Cardiac device therapy 2: indications, techniques and complications

With advancing technology and ever-expanding indications for implantable cardiac pacing and defibrillation devices, this article reviews modern day practice in this field. This article focuses on topics pertinent not only to cardiologists but also to general physicians, medical trainees and allied medical specialties.

Part one of this series presented a basic understanding of the principles and theory of device therapy. Part two covers the day-to-day practice of cardiac device therapy in cardiology, looking in depth at international guidelines for device indication, the implant technique together with problems and complications of the procedure.

Indications for pacing

Bradycardia

Indications for bradycardia pacing can be divided into three major categories: sinus node disease, atrioventricular (below or above the bundle of His) disease and autonomic disease (*Figure 1a* and *b*). Secondarily to this, the presence or absence of symptoms can sometimes sway a decision to pace, such as in sinoatrial disease where absence of symptoms carries a much reduced indication for pacing. In certain circumstances the decision to pace is not related to symptoms, but to the long-term prognostic risk, such as adult-onset third degree atrioventricular block. Reversible causes such as early post-myocardial infarction and drug intoxication must also be taken into account when making a decision to pace. For a comprehensive list of pacing indications see *Tables 1–5* (pacing in children and post-cardiac transplantation are not covered but can be found in Vardas et al (2007)).

Tachycardia

Indications for pacing for tachycardia have largely evolved around the development of smaller and more reliable devices with greater longevity. Cost has been a major factor for implantable cardiac defibrillator (ICD) availability in 'rationed' health-care systems, but several large trials have been paramount in emphasizing the mortality benefit attainable, particularly in post-myocardial infarction patients with impaired left ventricular function.

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Indications for ICD therapy can largely be split into primary and secondary prevention groups with further subdivisions into ischaemic and non-ischaemic, with the

Figure 1. a. Indications for bradycardia pacing: complete atrioventricular block with ventricular escape rhythm. Observe the P wave activity (white arrows), faster than, and independent of the broad complex ventricular escape rhythm (black arrows). b. Sinoatrial disease showing marked sinus node bradycardia with a junctional escape rhythm. Observe the narrow complex junctional rhythm (black arrows) with intermittent and very slow P waves (white arrows) following a period of sinus arrest (brackets).

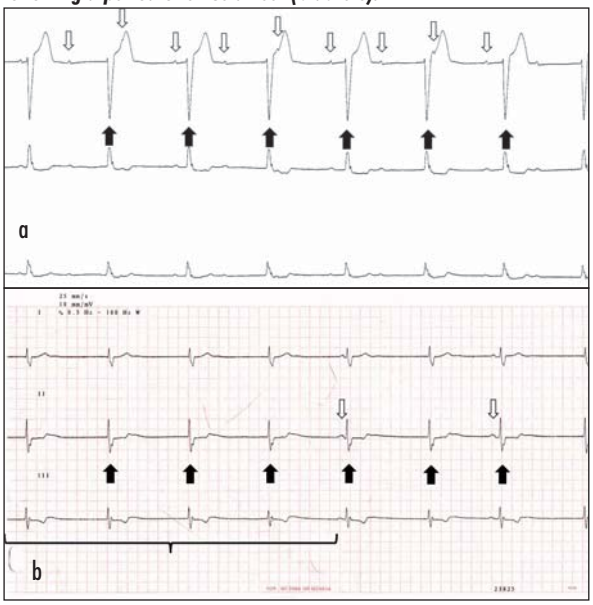

Table 1. Indications for pacing in sinus node dysfunction

Class I	Sinus node dysfunction with documented symptomatic sinus bradycardia/pauses
	Symptomatic chronotropic incompetence
Class II	Class IIa: Symptomatic patients with sinus node dysfunction and with no clear association between symptoms and bradycardia
	Class IIb: Chronic heart rate < 30 bpm in minimally symptomatic patients while awake
Class III	Asymptomatic sinus node dysfunction

