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Pacemakers and defibrillators: anaesthetic implications

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Cardiac pacemakers and implantable cardioverter-defibrillators have revolutionized the treatment of patients with cardiac arrhythmias. Since implantation of the first pacemaker in 1958, cardiac device therapy has seen a steady expansion. This is mainly attributable to phenomenal progress in device technology and software sophistication.

Recent data from landmark trials suggest that the indications for cardiac pacing and implantable defibrillators are set to expand further, to include, for example, heart failure, sleep disordered breathing and perhaps even routine defibrillator implantation in patients with myocardial infarction and poor ventricular function. This will inevitably result in more patients with cardiac devices being encountered by medical practitioners other than cardiologists.

This article reviews the basic principles of device nomenclature, function and physiology for pacemakers and defibrillators commonly encountered in surgical patients who may require anaesthesia.

Pacemaker and defibrillator mode codes

Pacemaker mode codes

The fundamental information required to understand normal pacemaker behaviour is the pacemaker code. The currently used North American Pacing and Electrophysiology/British Pacing and Electrophysiology Group (NASPE/BPEG) pacemaker codes[3] are used to describe pacemaker types and function (see Table 1). The first letter of this code indicates the chamber(s) paced and is designated *V* for ventricular pacing, *A* for atrial pacing and *D* for dual-chamber (atrial and ventricular) pacing. The second letter indicates the chamber in which the electrical activity of the heart is being sensed and is also denoted by *A*, *V* or *D*. An additional designation, *O*, has been used when pacemaker discharge is not dependent on sensed electrical activity. The third letter refers to the response to a sensed electrical signal. The letter *O* represents no response to an underlying electrical signal and is usually related to the absence of an associated sensing function; *I* represents inhibition of pacemaker output; *T* represents triggering of pacemaker output; *D* indicates a dual response: spontaneous atrial and ventricular atrial activity inhibiting atrial and ventricular pacing and atrial activity triggers a ventricular response.

The fourth and fifth positions are used to describe additional features of programmability and therapeutic pacing options. The fourth position of the code serves to describe the presence or absence of rate modulation (*R*). The fifth letter of the code, although seldom used, indicates the presence of one or more 'active' anti-tachycardia functions, whether initiated automatically or by command from a telemetric programmer.

Defibrillator mode codes

In 1993 a NASPE/BPEG defibrillator code was approved.[4] The four-position code describes defibrillator, arrhythmia diagnostic and data storage capabilities. The first position of the code indicates the shock chamber – none, atrium, ventricle or dual (*O*, *A*, *V* or *D*). The second position indicates the chamber in which anti-tachycardia pacing is delivered – also coded *O*, *A*, *V* or *D*. Position three indicates the means by which tachyarrhythmia is detected, either with the intracardiac electrogram (*E*) or by haemodynamic means (*H*). It is assumed that haemodynamic monitors include electrogram diagnostics. The fourth position of the code is the three- or five-letter code for the pacemaker capability of the device. For example, a ventricular defibrillator with haemodynamic or ECG tachyarrhythmia detection and with adaptive rate ventricular antibradycardia pacing would be labelled VOH-VVIR.

In Europe, patients with pacemakers or defibrillators carry device identification cards that include the current device programme code, the indication for implantation, and generator and lead information. Understanding the programme code is fundamental to anticipating pacemaker behaviour.

Table 1 The North American Pacing and Electrophysiology/British Pacing and Electrophysiology Group (NASPE/BPEG) pacemaker codes

Position I	Position II	Position III	Position IV	Position V
Pacing chamber	Sensing chamber	Response to sensing	Programmability	Anti-tachycardia functions
0=none	0=none	0=none	0=none	0=none
A=atrium	A=atrium	I=inhibited	R=rate modulation	P=pace
V=ventricle	V=ventricle	T=triggered		S=shock
D=dual (atrium and ventricle)	D=dual (atrium and ventricle)	D=dual (inhibited and triggered)		D=dual (pace and shock)

Basic physiology of cardiac pacing

When permanent cardiac pacing was first introduced, the principle aim – to prevent syncope or death from ventricular asystole – was easily achieved with single-chamber right ventricular pacing. Today, improved understanding of cardiac haemodynamics and parallel progress in pacemaker technology have expanded the indications for pacing and increased our expectations for symptomatic relief.

Rate-adaptive/rate-modulated pacing

A significant proportion of pacemaker recipients will have sinoatrial node disease or will eventually show sinoatrial dysfunction. The resulting chronotropic incompetence can manifest at rest, during exercise or both. Exercise is just one of the many circumstances requiring heart rate variation; emotion, anxiety, baroreflexes and vagal manoeuvres are all potentially important. A compensatory response is especially important in pathophysiological conditions such as hypovolaemia, fever and anaemia. The appropriateness of a pacemaker-generated rate response to a physiological stress is entirely dependent on the type of sensor used to detect the increased demand for the response of heart rate and the programmed algorithm to deliver the response.

