OXFORD SPECIALIST HANDBOOKS IN CARDIOLOGY

# PACEMAKERS AND ICDS

Edited by
Jonathan Timperley
Paul Leeson
Andrew R J Mitchell
Timothy Betts

SECOND EDITION


CARDIOLOGY

OXFORD MEDICAL PUBLICATIONS

# Pacemakers and ICDs

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# Oxford Specialist Handbooks in Cardiology

# Pacemakers and ICDs

**2nd edition**

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# Foreword to the first edition

This is a welcome addition to the Oxford Handbook Series. As usual in this series the advice is down to earth and very practical. The book is written by a team of contributors from the John Radcliffe Hospital in Oxford, although two are now consultant cardiologists in Southampton. In addition to the consultants the team includes specialist registrars and an ICD nurse specialist, some of whom have taken the lead on major sections of the book. One specialist registrar and three consultants have edited the book. This blend of talent and experience, which accurately reflects the range of work undertaken by an ICD/Pacing unit, is essential for such a practical and technical book which is intended to be read by a similarly wide range of professionals.

The illustrations are simple line drawings with a smattering of photographs and reproduced ECGs/electrograms. The timing cycles are consistently drawn and are an invaluable aid to the understanding of pacemaker function. There are good quality X-rays and fluoroscopy screen shots which are helpful to those who will use this book as a practical guide to providing a pacing/ICD service. The subject of the book is largely technical and is not concerned with the evidence base from which stem the recommendations with regard to indications or pacing system choice, although a little history is given, largely to add perspective rather than to inform a discussion on the merits of device selection for specific indications.

The content of this book concerns the whole range of rhythm management devices: pacemakers, implantable cardioverter defibrillators (ICDs), cardiac resynchronization therapy (CRT), usually with bi-ventricular (Bi-V) pacing), and combinations such as dual chamber or Bi-V ICDs (CRT-D). Implantable loop recorders (ILRs) are also covered in detail, which is relatively unusual but very welcome in a book of this sort.

There are many guidelines which have been published to assist the choice of device indication, selection of pacing mode, type of follow up, level of competence of staff, etc. Of these, the most comprehensive guidelines with the widest geographical authority are those that are constructed and published by the European Society of Cardiology, the American Heart Association, and the American College of Cardiology. Several agreed guidelines from all three of these professional societies are available in this area and it is these guidelines on which the authors most rely. This is fitting for a book which is designed to be valuable at an international level.

The utilization of implantable devices differs greatly from country to country and from district to district. Nothing could illustrate this better than the latest Eucomed figures for Europe (international comparisons) and the results of the *Pacemakers and Implantable Defibrillators: A Two Year National Survey for 2003 and 2004* carried out in the UK by the Network Devices Survey Group. The European data demonstrate that utilization of these implantable therapies is consistently far less than in the USA, and that there is very wide variation in the use of pacemakers, ICDs, and CRT devices which is not closely related to national wealth. The UK information, which

is now being collected on a regular basis, shows that modest national implantation targets are not generally being met, and that there is enormous variability between device utilization across the country, which probably reflects the concentration of trained electrophysiologists in the various health districts. The so-called 'postcode lottery' in this case is not directly related to finance but is probably due to a shortage of trained medical, nursing, and physiology staff. This book will provide the information base to assist the training of staff in these areas and as such it is most welcome.

This is a concise and helpful practical handbook for those charged with providing or contributing to a pacing, ICD, and ILR service. The origin of the book predominantly from a single centre and its reliance on accepted international guidelines give the book a consistent uniformity and an undisputed authority. The authors should be congratulated.

Professor John Camm
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UK

# Preface to the second edition

The development of implantable cardiac device technology and the rapid expansion in implant procedures has continued at pace since the first edition of this handbook. Implantation and management of pacemakers and complex devices are now well established outside tertiary centres, and new technologies such as injectable loop recorders and subcutaneous defibrillators have become familiar tools. Increasingly, even the most complex procedures can be done as day cases, and the patient experience has been further enhanced by greater use of home monitoring.

This second edition of the handbook has been extensively revised to reflect the changes in implantable cardiac devices over the last decade. It remains suited to cardiology trainees in electrophysiology and devices, as well as the growing cadre of specialist cardiac physiologists who perform much of the frontline care for device patients.