Table 2. Indications for pacing in atrioventricular block

Class I	Third degree atrioventricular block associated with:	Symptomatic bradycardia (including those from arrhythmias and other medical conditions)
		Documented periods of asystole >3 seconds
		Escape rate <40 beats per minute in awake, symptom-free patients
		Post atrioventricular junction ablation
		Postoperative atrioventricular block not expected to resolve
	Second degree atrioventricular block regardless of type or site of block, with associated symptomatic bradycardia	
Class II	Class IIa	Asymptomatic complete heart block with a ventricular rate > 40 beats per minute
		Asymptomatic type II second degree atrioventricular block
		Asymptomatic type I second degree atrioventricular block within the His–Purkinje system found incidentally at electrophysiology study
		First degree atrioventricular block with symptoms suggestive of pacemaker syndrome and documented alleviation of symptoms with temporary atrioventricular pacing
	Class IIb	First degree atrioventricular block > 300 ms in patients with left ventricular dysfunction in whom a shorter atrioventricular interval results in haemodynamic improvement
Class III	Asymptomatic first degree atrioventricular block	
	Asymptomatic type I second degree atrioventricular block at supra-His level	
	Atrioventricular block expected to resolve and unlikely to recur (e.g. drug toxicity, Lyme disease, myocardial infarction, postoperative)	

overwhelming body of evidence being in the ischaemic group because of the high prevalence of the disease. Non-ischaemic hearts can be further subdivided into structurally normal or abnormal. The structurally normal hearts include those with inherited abnormalities of ion channels leading to rare conditions such as Brugada syndrome and long QT syndrome, whereas the most prevalent condition in structurally abnormal hearts is hypertrophic cardiomyopathy (Zipes et al, 2006).

Unless a clearly reversible cause is found, most cardiologists would find it difficult not to recommend ICD therapy in the setting of secondary prevention (i.e. survived out of hospital ventricular fibrillation or ventricular tachycardia arrest). In the UK, the National Institute for Health and Clinical Excellence (NICE) has recommended ICD implantation for primary prevention of sudden cardiac death based around criteria found in the large ICD trials such as SCD-HeFT, MADIT 1 and 2 and MUSTT (Moss et al, 1996; Buxton et al, 1999; Moss et al, 2002; Bardy et al, 2005), and relies on prior myocardial infarction with impaired left ventricular function and evidence of intra- or interventricular conduction delay in the form of a broad QRS. Patients without this documented bundle–branch block are required to demonstrate inducible ventricular tachycardia at electrophysiology studies to qualify (*Table 6*) (NICE, 2006).

The other largely studied group for primary prevention ICD is the hypertrophic cardiomyopathy group who require two high-risk features in their disease to qualify for an implant (*Table 7*) (Maron et al, 2003).

Heart failure

As biventricular pacing involves resynchronization of right with left ventricular contraction, the indications for pacing in heart failure generally require evidence of intra- or interventricular dyssynchrony on the 12-lead electrocardiogram. This is usually manifest as a broad QRS (i.e. bundle–branch block) with left bundle–branch block

Table 3. Indications for pacing in bi/trifascicular block

Class I	Intermittent third degree atrioventricular block	
	Type II second degree atrioventricular block	
Class II	Class IIa	Syncope not proved to be caused by atrioventricular block when other causes have been excluded, specifically ventricular tachycardia
		Prolonged His to ventricular interval (>100 ms)
		Pacing-induced infra-His block that is not physiological
	Class IIb	None
Class III	Asymptomatic fascicular block without atrioventricular block	
	Asymptomatic fascicular block with first degree atrioventricular block	

Table 4. Indications for pacing in atrioventricular block associated with myocardial infarction

Class I	Persistent and symptomatic second or third degree atrioventricular block	
	Persistent type second degree atrioventricular block in the His–Purkinje system with bilateral bundle–branch block or third degree atrioventricular block within or below the His–Purkinje system	
	Transient advanced second or third degree infranodal atrioventricular block and associated bundle–branch block	
Class II	Class IIb	Persistent second or third degree atrioventricular block at the atrioventricular node level
Class III	Transient atrioventricular block in absence of intraventricular conduction defect	
	Pre-existing first degree atrioventricular block with bundle–branch block	

Table 5. Indications for pacing in carotid sinus hypersensitivity and neurocardiogenic syncope