The normal heart rate response to increased physiological demand is linearly related to oxygen demand/consumption. The ideal sensor and response algorithm would mimic this relationship. In addition, the change in pacing rate should occur with kinetics similar to those of the sinoatrial node. Ideally, sensors should be sensitive enough to detect both exercise and non-exercise needs for change in heart rate and yet be specific enough not to be affected by spurious signals.

Physiological sensors that detect the primary determinants of sinoatrial node function (e.g. circulating catecholamine levels or autonomic nervous activity) are still in development and are not available for implantation. The greater proportion of rate-adaptive sensors belong to a secondary class of sensors that detect physiological changes as a consequence of exercise, such as QT interval shortening,[37] increase in respiratory or minute-ventilation rate,[30 31 38 39] increased mean atrial rate,[16] rise in central venous temperature,[14 19] decrease in venous blood pH,[8] increase in right ventricular stroke volume[40] and increase in ventricular inotropy (e.g. peak endocardial acceleration[36] and ventricular impedence variation[43]). Each of these

sensors responds to an increased physiological stress with its own kinetics and has a different proportionality to demand. A third group of sensors and perhaps the most common used in clinical practice are tertiary sensors that detect external changes in response to exercise, for example, body movement.[23] As expected, the relationship between this variable and actual rate demand is less reliable and is therefore more susceptible to spurious signals.

There are few pre-anaesthetic considerations for the rate modulation function of pacemakers. Generally, rate modulation can safely be left activated during anaesthesia and surgery. It is important, however, to consider the location of an operative procedure in relation to the site of the generator when a movement sensor for rate modulation is in use. Operative movement in close proximity to such generators may stimulate unwanted tachycardia. Deactivating or reducing the sensitivity of rate-adaptive mode should be considered in these circumstances.

The importance of atrioventricular timing

In addition to restoration of rate adaptation, the aim of physiological pacing is to restore the sequence of activation. Initially, patients with complete heart block were treated with single-chamber right ventricular pacing. Although this pacing mode affords no synchrony with atrial systole, its benefits were clear as it prevented syncope and improved survival. These benefits were entirely related to improved heart rates. In patients with complete heart block, ventricular pacing produces a more physiologically appropriate heart rate and increases cardiac output. The increased cardiac output is associated with reductions in sympathetic tone, atrial rate and end-diastolic ventricular volume, and occurs despite a reduction in stroke volume and ventricular contractility.[25 41 46]

Early studies suggested that the maximal increase in cardiac output during ventricular pacing at rest occurs between 70 and 90 beats min^{-1}. Any further increase or decrease in pacing rate diminishes cardiac output and increases peripheral vascular resistance.[25 41 46] Two patterns of haemodynamic response to increased right ventricular pacing rate were demonstrated by Sowton.[46] In the flat response, cardiac output increased initially with pacing rate, and then remained relatively constant as stroke volume began to decrease with an increasing heart rate. This type of response was typical of patients with a structurally normal

heart with normal function and suggested that, at rest, cardiac output is relatively independent of heart rate. In the peaked response, cardiac output rises more gradually with heart rate until an optimum is reached, after which any further increase in rate results in a fall in cardiac output. This type of response is seen more commonly in patients with myocardial disease, for example, dilated cardiomyopathy, in whom cardiac output is more dependent on cardiac preload, afterload and myocardial contractility. Increasing pacing rate shortens diastolic filling times, reduces ventricular compliance and increases systemic vascular resistance, all of which limit the cardiac output achieved by increasing ventricular pacing rate alone.

Ventricular pacing can successfully maintain resting cardiac output and prevent syncope, yet it does not preserve the physiological relationship between the atrium and ventricle. The importance of synchronous atrial contraction has long been appreciated. Loss of atrial systole in atrial fibrillation can cause symptoms of congestive heart failure in individuals with normal hearts and a physiologically appropriate ventricular rate.[27 29 34 45] It was noticed that in patients with ventricular pacing for complete heart block, paced ventricular complexes which were immediately preceded by a p wave produced significantly greater stroke volumes and systemic pressure than those complexes without p waves. Moreover, the relative impact of a fortuitously timed p wave on stroke volume was greater at higher heart rates. These observations highlighted the physiological importance of atrial and ventricular timing. By the late 1970s, the appreciation of the haemodynamic significance of atrial systole and development in atrial lead and pulse generator technology, resulted in the clinical application of atrial-synchronous ventricular (AV) pacing.

