Cardiac Pacing for the Clinician
Second Edition

Edited by

Fred M. Kusumoto, M.D.
Associate Professor of Medicine
Director Electrophysiology and Pacing Service
Division of Cardiovascular Diseases
Department of Medicine
Mayo Clinic
Jacksonville, Florida

and

Nora F. Goldschlager, M.D.
Professor of Clinical Medicine
Co-Director Cardiology Division
Director, Pacemaker Clinic
ECG Laboratory and Coronary Care Unit
San Francisco General Hospital
University of California San Francisco School of Medicine
San Francisco, California

Springer
To my wife, Laura, and my children, Miya, Hana, and Aya
Fred Kusumoto

To my husband, Arnie, and my children, Nina and Hilary
Nora Goldschlager
As early as the late 1700s, Physicians speculated that electrical current could be used to stimulate the heart. In 1882, von Ziemssen used electrical current to directly stimulate the heart of a woman whose anterior chest wall had been removed after resection of a chest tumor. In 1952, Zoll used transthoracic current to pace the heart, and in 1958 the first implantable pacemaker was placed by Ake Senning and Rune Elmquist. At the same time, Furman and Robinson demonstrated the feasibility of transvenous cardiac pacing. In the late 1960s, Mirowski and colleagues pioneered the concept of an implantable device that could be used to defibrillate the heart. Over the last 50 years, implantable cardiac devices have become the primary treatment for bradyarrhythmias and ventricular tachyarrhythmias and have emerged as an important adjunctive therapy for patients with heart failure. It is currently estimated that almost 400,000 pacemakers and defibrillators are implanted annually in the United States.

With exponential expansion of the use of implanted cardiac devices, it has become critical for all physicians to become knowledgeable about them, as they continue to increase in complexity. The second edition of Cardiac Pacing for the Clinician has the same goal as the first edition: To provide a succinct yet comprehensive reference for the implantation and follow-up of implantable cardiac rhythm devices. The book is intended to be a practical guide for the day-to-day management of these increasingly complex devices, and is intended for all physicians caring for patients with devices. We also hope that the emphasis on clinical care will be useful for implanting surgeons, nonphysician medical associates, and clinical members of industry.

The book is divided into four sections. The first section describes pacing leads and pacemaker function. The second section focuses on device implantation. New to this edition is a chapter on implantation of left ventricular leads, used in the biventricular pacing systems intended to treat patients with heart failure. Purposely we have asked two experienced implanters to discuss their personal methods for placing leads in the cardiac venous systems to illustrate the diversity of techniques and “tricks of the trade.” The third section reviews the use of implantable cardiac devices in particular clinical situations. All of the chapters from the first edition have been extensively revised; new to this edition are chapters on device use for patients with atrial fibrillation, heart failure, and syncope, providing further evidence for the expanding indications
for implantable devices. The final section is devoted to device follow-up. It is our belief that the greatest impact for device therapy on patient outcomes is in follow-up. Important topics such as avoiding inappropriate therapies in patients with defibrillators, ensuring optimal and individualized device function for patients, and techniques for minimizing the risks of environmental electromagnetic interference are extensively reviewed.

The last 6 years have shown rapid evolution of cardiac implanted devices that can provide not only therapy but also information on the clinical status of a patient. While beneficial, this increasing complexity also means that all clinicians must be knowledgeable about device function, indications for device use, and device follow-up. We believe that the second edition of Cardiac Pacing for the Clinician will provide the essential clinical information necessary for treating patients with implanted cardiac devices.
Acknowledgments

All books require the concerted effort of a number of people. We wish to thank our contributors for providing their expertise and taking time out of their busy schedules to write chapters for this project. We would like to thank Melissa Ramondetta from Springer Publishing for providing the enthusiasm and resources for making the second edition of Cardiac Pacing for the Clinician a reality. We also would like to thank Dianne Wuori of Springer Publishing for providing editorial and organizational support. Candy Richards provided invaluable administrative support for the second edition. Finally we wish to thank our families for the missed soccer tournaments, late dinners, and “working weekends” that a project like this requires.
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Contributors

_Ejigayehu Abate, MD_
Department of Medicine
Mayo Clinic
Jacksonville, FL 32224

_Amin Al-Ahmad, MD_
Division of Cardiovascular Medicine
Stanford University Medical Center
Stanford, CA