Class I		Recurrent syncope caused by carotid sinus stimulation; minimal carotid sinus pressure induces a period of asystole >3 seconds in duration (carotid sinus syndrome)
Class II	Class IIa	Recurrent syncope without clear, provocative events and with a hypersensitive cardioinhibitory response
		Syncope of unexplained origin when major abnormalities of sinus node function or atrioventricular conduction are discovered or provoked in electrophysiology studies
	Class IIb	Neurally mediated syncope with significant bradycardia reproduced by a head-up tilt table testing (vasovagal syncope)
Class III		Asymptomatic with a positive response to carotid sinus massage (carotid sinus syndrome)
		Recurrent syncope, lightheadedness, or dizziness without a cardioinhibitory response (carotid sinus syndrome or vasovagal syncope)
		Situational vasovagal syncope in which avoidance behaviour is effective
		Vague symptoms such as dizziness, light-headedness, or both, with hyperactive cardioinhibitory response to carotid sinus stimulation

Table 6. National Institute for Health and Clinical Excellence criteria for implantable cardiac defibrillator implantation

Secondary prevention	Out of hospital cardiac arrest
	Spontaneous sustained symptomatic ventricular tachycardia or ventricular fibrillation
	Spontaneous sustained asymptomatic ventricular tachycardia with left ventricular impairment (<35%)
Primary prevention	Left ventricular impairment (<30%) and QRS duration >120 ms
	or left ventricular impairment (<35%) and non-sustained ventricular tachycardia and inducible ventricular tachycardia on electrophysiology testing

From National Institute for Health and Clinical Excellence (2006)

Table 7. High-risk features for hypertrophic cardiomyopathy

Major features	Prior cardiac arrest
	Ventricular fibrillation, spontaneous sustained ventricular tachycardia
	Family history of premature sudden cardiac death
	Unexplained syncope
	Left ventricular wall thickness >30 mm
	Non-sustained ventricular tachycardia
	Abnormal exercise blood pressure response or left ventricular outflow tract obstruction
Possible features	Atrial fibrillation
	Ischaemic heart disease
	High risk mutations
	Extreme sporting activity
	Dilating phase or bridge to transplant

(compared with right bundle–branch block) predicting a favourable response (Egoavil et al, 2005). If the QRS duration is only marginally prolonged then an echocardiography tissue Doppler study, which accurately measures ventricular wall motion, can be used to further seek dyssynchronous contraction. Exact figures for QRS duration vary, but current guidance by NICE recommends biventricular pacing for QRS durations >150 ms and dyssynchrony studies for QRS durations of 120–149 ms. Indications for concomitant defibrillator implant are the same as described above (Barnett et al, 2007).

Implant techniques

Pacemaker implantation techniques are as wide and varied as the equipment available for the implant (*Figure 2*).

Asepsis

Where once upon a time pacemakers were implanted in a room just off the coronary care unit, more and more hospitals are moving to a sterile theatre environment with laminar air flow, deep cleaning schedules and all the precautions befitting of any operation. Most units will routinely screen patients for meticillin-resistant *Staphylococcus aureus* and use prophylactic antibiotics, either intravenously or 'in the pocket', to combat their local skin pathogens. The serious consequence of infection usually means the removal of the entire system.

Anticoagulation and/or antiplatelet agents

Perioperative anticoagulation and antiplatelet agents are usually stopped several days before the procedure. Local policy will govern exact regimens.

Preparation

Patients are generally preassessed and consented. Local policy may differ but will generally advocate a recent renal biochemistry profile, full blood count and group and save for the procedure. Patients shave pre-pectoral hair and wash using an antiseptic preparation preoperatively. Most implants use the left pre-pectoral region. Left-handers and patients with prior left-sided implants may have a right-sided implant. A cannula is usually inserted in the hand on the side of the implant in case a perioperative venogram is needed to visualize the ipsilateral subclavian vein.