Numerous non-invasive and invasive experiments have demonstrated a 10–50% improvement in cardiac output with AV sequential pacing compared with ventricular pacing alone in patients with normal and diseased hearts.[10 13 18 24 35 42] Higher left ventricular (LV) end-diastolic volumes[35 42] and mean and systolic arterial pressures[10 13] and lower venous pressures and pulmonary wedge pressures[18 25] have all been reported in AV sequential pacing compared with VVI pacing. The atrial contribution to resting cardiac output is therefore substantial; moreover, its proportion of contribution to cardiac output increases at higher heart rates. In addition to the loss of atrial contribution to cardiac output with VVI pacing, persistent AV asynchrony can result in significant mitral and tricuspid regurgitation due to the mistiming of AV valve closure. A variation of this phenomenon also occurs in first-degree heart block and can have significant haemodynamic consequences, especially if it occurs in the context of LV disease. In such circumstances, the regurgitation at the AV valves typically has a pre-systolic component and is particularly evident in patients with a long PR interval and LV disease. This pre-systolic (or late diastolic) component of regurgitation encroaches into and thereby reduces the duration of

ventricular filling time, and becomes more adverse the higher the heart rate. The pre-systolic component of regurgitation can even occur with a normal PR interval in patients with severe ventricular disease and broad QRS.[9] This pathophysiology can be readily corrected by re-establishing an appropriate AV delay with dual-chamber (DDD) pacing. Brecker and colleagues[6] first described this mechanism in the treatment of heart failure with dual-chamber pacing in patients with dilated cardiomyopathy and prolonged PR interval. However, with the development of atrio-biventricular (BV) pacing and the growing evidence of the harmful effects of chronic right ventricular pacing from the apex of the chamber,[50] the use of DDD pacing for the specific treatment of heart failure has been somewhat overshadowed by the ability to resynchronize the maltimed ventricular contraction by pacing both the left and right ventricles.

Ventricular asynchrony and biventricular pacing

Functional haemodynamic abnormalities in patients with asynchronous ventricular contraction have been well documented, particularly in those with depressed systolic function and delayed inter- and intra-ventricular conduction.[20 49] QRS duration exceeds 140 ms in almost 30% of symptomatic patients,[1 44 52] and mortality from heart failure increases dramatically with QRS duration greater than 170 ms.[17] Such conduction delays are associated with AV asynchrony, right and left interventricular asynchrony, and LV septal-to-free-wall intraventricular asynchrony. These patterns can result in prolonged isovolumic time, compromised diastolic filling time and ineffective atrial contribution to LV filling. The aim of BV pacing or cardiac resynchronization therapy is to optimize segmental electrical excitation, timing of contraction and relaxation, and consequently cycle efficiency.

Atrio-BV pacing uses a specialized LV lead in addition to standard right atrial and right ventricular leads. The LV lead is positioned (transvenously to the right atrium and thence to the coronary sinus and epicardial veins) on the lateral wall of the left ventricle. Simultaneous stimulation of the left and right ventricles can, to some extent, overcome the asynchrony caused by intraventricular conduction abnormalities. The advantages of ventricular 'resynchronization' with atrio-BV pacing have been demonstrated by improvements in cardiovascular haemodynamics,[5 26 28 32] exercise performance and patients' quality of life.

Current indications for this atrio-BV pacing include moderate or severe chronic heart failure. Therefore, unlike most patients with conventional single- and dual-chamber pacemakers, patients with these devices will already have significant morbidity and an increased anaesthetic risk. In patients with chronic heart failure, optimizing medical therapy before surgery with, for example, vasodilators, diuretics or beta-blockers is vital. It is equally important, however, to ensure that pacemakers are programmed

optimally. This is particularly important for BV pacemakers because, unlike most pacemakers, for which it is acceptable that the device paces only when required, BV pacemakers deliver a therapy (electrical ventricular resynchronization) with each ventricular paced beat.

Electromagnetic interference

The principle sources of electromagnetic interference (EMI) that affect implantable devices are found within the hospital environment. Radio frequency waves with frequencies between 0 and 10^9 Hz (e.g. AC power supplies and electrocautery) and microwaves with frequencies between 10^9 and 10^{11} Hz (including ultra high frequency radio waves and radar) can cause device interference. Higher frequency waves such as X-rays, gamma rays and infrared and ultraviolet light do not cause interference.

EMI may enter the pacemaker or defibrillator by direct contact between the patient and the source, or exposure to an electromagnetic field, with the device leads acting as antennae. Not surprisingly, bipolar leads (which have both electrical poles located at the lead tip) are much less vulnerable to EMI than unipolar leads, which sense and/or pace between an electrical pole at the lead tip and another within the pacemaker generator and therefore have a larger antenna loop that is susceptible to EMI. Bipolar leads can be programmed to be either unipolar or bipolar, whereas dedicated unipolar leads cannot sense or pace in a bipolar mode. Devices are generally protected by circuit shielding using titanium casing and by noise protection algorithms that filter out unwanted signals.

Possible responses to external interference include inappropriate inhibition or triggering of a paced output, asynchronous pacing, reprogramming (usually into a back-up mode), damage to device circuitry and triggering a defibrillator discharge. Asynchronous pacing and mode resetting are the most common outcomes of EMI and should be considered if pacing modes appear to change suddenly or intermittently on ECG monitors.