_Nitish Badhwar, MBBS, FACC_
Cardiac Electrophysiology and Arrhythmia Service
Division of Cardiology
University of California
San Francisco, CA

_S. Serge Barold, MD, FRACP, FACP, FACC, FESC, FHRS_
Clinical Professor of Medicine
University of South Florida College of Medicine
And Division of Cardiology
Tampa, FL 33615

_Peter H. Belott, MD_
Sharp Grossmont Hospital
El Cajon, CA

_Jane Chen, MD_
Assistant Professor of Medicine
Washington University School of Medicine
St. Louis, MO

_Louise Cohan_
Electrophysiology and Pacing Service
Division of Cardiovascular Diseases
Department of Medicine
Mayo Clinic
Jacksonville, FL 32224

Robert E. Eckart, MD
Tripler Army Medical Center
Honolulu, Hawaii

Ehab A. Eltahawy, MD
Fellow in Cardiology
Division of Cardiology
Department of Medicine
Health Science Campus
University of Toledo
Toledo, OH

Laurence M. Epstein, MD
Chief, Arrhythmia Service
Brigham and Women’s Hospital
Boston, MA

Westby G. Fisher, MD
Associate Professor, Division of Cardiology
Northwestern University
Evanston, IL

Anne M. Gillis MD, FRCPC
Professor of Medicine
Department of Cardiac Sciences
Health Sciences Centre
Calgary, AB T2N 4N1

Nora F. Goldschlager, MD
Professor of Clinical Medicine
Co-Director Cardiology Division
Director, Pacemaker Clinic
ECG Laboratory and Coronary Care Unit
San Francisco General Hospital
University of California San Francisco Medical School
San Francisco, CA

Blair P. Grubb, MD
Professor of Medicine and Pediatrics
Division of Cardiovascular Medicine
Department of Medicine
Health Science Campus
University of Toledo
Toledo, OH
David L. Hayes, MD
Professor of Medicine
Division of Cardiovascular Diseases
Mayo Clinic College of Medicine
Rochester, MN

Bengt Herweg, MD
Associate Professor of Medicine
Director Cardiac Electrophysiology and Arrhythmia Service
University of South Florida College of Medicine and Tampa General Hospital
Tampa, FL

Richard H. Hongo, MD
ECG Clinic and Coronary Care Unit
San Francisco General Hospital
University of California School of Medicine
San Francisco, CA

Henry H. Hsia, MD
Associate Professor
Division of Cardiovascular Medicine
Stanford University Medical Center
Stanford, CA

Dale M. Isaeff, MD, FACC
Professor of Medicine
Loma Linda University School of Medicine
Director of Pacemaker Surveillance Program
Loma Linda University Medical School
Loma Linda, CA

Jonathan M. Kalman, PhD
Royal Melbourne Hospital
Melbourne, Victoria
Australia

Fred M. Kusumoto, MD
Associate Professor of Medicine
Director, Electrophysiology and Pacing Service
Division of Cardiovascular Diseases
Department of Medicine
Mayo Clinic
Jacksonville, FL 32224

Byron K. Lee, MD
University of California at San Francisco
San Francisco, CA
Paul A. Levine, MD
St. Jude Medical Center
Sylmar, CA

Harry G. Mond, MD, FRACP, FACC, FHRs, FCSANZ
The Royal Melbourne Hospital
Cardiology Department
Melbourne, Victoria 3050
Australia

Brian Olshansky, MD
Professor of Medicine
University of Iowa Hospital
Iowa City, IA 52242

Michael A. Platonov, MD
Electrophysiology Fellow
University of Calgary
Cardiac Sciences
Calgary, Alberta
Canada

Richard S. Sanders
VP Sales and Marketing
Cameron Health, Inc.
San Clemente, CA

Salam Sbaity, MD
University of Iowa Hospitals and Clinics
Department of Internal Medicine
Iowa City, IA

Paul B. Sparks, MD, PhD
Royal Melbourne Hospital
Melbourne, Victoria 3050
Australia

Irene H. Stevenson, MD
Royal Melbourne Hospital
Melbourne, Victoria 3050
Australia

George F. Van Hare, MD
Director of the Pediatric Arrhythmia Center
University of California San Francisco
Stanford University
Pediatric Cardiology
Palo Alto, CA
Paul J. Wang, MD
Stanford University Medical Center
Stanford, CA

Paul C. Xei, MD
Instructor of Medicine
Division of Cardiovascular Medicine
Stanford University Medical Center
Stanford, CA
Section I

Pacing Leads and Modes of Function
Introduction

The cardiac pacemaker lead is a relatively fragile cable of insulated conductor wire implanted into the hostile environment of the human body. Its function is to interface the power source and sophisticated electronics of the pulse generator with the heart. The pacemaker lead plays a critical role in delivering both the output pulse from the pulse generator to the myocardium and the intracardiac electrogram from the myocardium to the sensing circuit of the pulse generator.