Opening and access

Lidocaine is used to anaesthetize an area below the clavicle down to the surface of the pectoral muscle. An incision can be made parallel to or laterally and perpendicular to the line of the clavicle. The latter, more vertical incision, allows easier access to the cephalic vein. Most operators will at this point bluntly dissect down to the surface of the

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pectoral muscle to create a pocket to house the generator. Venous access is then sought and this is either done by directly visualizing the cephalic vein (a small tributary of the axillary vein) and opening this vein to allow the passage of the pacemaker leads, or by blind cannulation of the subclavian vein. Once venous access is secured, fluoroscopy is used to advance and position the leads.

Lead siting

Atrial

Atrial leads are generally positioned in the right atrial appendage but can occasionally be placed on the lateral wall, particularly in the post-coronary artery bypass grafting patient. Operator preference dictates the use of active or passive fixation, but the thin wall of the atria lends itself more to the gentler fixation of passive tines

Right ventricular lead

Right ventricular leads, classically, were placed in the right ventricular apex and fixed passively. The potentially deleterious effects of right ventricular apical pacing, however, has gained notoriety over the last few years and more operators are moving to positioning their leads actively either in the right ventricular outflow tract or on the interventricular septum. Clearly this has to be done using active fixation leads and is thought to provide a more normal physiological conduction and thereby reduce the negative consequences of apical pacing, namely heart failure and atrial fibrillation.

Left ventricular lead

Left ventricular leads used in biventricular pacing are not actually positioned within the left ventricle but in a posterolateral vein tributary of the coronary sinus which overlies the epicardial surface of the left ventricle. This is accessed by guiding the lead from the right atrium, into and up the coronary sinus and into the posterolateral vein. These leads are usually fixed passively.

Implantable cardiac defibrillator

ICD shocking leads follow generally the same principle as right ventricular apical lead placement.

Testing

All implanted leads are connected to an external pacemaker programmer. They are checked for their sensing and pacing parameters, together with their intracardiac electrocardiogram signals and impedance. Leads are often tested for their stability during manoeuvres such as sniffing and coughing and to ensure that they do not cause diaphragmatic stimulation through phrenic nerve stimulation.

ICDs are usually tested under general anaesthesia before closure of the wound. The device is connected and by a number of methods is used to induce ventricular fibrillation from the normal heart rhythm. It is then allowed to sense, recognize and internally cardiovert the ventricular fibrillation or ventricular tachycardia. Failure to do so usually requires external cardioversion followed by meticulous repositioning of the lead. After several failed repositioning attempts it may be necessary to add a second shocking coil to the superior vena cava to change the axis of depolarization of the shock.

Closing

Once the lead parameters are satisfactory they are secured and connected to the pacemaker header. The redundant portions of the leads are generally coiled behind the generator and the whole system implanted into the pre-pectoral pocket. The wound is closed using a variety of methods from sutures to glue.

Postoperative tests

Devices are usually tested in theatre and therefore do not require further checks on the ward. Electrocardiograms are usually performed to document appropriate pacing as is a routine antero-posterior and lateral chest X-ray to exclude pneumothorax and document lead position (*Figure 3*).

Complications

Pacemaker implantation is generally safe, especially when implanted in high volume centres by experienced operators. Most centres will quote a 1–2% risk of complications. The most common of these complications include:

Pain

Local pain and discomfort is the most common perioperative complaint, and to some degree depends on operator experience, difficulty of procedure, appropriate