Asynchronous pacing

The refractory period

To avoid inappropriate sensing of electrical signals which may follow a paced ventricular beat, most devices have a refractory period which consists of two parts. The first period – the ventricular blanking period (VBP), also called the absolute refractory period – occurs immediately after each paced or sensed beat and lasts 200–300 ms. During the VBP, the lead is 'blinded' and will ignore any signals; in particular, it will not sense ventricular pacing pulse after-potentials or the evoked QRS and T waves. The following period is the noise sampling period (NSP) (also called the relative refractory period), during which time any sensed activity will reset the refractory period but does not reset the counter controlling the pacing interval (see Figure 1).

Interference behaviour

If an event is sensed during the NSP, it is interpreted as noise and, as a consequence, the entire refractory period is restarted. If further noise is detected, the refractory period is again restarted. Repetitive or continuous noise will eventually cause the lower rate (baseline pacing rate) to time out and a pacing impulse is delivered. Continuous noise therefore results in asynchronous pacing at the lower rate limit. These temporary changes will occur as long as noise is detected (see Figure 2). The lowest interference frequency at which the pulse generator switches to asynchronous pacing depends on the duration of the noise sampling window, which is typically 125 ms and corresponds to a frequency of 8 Hz.
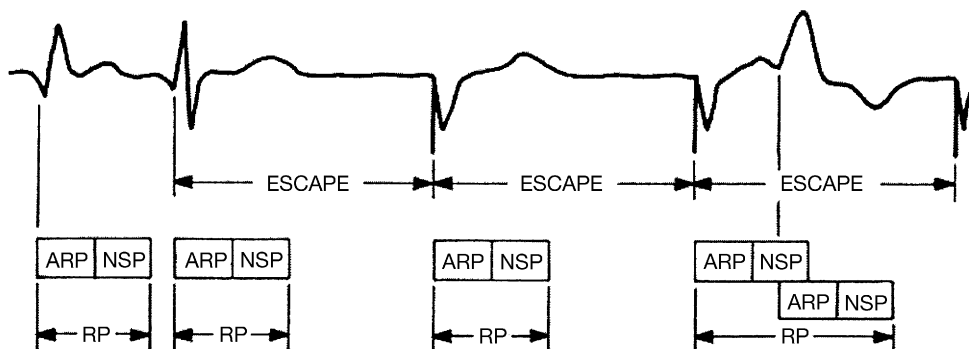

Fig 1 Pacemaker refractory periods (RP). This ECG demonstrates the timings of an inhibited pacemaker: the escape interval (the maximum time interval between ventricular complexes that the pacemaker will allow (i.e. the programmed basic pacing rate)), the absolute RP (ARP), the noise sampling period (NSP) and the RP. A sensed or paced event starts the RP. Sensing outside the RP restarts the escape interval (first and second complexes). The fifth complex is detected during the NSP – this restarts the RP but does not reset the escape interval. (Reproduced with permission from Sutton R, Bourgeois I. *The Foundations of Cardiac Pacing*, *Pt 1: an Illustrated and Practical Guide to Basic Pacing*. Mount Kisco, NY: Futura Publishing Company Inc., 1991.)

Pulsed interference outside the refractory period will reset the counter to allow an escape interval from the sensed event. Repetitive pulsed interference with intervals longer than the refractory period will continuously inhibit the generator and lead to prolonged asystole (see Figure 3).

To improve electrogram detection, modern pacemakers have a built-in interference monitor. The level of interference is stored and detection results in an abrupt rise of the input signal above the stored interference level. This type of detector response will eliminate low and medium levels of interference. At higher levels of interference, however, ECG detection is dependent on the phase relation between the electrogram and the interference. Because the electrogram has its own repetition rate, which is different from the interference rate, a quasi-random phase relationship for detection between the two signals results in intermittent sensing (see Figure 4). At extremely high interference levels the interference detector is saturated, resulting in asynchronous pacing.[47]

Mode resetting

In some devices EMI may also cause a pacing mode change. This is usually the 'backup' or 'reset' mode and is often the same mode as the 'battery depletion' mode. This is a potential cause of confusion, as EMI and mode resetting may be misinterpreted as battery depletion and result in unnecessary replacement. Conversely, battery depletion may be misinterpreted as EMI and cause an unwary operator to reprogram the device when the battery is truly depleted. Both mistakes can easily be avoided with careful attention to telemetric interrogation.