JOMO
JT
PL
ARJM
TB
October 2018

# Preface to the first edition

From the earliest days of pacemakers there has been a rapid advance in both the technology and ease of implantation of cardiac rhythm devices. Indications have expanded, implant numbers have soared, and device therapy now encompasses treatment of tachyarrhythmias with ICDs and heart failure with CRT. Device management is no longer the preserve of specialist tertiary centres.

This handbook is not designed to be a fully comprehensive manual of all current pacemakers and defibrillators; rather it is a practical book that explains concepts, principles, and a systematic approach to implantation, programming, and follow-up. It is as ideally suited to the cardiology trainee undergoing training on implantation as to the technician assisting at implantation and follow-up. It will also act as an easily accessible reference in times of need. Incorporating hints and tips from experts in the field, the familiar Oxford Handbook style, and clear diagrams and illustrations, we expect that this guide will become the standard for helping with implantation, follow-up, and troubleshooting for most implantable device procedures.

JT
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April 2007

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# Useful websites

American Heart Association: *www.heart.org*
American College of Cardiology: *www.acc.org*
Arrhythmia Alliance: *www.arrhythymiaalliance.org.uk*
Biotronik: *www.biotronik.com*
British Cardiovascular Society: *www.bcs.com*
British Heart Foundation: *www.bhf.org.uk*
Driver and Vehicle Licensing Agency: *www.dvla.gov.uk*
European Society of Cardiology: *www.escardio.org*
Boston Scientific: *www.bostonscientific.com*
Heart Rhythm Society: *www.hrsonline.org*
British Heart Rhythm Society: *www.bhrs.com*
Medtronic: *www.medtronic.com*
National Institute for Clinical Excellence: *www.nice.org.uk*
LivaNova (formerly Sorin): *www.livanova.com*
Abbott (St Jude Medical): *www.sjm.com*

# Symbols and abbreviations

| | |
|---|---|
| ABP | atrial blanking period |
| AEI | atrial escape interval |
| AF | atrial fibrillation |
| AP | anteroposterior |
| ARP | atrial refractory period |
| AS | atrial sensing |
| ASD | atrial septal defect |
| ATP | anti-tachycardia pacing |
| AV | atrioventricular |
| AVI | atrioventricular interval |
| Bi-V | biventricular |
| BOL | beginning of life |
| CL | cycle length |
| CO | cardiac output |
| CPR | cardiopulmonary resuscitation |
| CRT | cardiac resynchronization therapy |
| CRT-D | cardiac resynchronization therapy defibrillator |
| CRT-P | cardiac resynchronization therapy pacemaker |
| CS | coronary sinus |
| CW | continuous wave |
| DFT | defibrillation threshold |
| ECG | electrocardiogram |
| EDS | electrosurgical-dissection sheaths |
| EF | ejection fraction |
| EGM | electrogram |
| EMI | electromagnetic interference |
| EOL | end of battery life |
| EPS | electrophysiological study |
| ERI | elective replacement indicator |
| FVT | fast ventricular tachycardia |
| HVX | high voltage |
| ICD | implantable cardioverter defibrillator |
| IEGM | intracardiac electrogram |
| ILR | implantable loop recorder |
| LAFB | left anterior fascicular block |
| LAO | left anterior oblique |
| LBBB | left bundle branch block |

| | |
|---|---|
| LPFB | left posterior fascicular block |
| LRI | lower rate interval |
| LRL | lower rate limit |
| LVEF | left ventricular ejection fraction |
| MAP | mean arterial pressure |
| MD | morphology discriminator |
| MI | myocardial infarction |
| MTR | maximum tracking rate |
| NYHA | New York Heart Association |
| PAVB | post-atrial ventricular blanking |
| pAVI | paced atrioventricular interval |
| PCI | percutaneous coronary intervention |
| PFO | patent foramen ovale |
| PMT | pacemaker-mediated tachycardia |
| PSA | pacing system analyser |
| PVAB | post-ventricular atrial blanking period |
| PVARP | post-ventricular atrial refractory period |
| PVS | premature ventricular stimulation |
| QOL | quality of life |
| RAO | right anterior oblique |
| RBBB | right bundle branch block |
| RDR | rate drop response |
| SAN | sinoatrial node |
| SCD | sudden cardiac death |
| SVT | supraventricular tachycardia |
| TARP | total atrial refractory period |
| TDI | tissue Doppler imaging |
| TOE | transoesophageal echocardiography |
| TV | tricuspid valve |
| URI | upper rate interval |
| URL | upper rate limit |
| VBP | ventricular blanking period |
| VF | ventricular fibrillation |
| VRP | ventricular refractory period |
| VS | ventricular sensing |
| VSD | ventricular septal defect |
| VSP | ventricular safety pacing |
| VT | ventricular tachycardia |
| VTC | vector timing and correlation |
| WPW | Wolff-Parkinson-White |