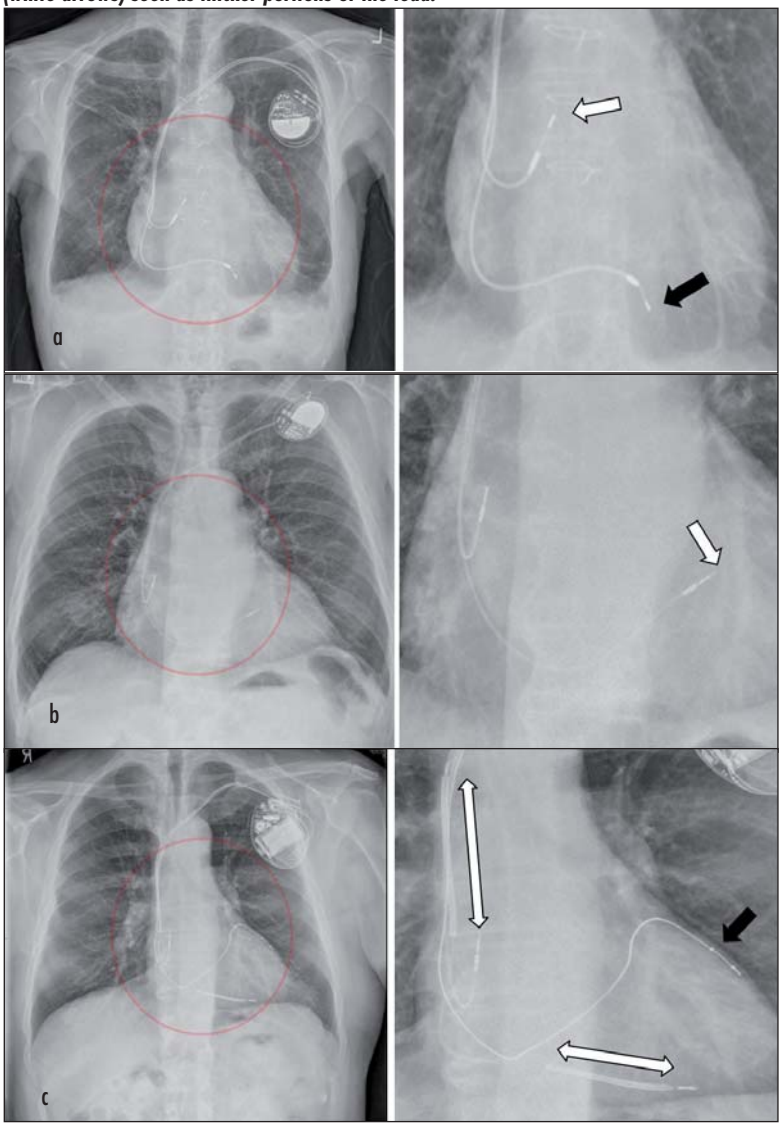
Figure 2. Pacing theatre and the implant procedure. a. Sterile theatre environment for device implantation. b. Fluoroscopic C-arm and imaging screens. c. Visualization and ligation of the cephalic vein. d. Blind subclavian vein puncture. e. Insertion of generator into pocket. f. Final result using glue.

sedation and pain threshold. Commonly, the most uncomfortable part is the creation of the pre-pectoral pocket.

Pneumothorax

During blind cannulation of the subclavian vein there is a risk of lung apex trauma and resulting pneumothorax (*Figure 4*). This likelihood is reduced by attempting an extra-thoracic approach to blind subclavian cannulation or further by direct cannulation of the cephalic vein. Although more time consuming, this latter method theoretically carries no risk of pneumothorax. Routine postoperative erect chest X-rays taken at >4 hours are reliable in excluding pneumothorax.

Figure 3. a. Postoperative chest X-ray confirming lead positions. Observe the dual chamber ventricular device with a lead in the right atrial appendage (white arrow) and a right ventricular lead pointing laterally and inferiorly in the right ventricular apex (black arrow); b. Observe the dual chamber pacemaker with a standard atrial lead and an active fix ventricular septal lead (arrow). c. Observe the biventricular implantable cardiac defibrillator with standard right atrial appendage and right ventricular apical leads together with a left ventricular lead entering the coronary sinus and positioned in a posterolateral cardiac vein (black arrow). The right ventricular lead is an implantable cardiac defibrillator shocking lead as evidenced by the distal and more proximal coils (white arrows) seen as thicker portions of the lead.



Cardiac perforation

Pericardial effusion or cardiac tamponade can occur during any implant procedure and is secondary to direct perforation of the myocardium with the pacing lead. Procedures requiring multiple lead repositioning, the use of active fixation leads and procedures in elderly, frail patients carry a higher risk of perforation. Symptoms of cardiac perforation range from local pericarditic pain to impending shock as a result of cardiac tamponade.

Vessel damage

Vessel damage in the form of venous tears and inadvertent subclavian artery puncture occur rarely and can present with extensive oozing and haemothorax.