The backup or reset mode is usually VVI or VOO – the latter resulting in competition with intrinsic rhythm. If the pulse generator has programmable polarity, then the backup polarity is usually unipolar. This may be particularly significant in patients with implantable defibrillators. The unipolar configuration is more susceptible to EMI pulses, which may be falsely interpreted by the device as a
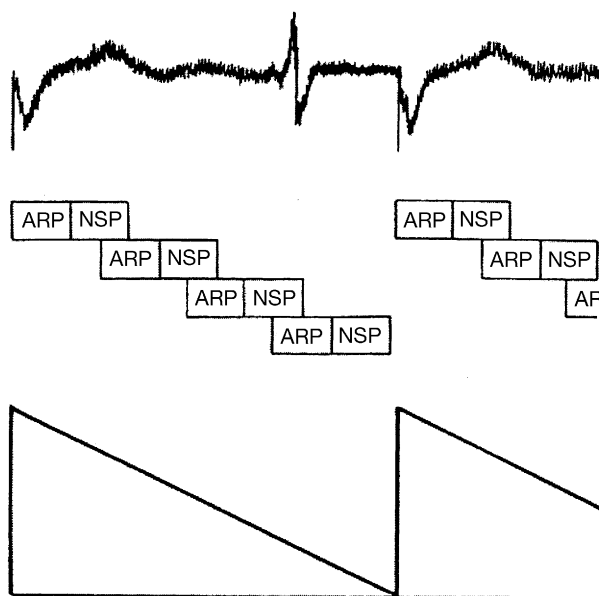

Fig 2 The effect of continuous interference. The ECG (upper panel) demonstrates continuous asynchronous pacing caused by continuous interference. Interference sensed during the noise sampling period restarts the refractory period, as shown in the middle panel. The lower panel demonstrates the escape counter, which generates the pacing pulse. (Reproduced with permission from Sutton R, Bourgeois I. *The Foundations of Cardiac Pacing*, *Pt 1: an Illustrated and Practical Guide to Basic Pacing*. Mount Kisco, NY: Futura Publishing Company Inc., 1991.)
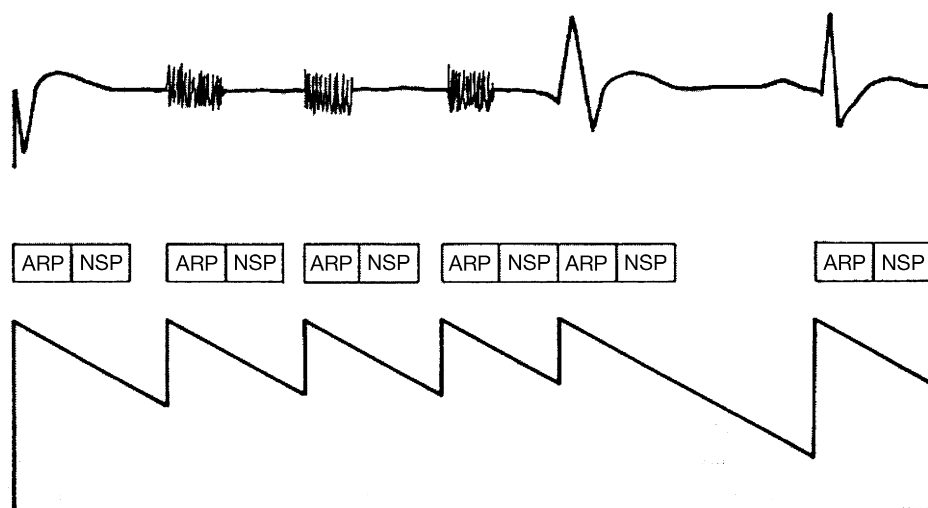

Fig 3 The effect of pulsed interference. The ECG (upper panel) demonstrates an asystolic pause with ventricular escape (second complex). The pacemaker was inhibited by pulsed interference. The middle panel indicates when pulsed interference was sensed. The lower panel shows the behaviour of the escape counter, which is reset three times because of interference and a fourth time by a ventricular escape beat. (Reproduced with permission from Sutton R, Bourgeois I. *The Foundations of Cardiac Pacing*, *Pt 1: an Illustrated and Practical Guide to Basic Pacing*. Mount Kisco, NY: Futura Publishing Company Inc., 1991.)
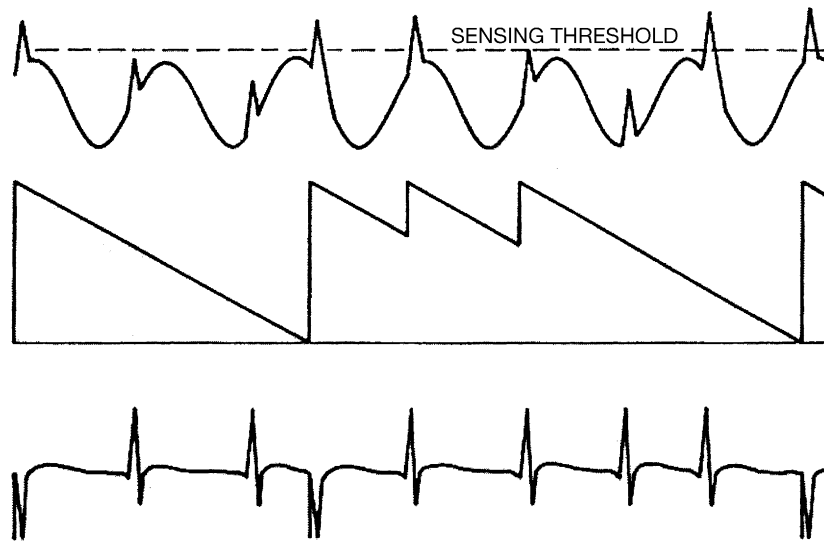
Fig 4 The effect of high levels of interference. The intracardiac electrogram (upper panel) demonstrates a high level of interference by a sine wave. All electrical activity above the sensing threshold is detected by the pulse generator. Escape counter behaviour is demonstrated in the middle panel. The ECG in the lower panel demonstrates intermittent sensing. (Reproduced with permission from Sutton R, Bourgeois I. *The Foundations of Cardiac Pacing, Pt 1: an Illustrated and Practical Guide to Basic Pacing.* Mount Kisco, NY: Futura Publishing Company Inc., 1991.)