# Chapter 1

# Pacemaker principles

**James Gamble and Paul Leeson**

# History of devices

## Pacemakers

The first artificial pacemaker was an external device designed and built in 1950 by John Hopps. The first totally internal device was implanted into a patient in 1958 by Elmqvist and Senning from Sweden. The patient, Arne Larson, suffered with Stokes-Adams attacks following a viral myocarditis, leading to complete heart block. His first device lasted only a few hours before the battery leaked acid into the epoxy casing. The second device lasted for six weeks. Mr Larson eventually died at the age of 86 having received a total of 23 device implants.

Initial internal devices used zinc-mercury batteries, which were prone to leaking. They soon improved, with changes to the original design including welded connections between the inner and outer cans and improved double wrap separators. In the late 1970s lithium batteries became standard and resulted in greatly extended battery life.

The first systems relied on surgically placed epicardial leads. The first endocardial systems were implanted in the 1960s. Initial devices were 'fixed rate'. 'Demand' pacemakers were also introduced in the mid-1960s. During subsequent years there were advances in lead and device design, leading to smaller pacemakers with longer battery life and with more advanced diagnostic features.

## Implantable defibrillators

Despite a large amount of scepticism (even from within the cardiology community), the first implantable cardioverter defibrillator (ICD) was developed by Michel Mirowski's team and implanted in 1980. Initial systems were large and required a thoracotomy with epicardial electrode positioning. The device was implanted in the abdominal wall. Mirroring the advances in pacemakers, advances in electronics, battery, and lead technology resulted in the first transvenous system implanted prepectorally in 1995.

## Cardiac resynchronization therapy

The interest in pacing the left ventricle (LV) for haemodynamic reasons began in the early 1990s. Following on from initial systems using epicardial electrodes implanted surgically, transvenous systems pacing the LV via the coronary sinus transformed this type of therapy. Initially a Y-connector to the right ventricle (RV) port of a standard dual chamber pacemaker was used. This approach was followed by devices with a dedicated LV port, allowing for separate timings and outputs to the RV and LV channels.

## Timeline of devices

| | |
|---|---|
| 1957 | First transistorized, battery-powered wearable pacemaker |
| 1960 | First totally implanted system |
| 1962 | First endocardial pacing lead |
| 1969 | First 'demand' pacemaker |
| 1975 | Introduction of the lithium iodine battery |
| 1980 | First ICD inserted |
| 1988 | First rate-responsive pacemaker |
| 1994 | First demonstration of potential benefits of multisite pacing |
| 1995 | First prepectoral ICD |
| 2001 | First combined cardiac resynchronization therapy (CRT) and ICD device (CRT-D) |
| 2001 | First devices with wireless telemetry and remote monitoring |
| 2005 | First totally subcutaneous defibrillator |
| 2009 | First quadripolar left ventricular lead |
| 2011 | First MRI-conditional pacemakers |
| 2013 | First leadless pacemaker |
| 2013 | First injectable miniaturized loop recorder |
| 2014 | First leadless CRT device |

# Anatomy of the conducting system

The conduction system of the heart (Figure 1.1) generates coordinated waves of electrical activity that pass sequentially through the atria and ventricles. The system is required to have an intrinsic ability to generate impulses and direct them through the myocardium. Impulses are generated in the sinoatrial node (SAN), travel in the longitudinal direction of atrial muscle fibres, and then converge on the atrioventricular (AV) node. From the AV node they are funnelled through the His bundle and spread out through the ventricles via the branch bundles. They are delivered to the myocardium by the Purkinje fibres.

**Sinoatrial node** The SAN is a distinct structure lying within the epicardium at the junction of the superior vena cava (SVC) and right atrium. It is composed of P cells (which have an intrinsic ability to initiate impulses) and transitional cells (which morphologically lie between P cells and atrial myocardium) held within a collagen framework. Conduction fibres extend through the node into the atrium. The node has an autonomic nervous supply and its own arterial branch (the sinoatrial nodal artery) that arises from the right coronary artery in around 55% of people and the left circumflex in 45%.