Haematoma

Postoperative haematoma is commoner in patients taking antiplatelet or anticoagulant medication. It can present with local bruising and swelling and is often very painful. Unless extensive and tense, conservative management is appropriate because of the greatly increased risk of infection in reoperation and evacuation. Local pressure dressing and analgesia can help (*Figure 5*).

Lead failure or damage

Lead failure can occur as a result of a variety of reasons – some operator dependent, others not. Intraoperative lead damage to already existing leads (e.g. during generator change) often results in the need for a new lead insertion and possibly explant of the damaged lead.

Abnormal parameters picked up at pacing checks can often guide to the cause of lead failure. Lead displacement will usually occur early (within 6 weeks) and usually results in reoperation and resiting. left ventricular (coronary sinus) leads are more prone to displacement than right ventricular leads. These usually present with a high pacing threshold or with failure to capture or sense, but occasionally present with syncope in pacemaker-dependent patients.

Insulation or conductor failure may occur over time and is usually picked up on routine pacemaker checks with alterations in the lead's impedance or noise on the intracardiac electrocardiogram. Most leads will generally outlast one or two generators but can fail prematurely as a result of manufacturing defects or damage. The most common form of chronic damage to leads is caused by subclavian crush, a phenomenon where repetitive abrasion from the lead on the inferior surface of the clavicle damages the lead (e.g. in repetitive upper torso bodybuilding) (*Figure 6*). Lead fractures usually result in a very high measured lead impedance (>1500 Ω), whereas insulation breaks give rise to a very low impedance (<250 Ω).

A final cause of lead failure is as a result of exit block. In this situation the lead itself is intact but the pacing threshold rises significantly following the formation of a non-conductive fibrous cap at the lead–endocardium junction. This invariably needs replacing.

Pain, prominence, pre-erosion and erosion

Localized pain can be felt within the first few weeks but should not be ongoing. Conservative treatment of a painful or prominent pacemaker site is often favoured because of the increased risk of infection in repeat procedures, but a small proportion do require system revision. Occasionally burying the generator deep to the pectoral muscle can alleviate these symptoms.

Pre-erosion usually presents as tethering and redness of the skin overlying either the generator or loops of lead. The skin becomes thin and will ultimately break down if left, leading to full erosion (*Figure 5*). This latter condition should be avoided at all cost because of the usual requirement for explant and contralateral side re-implant.

Infection

Infection can occur on the skin, within the pocket, on the leads or on heart valves, particularly the tricuspid. It can occur early or late and is usually caused by skin commensals (*Staph. aureus* or *Staph. epidermidis*). All infections other than simple scar infections are likely to require whole system explants and contralateral re-implants (*Figure 5*). This becomes an issue when a lead has been in situ for over a year and is heavily fibrosed in place. Often the use of laser or rotational cutting equipment is then required with subsequent risk of cardiac or vessel damage. System explants generally carry a 1–2% mortality risk and so measures to prevent their necessity are important.

Costing

Pacemakers cost as little as £700 for a basic single chamber generator and approximately £17 000 for a state of the art biventricular ICD. Subtleties within the pricing brackets often depend on the purchaser's relationship with the device company and bulk ordering.

Leads themselves cost much less than the generators (approximately £200) but as their design becomes more sophisticated with lower profiles and defibrillation capability the price increases.

Device implantation nowadays is being carried out frequently as a day-case procedure thereby cutting the overall cost of the implantation procedure. It remains to be seen whether this strategy will also reduce infection rates, particularly hospital-acquired infections.

Conclusions

Cardiac device therapy is an advancing specialty, guided largely by the rapid increase in technological development. It encompasses not only standard pacemakers for bradycardia, but also defibrillation devices to prevent sudden arrhythmic death and resynchronization devices for heart failure. Where the standard implant technique (barring left ventricular lead placement) has largely stayed the same, the post-procedure programming and follow up has moved on immeasurably and requires a dedicated specialist to maximize the potential benefit attainable from the newer more sophisticated devices. BJHM

Conflict of interest: Dr Modi has received sponsorship and honoraria received from both Medtronic and Boston Scientific.

Figure 4. Chest X-ray showing left apical pneumothorax following pacemaker implantation.