tachycardia and result in delivery of unwanted electrical therapy. It is therefore essential that the implanted defibrillator is programmed bipolar or that a unipolar pacing configuration is thoroughly tested to prevent under- or over-sensing by the device. Bipolar programming is always preferable.

Hospital sources of interference

The most common sources of EMI are found within the hospital environment and a few commonly encountered sources are discussed here.

Electrocautery

Electrocautery remains one of the most common causes of EMI. Electrocautery uses radio frequency current to cut or coagulate tissues and is usually applied in a unipolar configuration between the handheld instrument (cathode) and the anode plate attached to the patient's skin. The radio frequency is usually between 300 and 500 kHz. The EMI generated by electrocautery that may affect the device is related to the distance and orientation of the current to the patient's device and leads.[11]

Electrocautery can evoke several responses from the pacemaker. Radio frequency signals may be interpreted as cardiac impulses, leading to inappropriate inhibition. Prolonged application of cautery can repeatedly trigger the NSP, resulting in asynchronous pacing, with function returning to normal when electrocautery is stopped. If electrocautery interference results in mode resetting, for example from DDD to VVI or VOO, AV synchrony will be lost and may result in haemodynamic embarrassment. Such resetting will persist even after electrocautery is stopped. In addition, implanted defibrillators may interpret electrocau-

tery interference as ventricular fibrillation, resulting in an inappropriate shock.

Other recognized complications of electrocautery interference include pacemaker circuitry damage resulting in output failure, activation of maximum rate response pacing, pacing lead overheating and myocardial damage and transient threshold alteration. A prospective study has shown that unipolar devices are far more susceptible than bipolar devices to electrocautery inference.[48]

Diathermy

During short-wave diathermy, current is applied directly to the skin and can be a source of interference. It should therefore be avoided near the generator site. Potential problems include overheating of the generator circuitry and damage to electronic components.

External cardioversion/defibrillators

One of the largest amounts of electrical energy to which implanted devices may be exposed is from external cardioversion or defibrillation. Such energies are liable to damage generator circuitry and cardiac tissue in contact with leads. Defibrillation with internal cardiac paddles requires less energy but may also interfere with pacemaker function.[7 51] Although pacemaker circuits are protected with cutout diodes, the high energies involved in cardioversion/defibrillation may override these mechanisms. More commonly, backup or reset modes are activated.

External defibrillation may induce high-energy currents through the pacemaker leads, which may be sufficiently high to cause burn trauma to the myocardium–electrode interface. The risk of this type of injury is much greater in unipolar configurations.[47] Most device manufacturers recommend that paddles are positioned as far away from the

generator as possible (at least 10 cm). For generators positioned in the left pectoral region, apex–anterior or apex–posterior positions are advised. For generators positioned in the right pectoral region, apex–posterior positions are more appropriate. It is always advisable to have the appropriate programmer available and it is always good practice to interrogate the device after external cardioversion/defibrillation.

Magnetic resonance imaging

During magnetic resonance imaging (MRI), a large magnetic field is generated with an electromagnet, using a radio frequency electrical signal of 30–3000 Hz. When a pacemaker is directly within an activated MRI electromagnet, the reed switch within the device closes and asynchronous VVO pacing occurs. Other reported problems have included inhibition of pacing and rapid pacing induced by the radio frequency signal,[21][22] discomfort at the pacemaker pocket and death of the unmonitored patient.[15]

Another serious complication is significant heating of the electrode tip during MRI exposure. Although the clinical effects of overheating have not been documented, studies with devices and leads placed within the field have generated electrode tip temperatures of up to 89 °C.[2] In general, patients with pacemakers should not routinely undergo MRI scanning. If no alternatives exist then scanning is best done in a carefully monitored environment with full cardiac support facilities.

Transcutaneous electrical nerve stimulation

Transcutaneous electrical nerve stimulation (TENS) is a widely used method of analgesia. The unit delivers electrical impulses via several skin electrodes at a frequency of 20–110 Hz in rectangular pulses of 1–200 V at 0–60 mA. Each pulse lasts 20 ms. Although the frequency length of the pulse falls within that of a normal heart beat, which theoretically may cause pacemaker inhibition, only asymptomatic inhibition detected on ambulatory monitoring has been reported.[12][33] Not surprisingly, these events were only recorded in unipolar programmed devices. In general, TENS can be used safely in patients with bipolar pacemakers and defibrillators, although use in close proximity to the device is not advised.