**Atrioventricular node** The AV node is a less clearly defined subendocardial structure lying within the atrial septum. Anatomically, it is located at the apex of *Koch*, the borders of which are the coronary sinus os, the tendon of *Todaro*, and the septal leaflet of the tricuspid valve. It

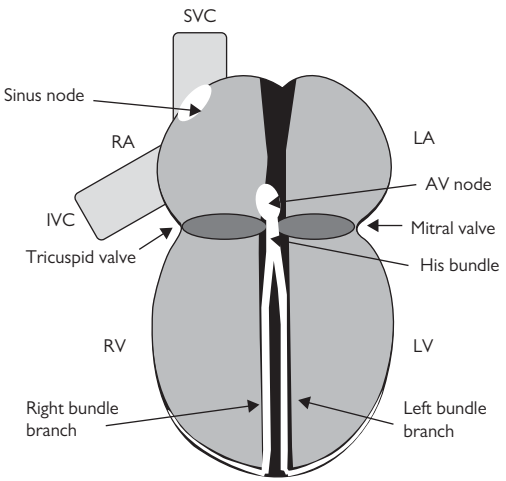


Fig. 1.1 Anatomy of conducting system.

is more loosely composed than the sinus node and contains a variety of atrial, transitional, P, and conduction cells within a collagen network. It has an autonomic nervous supply and its own arterial branch (the AV nodal artery) that arises from the right coronary artery in around 90% of people (left circumflex in the remainder).

**His bundle** The His bundle joins the AV node to the bundle branches and lies within the membranous septum. It is a tubular bundle of parallel conduction fibres within a collagenous framework. It has minimal nervous supply but arterial supply from the AV nodal artery and septal branches of the left anterior descending artery.

**Bundle branches** The bundle branches extend from the His bundle through the ventricles. There is significant interindividual variation in the arrangement of these ventricular conduction fibres. However, there is classically a right bundle that extends down the right side of the interventricular septum and a large left bundle that spreads through the LV as two or three distinct tracts. The bundle branches end in the Purkinje network that delivers the electrical impulses to the myocardium. There is minimal autonomic supply but extensive blood supply from all coronary arteries.

# Conduction system physiology

The physiology of the conduction system determines the impulse generation and transfer of the electrical activation throughout the myocardium.

## Sinoatrial node and atrium

The SAN has significant intrinsic impulse generation capabilities (automaticity) and is the main pacemaker in normal circumstances. The SAN cells generate slow (calcium-driven) action potentials that are then transferred rapidly across the atria via fast (sodium-driven) action potentials. Because of the position of the node the wave of depolarization tends to travel through both atria from superior to inferior.

## Atrioventricular node

The AV node is physiologically similar to the SAN, generating slow (calcium-driven) action potentials. Some people have two distinct pathways, or areas, within the node (slow and fast) that are relevant to the generation of supraventricular arrhythmias. The position of the node between atria and ventricles allows it to control passage of electrical impulses to the ventricle. The node delays transfer to allow complete atrial emptying before ventricular contraction and acts as a limit on the rate of ventricular activation in, for example, atrial fibrillation. The node also has some intrinsic automaticity that allows impulse generation.

## His bundle and bundle branches

The His bundle has distinct longitudinal fibres that direct electrical impulses to the ventricles. The bundle branches are direct continuations of these fibres. The *His-Purkinje* system facilitates rapid global depolarization through the ventricles, typically producing a narrow QRS complex on the surface electrocardiogram (ECG).

## Anatomy of the venous system

Venous access for permanent pacing is usually via the subclavian, cephalic, or axillary veins and for temporary pacing via the subclavian, internal jugular, or femoral veins. See Figure 1.2.

**Cephalic vein** This is a superficial vein starting from the dorsal venous network of the hand that winds up the radial side of the forearm, over the elbow to the lateral border of biceps. It then passes between the pectoralis major and deltoid muscles in the *delto-pectoral groove* (the access site for pacing). The cephalic vein then joins the axillary vein just below the clavicle.

**Axillary vein** This is an upper limb deep vein starting as a continuation of the basilic vein at the lower border of teres major. It joins the cephalic vein to form the subclavian vein at the lateral border of the first rib after the cephalic vein.