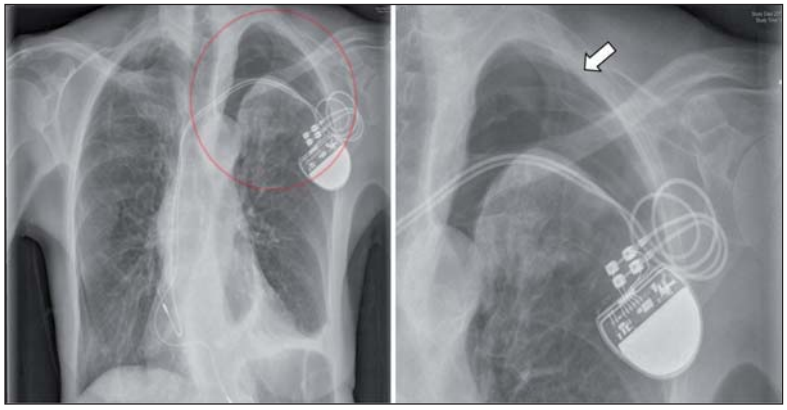


Figure 5. Pacemaker complications. a. Erosion of a pacemaker lead (arrow) through the skin in this very prominent pacemaker. b. Postoperative tense pocket haematoma. c. Infected, eroded pacemaker with tissue necrosis.

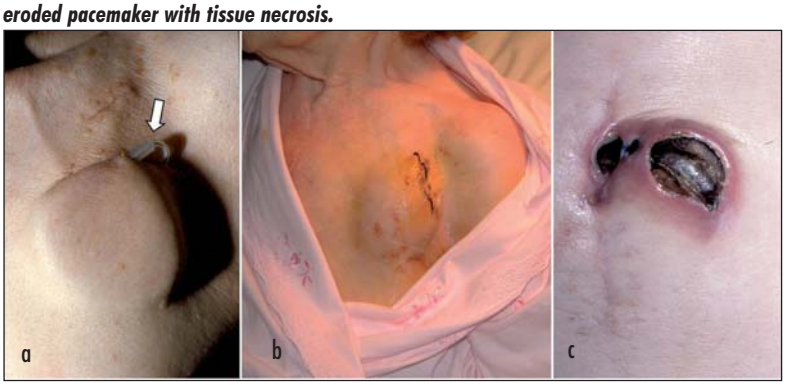
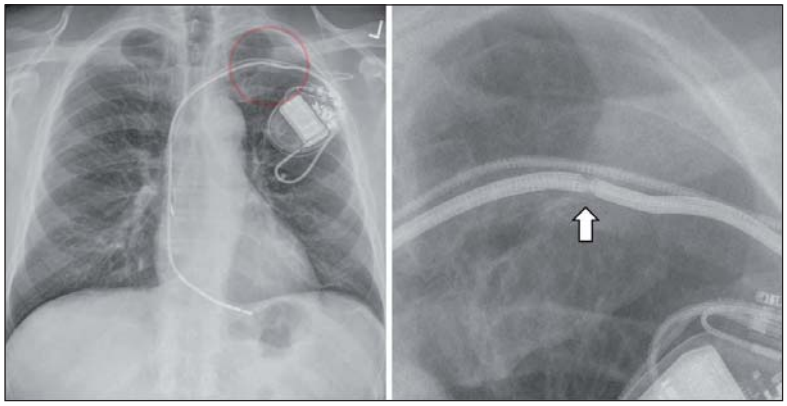


Figure 6. Chest X-ray showing damage to insulation as a result of subclavian crush syndrome. Observe the clear break in the insulation (arrow) at the point of most articulation with the clavicle.



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KEY POINTS

- Indications for bradycardia pacing are varied but generally can be classified as diseases of the sinoatrial or atrioventricular node, with or without symptoms.
- Indications for implantable defibrillators can be either primary or secondary, with primary indications varying depending on the underlying cardiac condition.
- Indications for biventricular pacing generally rely on left ventricular impairment and wide QRS morphology. Standard bradycardia indications for pacing are not a prerequisite.
- The device implant is a low risk procedure performed under local anaesthesia with serious complications occurring in approximately 1% of cases.

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