Pacemaker programming and the use of magnets

The pacemaker clinic and device interrogation

Regularly scheduled pacemaker clinic follow-up is an integral part of care for a patient with an implanted cardiac device. The three primary aims of the clinic are to predict the end of the life of the pulse generator battery, enabling elective replacement; to identify and permit prompt therapy of abnormality in the pacing system; and to provide clinical cardiological follow-up services when appropriate.

Fundamental information gained from device interrogation will include the pacing mode, stimulation thresholds, an assessment of sensing function and battery life status. Additional information useful for pre-anaesthetic evaluation may include a history of generator events (e.g. the frequency of defibrillator discharges or anti-tachycardia pacing). When planning elective surgery and anaesthesia, rescheduling pacemaker clinic appointments before the procedure is advisable, especially if device interrogation facilities are not available locally.

Pacemaker magnets

Magnet-operated reed switches within pacemakers were originally incorporated to produce pacemaker behaviour that would demonstrate remaining battery life and sometimes pacing thresholds. Activation of the reed switch shuts down the demand function of the device. The pacemaker will stimulate asynchronous pacing, allowing capture verification (see Figure 5). Some manufacturers have developed circuitry such that when the battery approaches depletion, the magnet pacing rate is different from the programmed baseline rate.

Applying the magnet can induce competitive pacing. This occurs when the magnet-induced asynchronous pacing competes with the patient's own heart rhythm, which may result in stimulation during a vulnerable period and introduce the risk of arrhythmia (see Figure 6). In modern pacemakers, the switch to asynchronous pacing is coupled to the next cardiac event to avoid competition at the outset.

In most programmable generators, applying the magnet over the generator opens the telemetric channel. After magnet application, some devices will pace, for a couple of beats, at a higher pacing rate but with shorter pulse width as a very basic threshold test (threshold margin test). This sequence simply confirms and reassures that capture still occurs at a stimulation energy level lower than what is actually programmed.

Magnets can be used to protect the pacemaker-dependent patient during diathermy, electrocautery or other sources of pulsed EMI and can be applied over the pacemaker to avoid inhibition by such pulsed interference. However, not all pacemakers will switch to a continuous asynchronous mode when a magnet is applied. Depending on the manufacturer and model, possible magnet responses include: no apparent change in rate or rhythm; brief asynchronous pacing (10–64 beats); continuous or transient loss of pacing; asynchronous pacing without rate response. It is always advisable to confirm magnet behaviour before magnet use. For any device, the manufacturer is the most reliable source of determining magnet behaviour. This information may be sought from the manufacturer directly or via the pacing clinic where the patient attends follow-up.
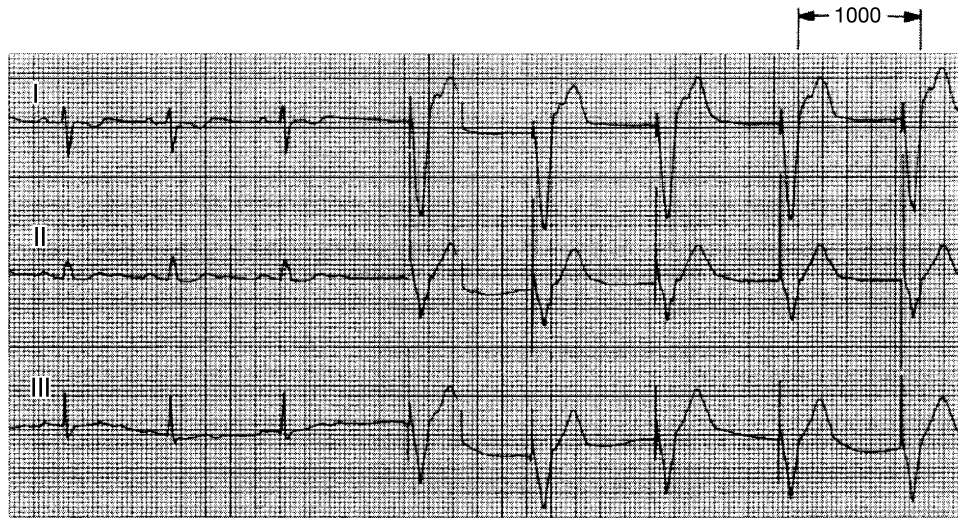
Fig 5 Magnet application to a VVI pacemaker. A three-lead ECG is shown. The first three complexes are of normal sinus rhythm with normal atrioventricular (AV) conduction. Magnet application at the fourth complex results in ventricular pacing, which continues. Note retrograde AV conduction with ventricular pacing. (Reproduced with permission from Sutton R, Bourgeois I. *The Foundations of Cardiac Pacing*, *Pt 1: an Illustrated and Practical Guide to Basic Pacing*. Mount Kisco, NY: Futura Publishing Company Inc., 1991.)
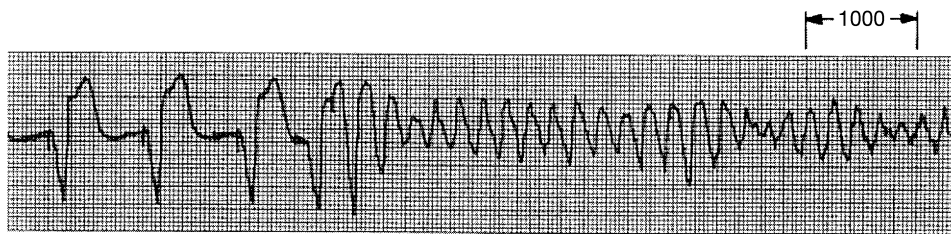