A pacemaker lead consists of one or two electrodes at the distal end and a connector at the proximal end for attachment to the pulse generator. In comparison with the marked advances in pulse generator and sensor technology, concomitant advances in pacing leads have occurred relatively slowly. This chapter addresses the cellular electrophysiology and physics of pacing as well as reviews the engineering concepts and clinical application of lead design.

History

By modern standards, the original pacing leads were simple and very unreliable. They were epicardial or epimyocardial and implanted by exposing the heart at thoracotomy. The electrode was part of the uninsulated conductor attached directly to the myocardium. The lead, which emerged from the pulse generator casing, was directly attached to the pulse generator electronics. Failure of either the lead or pulse generator required replacement of both components, thus necessitating a further thoracotomy. It soon became apparent that a separate lead and pulse generator were required as well as a dedicated electrode. With time, the electrode became an epicardial plate or a sharp epimyocardial probe. These early leads frequently developed very high stimulation thresholds and because of continual cardiac and diaphragmatic movements, the lead body was subjected to extraordinary pressures, resulting in conductor fracture or insulation breakdown.

The necessity for repeated thoracotomies to repair or replace pacing leads stimulated an interest in insertion of pacing leads by the transvenous route.
Early transvenous leads were constructed with a Terylene or Teflon core (DuPont, Wilmington, DE) surrounded by a stainless steel ribbon conductor. They were insulated with polyethylene and had no fixation device (Fig. 1.1). The cathode electrode had a very large stimulating surface area up to 100 mm$^2$ and thus had a very low pacing impedance and high current drain. Because there was no provision for a central stylet for lead stiffening, these leads were very difficult to be inserted. During implantation the lead remained floppy, and positioning at the apex of the right ventricle presented a significant challenge. Once implanted, the absence of a fixation device commonly resulted in lead dislodgment. The lead was inserted via the cephalic or external jugular vein and burrowed subcutaneously down the chest wall into the abdomen where the large pulse generator was attached. At the proximal end, the lead connector was prepared at the time of surgery, simply by inserting bare conductor wire into a receiving port of the pulse generator. Insulation was achieved by using rubber O-rings and T-piece around the lead and covering with a plastic cap (Fig. 1.2). Not surprisingly, this connection was very unreliable.

Early pacing leads were either unipolar or bipolar. A bipolar epicardial or epimyocardial system required two leads whereas, a bipolar transvenous system could be achieved using a single lead composed of two parallel insulation tubes containing the anode and cathode conductors. By the late
1970s, most pacing leads were thin and unipolar and the cathode electrode had surface areas in the range of 8–12 mm$^2$. The conductor was a hollow helically coiled wire, which allowed a stylet to pass to the distal end to allow easier positioning in the heart. The earliest fixation device was a wedge, later replaced by tines positioned immediately behind the electrode. With smaller pulse generators, the implant site became the subclavicular region and because no subcutaneous burrowing was required, leads were designed with sealed reliable connectors attached.

**Cellular Electrophysiology and Physics of Pacing**

**Cardiac Depolarization**

As with the normal cardiac conducting system and its propagation of impulses through the His Purkinje network, electrostimulation by an artificial cardiac pacemaker depends on the depolarization of a single or a group of myocyte cell membranes which can then act as pacemaker cells. In order for these cells to depolarize, the electric field of the applied artificial pacemaker stimulus must exceed a threshold voltage. This initiates a complex cascade of ionic currents both in and out of the cell membrane referred to as the action potential. The impulse or wave of depolarization then propagates away from the site of stimulation from cell to cell across gap junctions or intercalated disks, which with normal cells provide very low resistance to depolarization.
In this way, the impulse penetrates all areas of either the atria or ventricles and thus initiates contraction. In pathological situations, such as myocardial ischemia, propagation may slow down because of a rise in resistance in the intercalated disks.