**Subclavian vein** This extends to the sternal end of the clavicle where it joins the internal jugular to form the innominate vein. It lies in a groove on the surface of the first rib and pleura where it is accessed for pacing. It has valves around 2 cm from its end.

**Internal jugular vein** This originates at the base of the skull and drains vertically, lateral to the internal carotid artery and common carotid artery, before joining the subclavian veins to form the innominate veins. Access is usually in the triangle formed by sternal and clavicular heads of sternocleidomastoid.

**Innominate vein** The right vein is short and passes directly downwards to join the left innominate (a longer vessel passing obliquely across the chest) below the first rib by the right side of the sternum. They join to form the superior vena cava.

**Superior vena cava** This empties into the right atrium. A *persistent left superior vena cava* is sometimes found. In this situation the venous drainage on the right is normal via a superior vena cava-like structure to the right atrium but there is a separate left-sided drainage, usually to the coronary sinus.

**Femoral vein** The femoral vein accompanies the femoral artery through the upper two-thirds of the thigh. By the inguinal ligament it lies medial to the artery. The vein continues as the external iliac at the inguinal ligament and joins the hypogastric vein to form the common iliac vein.

**Inferior vena cava** The inferior vena cava is formed by the union of the left and right common iliac veins usually around the level of the fifth lumbar vertebra. It travels upwards and enters the inferior aspect of the right atrium.

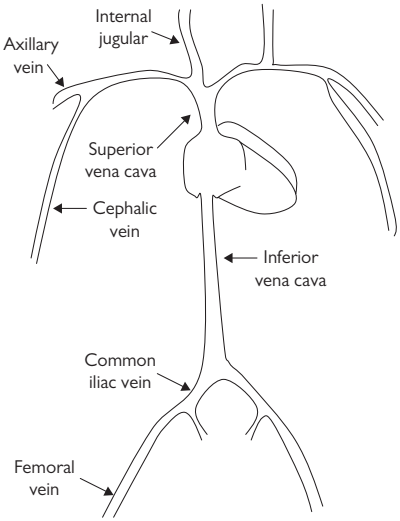


Fig. 1.2 Anatomy of the venous system.

# Pacemaker components

The pacemaker consists of a battery and programmed circuitry encased within a metal box that includes external lead connectors.

## Battery

The standard battery since the 1970s has been the lithium iodide battery. This battery usually has a median life span of around ten years, although this is affected by the frequency and amplitude of pulse generation. The anode produces lithium ions, and electrons are released, whereas the cathode collects electrons and produces iodide ions. In the centre of the battery these ions combine to form lithium iodide. As lithium iodide builds up it increases internal battery resistance. Eventually the middle layer becomes very thick and the cathode material is depleted, producing the characteristic high-battery impedance that indicates end of battery life (EOL).

Modern devices increasingly use variants of this battery chemistry.

## Circuitry

The circuitry defines what each pacemaker can do. Pacemakers are individually designed by the manufacturer to incorporate the necessary pacing and sensing algorithms, as well as a clock and data acquisition functions. The circuitry includes capacitors to generate the impulses and telemetry functions to allow interrogation. A magnet-responsive switch is included for activation of 'magnet mode'. Most pacemakers also have rate response sensors (➔ p. 16).

## Case and lead connectors

The generator case is made of titanium and welded together around the battery and circuitry to ensure it is air- and watertight. The connectors are epoxy plastic and fixed on the outside of the case. The details of the pacing device and manufacturer are usually engraved on the outside of the case. The lead connectors are also usually labelled to ensure they are connected correctly.

## MRI-conditional devices

It is estimated that two-thirds of people will develop an indication for an MRI scan at some point in their lifetime; this has prompted development of devices that allow MRI scanning. This requires component changes (i.e. to the reed switch) and extensive testing. The first MRI-conditional pacemakers went on the market in 2011 and are rapidly becoming the standard of care, with ICDs and CRT devices following over the next five years. It is vital that instructions are followed—conditionality depends on magnet strength, scanning area, energy, and duration. The device must be temporarily programmed into an MRI mode (asynchronous pacing with therapies, if present, switched off). The whole system must be conditional, that is, no mix of manufacturer between generator and leads, and no leftover hardware from previous systems. Close liaison with the radiology department is recommended in order to develop patient pathways. Further information is available on www.mrisafety.com.