Fig 6 Ventricular fibrillation induced by a pacing stimulus in a vulnerable phase of a ventricular extrasystole. (Reproduced with permission from Sutton R, Bourgeois I. *The Foundations of Cardiac Pacing*, *Pt 1: an Illustrated and Practical Guide to Basic Pacing*. Mount Kisco, NY: Futura Publishing Company Inc., 1991.)

Evaluation of the patient with an implanted cardiac device

Preoperative assessment

When pre-assessing a patient with an implanted cardiac device, identification of the pacemaker, determination of pacing mode and knowledge of the primary indication for pacing is the first crucial step in anticipating normal device behaviour. Useful information from the patient includes details of when the device was implanted, when and where it was last checked and the anatomical position of the current active generator. Telemetric data of particular importance are battery status, reset mode information and confirmation of satisfactory thresholds.

The 12-lead ECG is an essential routine test for all patients considered for general anaesthesia. Although it only provides a 12-second snapshot of rate and rhythm, useful information can be gained from it, including confirmation of expected function, for example, AV synchronicity, polarity of pacing (determined by the amplitude of the pacing artefact on most, but not all, ECG machines) and baseline rate.

Re-programming the device will generally not protect the device against internal damage or reset caused by EMI. Re-programming is generally indicated to disable rate responsiveness and other enhancements, for example, hysteresis (dynamic AV delay). This is particularly important for devices with minute ventilation sensors for rate modulation, in order to avoid inappropriate tachycardias as a result of mechanical ventilation. Other indications that may require re-progamming include patients with special pacing indications, for example, paediatric patients and patients with hypertrophic obstructive cardiomyopathy or heart failure. In patients with a heart failure indication for pacing, echocardiography combined with telemetric programming is advisable for optimization of timing intervals before anaesthesia. These re-programming indications also apply to implanted defibrillators. Current American College of Cardiology/American Heart

Association (ACC/AHA) guidelines also advise that all anti-tachycardia therapy should be disabled before anaesthesia.

If the anticipated risk of EMI is high, for example, when electrocautery in close proximity to the generator is required, then precautions must be taken in case of the most extreme outcome (loss of ventricular pacing and ventricular asystole). Specifically, these precautions may include the use of magnets or temporary cardiac pacing. When magnet behaviour is unknown or is inappropriate in the event of ventricular asystole (i.e. does not allow asynchronous pacing), temporary transvenous or transthoracic cardiac pacing is an alternative. However, the efficacy of transthoracic pacing in stimulating the myocardium is highly dependent on electrode position and the patient's girth. If there is any doubt over its effectiveness, a transvenous approach with a temporary pacing wire before the procedure is recommended.

Intraoperative care

For any patient with a pacemaker, it is essential to identify the device so that its response to electrocautery is known. In particular, the backup mode should be determined. If extensive close-proximity electrocautery is required and loss of AV synchrony may compromise the patient haemodynamically (for example, heart failure patients), then it is advisable that a telemetric programmer and an experienced operator is present during surgery.

During surgery, bipolar electrocautery should be used whenever possible; if not, then the anode plate should be positioned as far away from the pacemaker generator as possible. Similarly, the cathode should be kept as far away from the device as possible, the lowest possible amplitude should be used and the operator should apply electrocautery in short bursts rather than continuously. Careful monitoring of the pulse, pulse oximetry and arterial pressure is essential during electrocautery, as ECG monitoring can also be affected by interference.

For the patient with an implanted defibrillator, facilities for external defibrillation should be available immediately after the device is disabled. If possible, remote pads should be used and applied in a suitable orientation (see above).

Postoperative care

Specific postoperative care should include a full telemetric check and re-programming back to the original setting if preoperative re-programming was required. Anti-tachycardia therapies of implantable defibrillators should obviously be re-programmed to their original settings.

Summary

The assessment and management of a patient who has an implanted cardiac device does require some special atten-

tion. Anticipation of behaviour, both of the patient and of the device, is the key to safe management and an uncomplicated procedure. When device interrogation facilities are not available locally, the most important information can be gained from careful history taking, inspection of the patient's device information card and by contacting the pacing clinic that the patient attends. Device manufacturers are also useful sources of information and support, and contact details are available on device identification cards.

Acknowledgement

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