**Lead or System Impedance**

Whenever electricity flows across a circuit, there is a resistance to flow encountered by the electrons. For pacing systems, the resistance is determined by the complex interaction of multiple components. Because some of these components are also characterized by the ability to retain charge or capacitance, the term impedance is preferred. At the time of lead implantation, it is this complicated series of resistance and capacitance factors that are measured and are referred to as system impedance. For a pacing circuit, the system impedance has five basic components: a low, purely resistive conductor impedance, a high cathode electrode impedance, complex polarization effects at the electrode–tissue interface, a low tissue impedance, and the anode electrode impedance (Fig. 1.3).

**Conductor Impedance**

Within a pacemaker lead, the flow of electrons from the connector lead pin to the cathode electrode is relatively unimpeded, because the materials used for modern lead conductors have a very low resistance of the order of 5–50 Ω. This is necessary to prevent wastage in delivery of energy to the cathode. For instance, a very high resistance conductor would act like a radiator generating heat in the conductor; thus, the current or number of electrons eventually delivered to the cathode would be markedly attenuated. Similarly, partial lead fractures or complete lead fractures with the fractured ends in contact with each other will result in very high resistances. In both of these cases, despite the production of an adequate voltage, the current or electron density reaching the distal end of the conductor may be inadequate for the stimulation of the myocardium. This situation can be explained by Ohm’s Law:

\[
\text{Voltage (V)} = \text{Current (J) } \times \text{Resistance (R)}. \]

**Fig. 1.3** Schematic of a bipolar lead illustrating the factors involved in determining system impedance. The arrows denote current flow. Resistance to current flow occurs at the lead conductor (conductor resistance), at the cathode–tissue interface (cathode impedance and polarization), in the myocardium (tissue impedance) and at the anode (anode impedance). The largest contributors to system impedance are the cathode impedance and polarization effects.
For a given voltage, as the resistance rises, current flow is impeded and insufficient electrons reach the cathode and surrounding tissues to allow depolarization.

**Cathode Electrode Impedance**

Despite the necessity for a very low resistance in the conductor, the cathode electrode should be the opposite. A small, high-resistance electrode concentrates the current flow, resulting in a high current density, which allows a low voltage to depolarize the myocytes beyond the electrode–tissue interface. Conversely, a large electrode would have a low resistance and allows considerable current loss.

**Polarization**

There is an impedance factor or capacitance effect at the electrode–tissue interface referred to as polarization. An explanation of polarization requires an understanding of the complex electrochemical events that occur at the electrode–tissue interface. Electricity within metal conductors is ohmic, or simply the flow of electrons. However, in body tissues, current flow is due to the movement of charged molecules such as Cl\(^{-}\) ions. When current flows from the electrode to the tissues, there is a transfer of ohmic energy to ionic energy at the electrode–tissue interface. This involves an intense chemical reaction. The result is flowing away of the negatively charged ions leaving behind an alignment of oppositely charged particles attracted by the emerging electrons (Fig. 1.4).

The capacitance effect of these positively charged ions acts as a deterrent or resistance to ion flow and is the basis of polarization. It explains the change in impedance that occurs during the delivery of the pacing stimulus. The capacitance is zero at the leading edge of the constant-voltage stimulus. During the stimulus, the capacitance increases to reach its maximum at the trailing edge of the stimulus (Fig. 1.5). The capacitance gradually decreases after the stimulus,

![Polarization](image)

**Fig. 1.4** Polarization effect at the electrode–tissue interface. Within the electrode, current flow is due to movement of electrons (e\(^{-}\)). At the electrode–tissue interface, the current flow becomes ionic. The negatively charged ions (Cl\(^{-}\), OH\(^{-}\)) flow into the tissues toward the anode leaving behind oppositely charged particles attracted by the emerging electrons. It is this capacitance effect at the electrode–tissue interface, that is the basis of polarization.
owing to the dissipation of positive ions and the return of the electrode-tissue interface to electrical neutrality. The accumulation of these ions in the myocardium constitutes the afterpotential that is typically recorded following a pacing stimulus.

