patient's underlying rhythm and disease process prior to deactivating a device, as this will allow an informed decision to take place.

Some patients, having no intrinsic cardiac rhythm, are dependent on the pacemaker function. It is unusual for a patient to request deactivation of their pacemaker as they cause no discomfort; however, in some cases it may be ethically reasonable to deactivate the pacemaker. As the dying process ensues, cellular metabolic derangements can make pacing ineffective. An active pacemaker will not be felt to affect the mode or timing of death, and placing temporary or permanent pacemakers in patients who are nearing death would be unlikely to alter outcomes.

If patients progress into NYHA class IV, the main cardiac mode of death is progressive pump failure. In these patients, the ability of the ICD to prolong life becomes limited. As ICD treatment may be distressing, deactivation should be considered to allow for patient dignity and comfort.

Aspects that should prompt defibrillator review would include a new life-limiting diagnosis such as incurable malignancy, neurodegenerative illness, refractory heart failure, on completion of Do Not Resuscitate orders. Any patient with an ICD or CRT-D in situ receiving end-of-life care or in whom a Do Not Resuscitate order is being contemplated must have device deactivation discussed. For the vast majority, device deactivation is appropriate, but this should be considered on a case by case basis. It is important to inform patients that deactivating defibrillator function will not result in immediate death, but rather, should ventricular arrhythmia arise in the future, the device will not act as a safety net.

Devices can be formally deactivated and reactivated by a cardiac physiologist or device specialist. In acute scenarios, a magnet left over a defibrillator will prevent anti-tachycardia therapies from being delivered. Magnets placed over standard pacemakers will have differing effects but will not cause cessation of basic pacing function. One should exercise caution placing a magnet over a simple pacemaker as there is a small risk of triggering an arrhythmia should asynchronous pacing be delivered.

Key points and frequently asked questions

Cardiac device follow up

Patients are usually invited for follow up in device clinic 6 weeks following de novo implantation to check satisfactory system function. Follow up at 6 or 12 monthly intervals is usual to check device function and battery. The nature and frequency of follow up will vary depending on device and individual circumstances. Patients are usually provided with contact information for their local cardiac physiology service should they or their physician have concerns about the device. Patients within the last 6–12 months of battery life tend to be listed for a generator box change, which usually takes place as a day case procedure.

Pacemaker infection

Pacemaker infection is described in 1–5% of cardiac device procedures and is more common following generator change or lead revision.[22] It can comprise vegetations on the pacing leads and/or pacemaker pocket infection. Vegetations may be present on other cardiac structures and there is a risk of embolisation causing distant infective foci.[23] Patients may present with swelling or discolouration of the pocket, pacemaker erosion and discharge from the wound, or the symptoms one would expect in subacute infective endocarditis, e.g., lethargy, fever and weight loss. These patients should generally be managed as per local infective endocarditis guidelines with emphasis on identification of the infective organism with appropriate blood cultures and wound swabs, cardiac imaging and early liaison with microbiology and cardiology. Pacing system infection can be difficult to treat with antibiotics alone and many patients require full system extraction to aid resolution.

Concerns of pacemaker malfunction

It is very rare for cardiac device hardware or algorithms to malfunction under usual conditions.[24] Prolonged exposure

to a strong electrical or magnetic field can temporarily disrupt the device function. It is normal for a pacemaker to allow intrinsic cardiac activity to occur wherever possible. As such, if the intrinsic heart rate is above the threshold programmed into the device, it will not deliver therapy. If loss of capture is seen on ECG one should consider lead disruption or displacement. A heavy fall onto the arm or shoulder can damage or displace the leads, and chest X-ray allows for lead fracture or displacement to be identified acutely. Patients noticing a vibratory or auditory alert from their device should contact their local cardiac physiology service ; these alerts are rare but can occur if there is a battery or lead fault. Any concerns with device programming or function can be discussed with the local cardiac physiology service. Pacemaker interrogation will allow for programming and function to be more thoroughly checked or updated.[24]

Frequent defibrillator activation

This usually occurs either because appropriate shocks are being delivered in the case of recurrent or refractory VT, or inappropriate shocks are occurring due to mis-sensing of a non-lethal arrhythmia by the device. In these cases, a magnet placed over the device will facilitate underlying rhythm detection, prevent further shocks from occurring and permit external control of the arrhythmia. It is safe to touch a patient while their ICD activates.

Patients requiring surgery

Defibrillators are usually temporarily deactivated prior to surgery involving diathermy as the current can trigger device activation. National guidance from the British Heart Rhythm Society may aid those performing diathermy on patients with an implantable cardiac device as special precautions are required.[25]

Patients needing cardioversion

Cardioversion should ideally take place electively with the support of the local cardiology service. In the acute setting, defibrillation pads should be placed in the antero-posterior position to avoid electrical interference with the device. All devices should be interrogated following cardioversion.

Patients needing an MRI

An increasing number of newer devices are MRI-compatible under certain conditions. It is always advisable to assume patients with cardiac devices are non-MRI safe until discussion with a cardiologist and cardiac physiologists has occurred. MRI scanning can occur in those with both MR conditional (and in some centres non-conditional) devices with appropriate cardiac liaison.

Driving

Full advice can be found on the DVLA resource for physicians.[26]

Conclusion

Cardiac electronic devices are increasingly implanted in an ageing and expanding population. The majority of indications are for brady pacemakers for SA or AV node disease although increasingly we are seeing more complex devices being implanted includingthe ICD and CRT systems recognisable by their shock coils or left ventricular lead on chest X-ray respectively. Recent advances in technology have allowed the development of leadless systems (pacing and defibrillators) and MRI compatible devices.

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