### Battery indicators

- EOL: end of life (basic pacemaker functions no longer work).
- ERI: elective replacement indicator (the battery voltage is depleted and close to EOL. Generator replacement should be performed within 2–3 months).

Pacemakers may exhibit diagnostic ERI characteristics when the battery is nearly depleted. These include:

- A fixed decrease in the magnet rate (the rate the pacemaker switches to when a magnet is held over it).
- An increase in the pulse width to compensate for the lower voltage output.
- Change to a simpler pacing mode, for example DDDR to VVI or VOO to reduce battery current drain.

### How to increase battery life

- Minimize the amount of pacing required.
- Use pulses with a smaller voltage amplitude.
- Use pulses with a shorter duration/pulse width.
- Use automated capture functions (➔ p. 120).

# Pacing leads

Pacing leads are flexible insulated wires. They have two ends (the *electrode tip* and *connector pin*) joined by the *conductor*, which is surrounded by *insulation*. They also have some form of *fixation* mechanism at the tip.

## Electrode

The electrode tip usually has an irregular surface to maximize surface area (and optimize polarization for pacing) while maintaining a small radius (to increase current density for pacing). The tips also need to be biologically inert and not degrade. They may be coated with microspheres or metallic meshes to create these characteristics. A common structure is to have a combination of titanium and platinum. Carbon elements can also be used. *Steroid eluting tips* incorporate a steroid, often impregnated in a silicone core. The steroid elutes from the tip and reduces inflammation, stabilizing and improving pacing thresholds over time. Single-pass VDD (➔ p. 24) leads, which are standard ventricular leads with two additional ring electrodes more proximally on the lead in the right atrium allowing for atrial sensing (but not pacing), are available.

## Fixation

There are two types of fixation—active and passive. Passive leads usually have small *tines* at the end (similar to soft 'fish hooks') that hold in the myocardial trabeculae. Active fixation leads have a screw mechanism in the end. These may be a fixed screw that requires the whole lead to be screwed in, but more commonly the screw is advanced from the tip by twisting of a central wire. This also simplifies screw retraction if a lead explant is required.

## Conductor and insulation

The conductor needs to be flexible, strong, and to have minimal current loss along its length. Conductors usually consist of multiple nickel-alloy wires, wound together. Unipolar wires have a single conductor and bipolar leads have a coaxial (with the distal electrode in the centre and the more proximal electrode wrapped around the outside) or a side-by-side design. The insulation is usually silicone rubber or a type of polyurethane.

## Connectors

The connector is a metallic pin, with one or two separate junctions depending on whether the lead is uni- or bipolar. Around the connector there are sealing rings to ensure it fits snugly into the generator head. There is an internationally agreed connector design to allow leads from one manufacturer to be fitted to a generator from another. This is known as IS-1. Originally, however, there was significant variation in design between manufacturers. Problems can still occasionally occur during generator replacement of older systems. Prior knowledge of the implanted leads is required to ensure the correct adaptor is available. This is particularly relevant for more complex ICD and CRT devices where several different lead connection designs are in current use.

## Types of leads

*Unipolar pacing leads*

*Unipolar pacing leads* have a single wire core and electrode tip. The pacemaker generator (the 'can') acts as the other electrode. The pacing and sensing circuit therefore travels through the body from the can to the tip of the pacing lead and then back to the pacemaker can through the lead (or the other way around, depending upon the programmed polarities of the electrode and can). As the current travels through the body, the surface ECG records a large pacing spike. Unipolar leads are no longer routinely implanted, although devices can be programmed to use a bipolar lead in a unipolar configuration.

*Bipolar pacing leads*

See Figure 1.3.

In a *bipolar pacing lead* there are two inner wire cores, separated by insulation, with two separate electrodes—one at the tip of the lead (tip electrode) and the other usually a few millimetres behind it (ring electrode). The pacing and sensing circuit travels down one inner wire from the can to the tip, crosses a few millimetres of myocardium to the ring electrode, and travels back to the can via the other inner wire. The surface ECG registers a much smaller pacing spike.

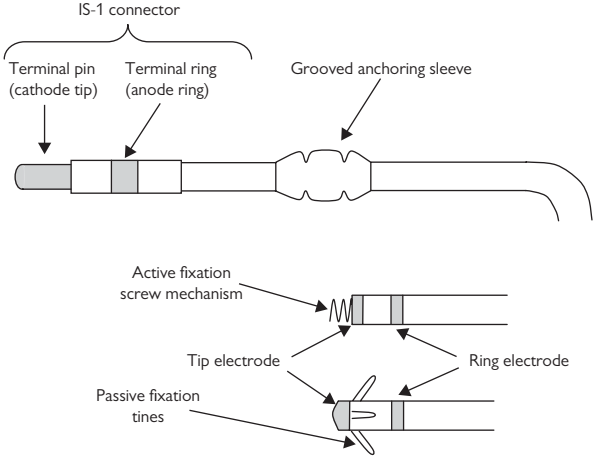


**Fig. 1.3** Pacing lead components of bipolar leads.

# Sensing

A pacemaker does not sense the surface ECG. It senses the potential difference between two electrodes: usually the *distal tip* and the *proximal ring electrode* in a bipolar lead or the *distal tip* and *pacemaker can* in a unipolar lead. The voltage difference produces an *intracardiac electrogram* (IEGM or EGM).

The timing of the EGM represents local depolarization at the tip of the pacing electrode. Typically, the right ventricular lead tip is positioned at the apex of the RV, which may be some distance from the area of earliest ventricular depolarization (distal right bundle branch). Therefore the right ventricular EGM occurs a short time after the onset of the QRS complex on the surface ECG.

## Pacing sensitivity

Sensitivity can be programmed. The sensitivity value represents the minimum local EGM amplitude that is registered as a sensed event. Any EGM component smaller than the programmed sensitivity is ignored.

- The ideal sensitivity will result in appropriate detection of intrinsic events (atrial or ventricular depolarization) with a large safety margin in case the amplitude of the local EGM decreases, for example ectopic beats, or, in the atrium, a change from sinus rhythm to atrial fibrillation (AF).
- Setting the sensitivity value too high may result in *undersensing* of atrial or ventricular activity, often leading to inappropriate pacing or failure to track (➔ p. 148).
- If the pacemaker is oversensitive then spurious electrical activation may result in inappropriate inhibition or tracking. The most common scenario is oversensing of ventricular repolarization (the T-wave component of the local electrogram) and counting this as another ventricular depolarization (➔ p. 141).

### Confusion over sensitivity

See Figure 1.4.

People easily become confused over what it means to *increase* or *decrease* sensitivity. Even if the operator understands, an instruction to an assistant to 'increase sensitivity' can lead to mistakes. This is because an *increase* in the 'value on the dial' of the pacemaker leads to a *decrease* in the number of events being sensed. If you want to make a pacemaker *less sensitive* the implication is that you want it to sense fewer events. However, this is achieved by an *increase* in the *sensitivity* (the 'value on the pacemaker dial') as this *increases* the minimum voltage level required for an event to be a sensed. *Sensitivity* is quantitative and defines the voltage level at which events start to be sensed. *Sensitivity in* a pacemaker is qualitative and to make it less sensitive you increase the sensitivity.

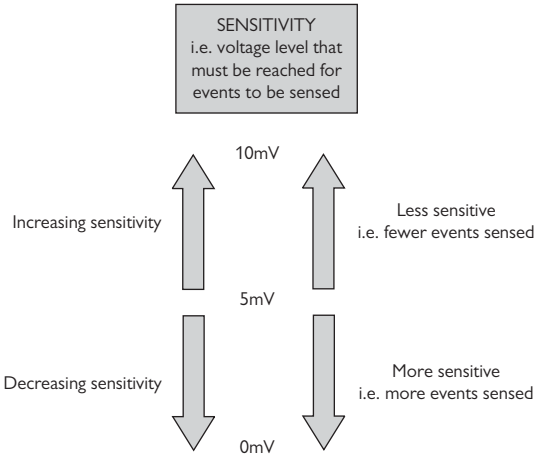


**Fig. 1.4** Sensitivity.

# Pacing

The pacemaker generates a pulse at a specific voltage for a defined period of time (pulse duration/pulse width) by a capacitor discharge. The capacitor is charged from the battery and, as battery output is generally around 2.8 V, higher pacing stimuli require simultaneous charging and discharge of two or three parallel capacitors (thereby shortening battery life).

## Threshold

- The *pacing threshold* (Figure 1.5) is expressed in terms of the voltage and represents the smallest output voltage required to initiate a depolarization wavefront. The pulse duration is fixed (e.g. at 0.5 ms) and the voltage is reduced stepwise to determine the minimum voltage required for capture.
- In some older devices voltage output was fixed (e.g. at 1 mV) and the pulse duration was varied in order to determine the shortest pulse duration required for capture.
- By recording the minimum stimulus strength required to capture heart muscle at a number of different pulse durations, it is possible to construct a *strength-duration curve* (Figure 1.6).
- For practical purposes the *rheobase* is the minimum stimulus strength at the longest programmable pulse duration that will produce a response. The true rheobase is the voltage to which the strength-duration curve asymptotes, that is, plateaus.
- The *chronaxie* is the shortest pulse duration that produces a response when the stimulus strength (voltage) is set to exactly 2 × the *rheobase*.
- The most efficient energy delivery is when the pulse duration is equal to the *chronaxie* value. However, an adequate safety margin is required. It is conventional to programme pacemakers to 2 × the voltage threshold at pulse duration of 0.4 or 0.5 ms.

### Pacing threshold evolution

During the first 2–4 weeks after pacing lead insertion, inflammation and oedema result in a rise in pacing threshold. This gradually resolves, leaving a small fibrous capsule, and the threshold decreases, although it may not return to the value at implant. Steroid eluting leads reduce the amount of inflammation and fibrosis and minimize these changes.

### High impedance leads

High impedance leads have a small electrode surface area in contact with the myocardium and a greater current density, resulting in more favourable thresholds. Higher impedances also result in decreased current drain and longer battery life.

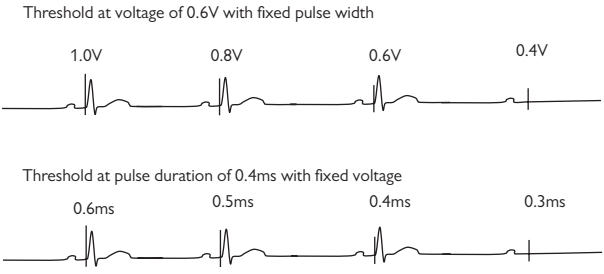


**Fig. 1.5** Ventricular pacing threshold. Note capture of ventricle is lost as the voltage (or pulse duration) is reduced.

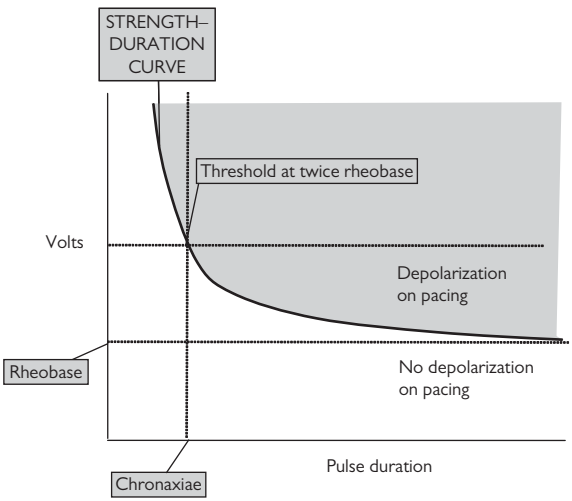


**Fig. 1.6** Strength-duration curve.

# Pacing mode nomenclature

The pacing mode is described by an alphabetic code that can have up to five letters, for example VVI, AAIR, DDO, DDDR.

First letter refers to which chambers are *paced*.

- A, atrium.
- V, ventricle.
- D, dual, that is, both atrium and ventricle.

Second letter refers to which chambers are *sensed*.

- A, atrium.
- V, ventricle.
- D, dual, that is, both atrium and ventricle.
- O, no sensing.

Third letter refers to the *action* taken when an event is *sensed*.

- I, inhibition.
- T, triggered, that is, pacing.
- D, dual, that is, inhibition and/or triggered.
- O, no action.

Fourth letter if present, is always R and means *rate response*.

Fifth letter if present, indicates *multisite pacing*.

- A, atrium.
- V, ventricle.
- D, dual, that is, both atrium and ventricle or ventricles.

Before implantation, a single chamber rate-responsive device is referred to as SR, but once implanted the S is changed to A or V depending on the chamber paced and sensed. Similarly, a dual chamber rate-responsive device is referred to as DR before implantation.