Like the cathode electrode impedance, the electrochemical polarization effect increases as the geometric electrode surface area is reduced. For this reason, although a small electrode with a high cathode electrode impedance is desirable to allow increased current density, these small electrodes are usually associated with a high polarization effect, which is energy expensive. In addition to electrode size, polarization is also dependent on the time that has elapsed following lead implantation, the electrode materials, the electrode surface structure, the current delivered (increases with low current), the pulse duration (increases with extended pulse duration), the tissue chemistry and the stimulation polarity. The polarization effect can represent 30–40% of the total pacing impedance, but this contribution may be as high as 70% for some smooth surface, small surface area electrodes. For these reasons, polarization effects have become very important in cathode electrode design.

**Tissue Impedance**

After leaving the electrode-tissue interface, current flows toward the anode. The resistance of all tissues between the cathode and anode is termed the tissue impedance. For a bipolar system in which the anode is a ring electrode located within the paced chamber at a short distance from the cathode, the tissue impedance is determined by the endomyocardium with its myocytes and extracellular components including fluids (Fig. 1.6). For a unipolar system, where a section or all of the pulse generator housing serves as the anode, both cardiac and noncardiac tissues contribute to the tissue impedance. In general, most of these tissues have a high concentration of water and electrolytes, resulting in similar low tissue impedance for both unipolar and bipolar pacing systems.
Anode Electrode Impedance
A final consideration is the impedance of the anode electrode. Because of the complex impedance characteristics of polarization, it makes sense that the anode is large so as not to impede current flow in order to complete the electrical circuit. However, in modern lead systems, whether they are bipolar or unipolar, the anode electrode impedance contributes very little to the overall system impedance.

Stimulation Threshold and Strength–Duration Relationships
By definition, the stimulation threshold is the lowest voltage or current necessary to consistently evoke cardiac depolarization outside the refractory period of the heart. By convention the term “consistently” refers to at least five consecutive beats. Although in clinical practice only voltage (volts = V) and pulse duration (milliseconds = ms) are used, nevertheless, a number of derived parameters are often described in lead studies but are of little value in day-to-day management. These include current (milliamperes = mA), energy (microjoules = µJ) and charge (microcoulombs = µC). These parameters are discussed later in this chapter.

The stimulation threshold is measured at lead implantation and should be remeasured at any reoperation whether it is suspected or proven malfunction or routine pulse generator replacement. Modern pulse generators also have algorithms that allow the voltage or pulse duration stimulation thresholds to be determined noninvasively. Depending on the method used to measure the stimulation threshold, the voltage output is generally programmed to at least two times greater than the voltage threshold (2:1 safety margin) or the pulse duration to at least three times greater than the pulse duration threshold (3:1 safety margin). More recently, ventricular voltage threshold stimulation...
algorithms have been introduced that repeatedly measure the ventricular stimulation threshold and then automatically reprogram the voltage output to a value just above this threshold. This has the advantage of allowing even lower voltage outputs to conserve the power source.

A number of factors determine the stimulation threshold at implantation and in the follow-up period. Most of these are discussed in detail later in this chapter. However, apart from the cathode electrode, a relevant factor is the tissue characteristics, including ischemic or fibrous tissue, which results in higher stimulation thresholds. Interestingly, the stimulation threshold of single myocytes has been shown to be dependent on the orientation of the cell to the electric field. A cell that lies parallel to the field has a lower stimulation threshold than one lying perpendicular to the field. Similarly, cathodal (negative) stimulation has a significantly lower stimulation threshold than anodal (positive) stimulation. Another critical factor determining stimulation threshold is the distance of the closest normal myocyte. The stimulation threshold is inversely proportional to the square of the distance to the closest excitable cell. It can be shown that a displacement of >0.5 mm can significantly change the stimulation threshold. It is probably this factor that results in many of the idiopathic high stimulation threshold problems seen within the first 6-month postimplantation. These cases often present as idiopathic high threshold exit block (voltage stimulation thresholds greater than the output of the pulse generator), and are called micro dislodgment or micro displacement.

An additional factor determining the voltage stimulation threshold is the period of time the stimulus, measured in terms of voltage or current, is applied to the myocardium. This period is referred to as the pulse duration or pulse width. For obvious reasons, the shorter the pulse duration the higher the stimulation threshold. This nonlinear exponential relationship between the voltage stimulation threshold and the pulse duration can be represented graphically as the strength–duration curve. Knowledge that the strength–duration curve is nonlinear is crucial in both design and output programming of pacing systems (Fig. 1.7). For modern atrial or ventricular endocardial

![Fig. 1.7 Strength–duration curves. See text for explanation.](image-url)
leads, the voltage or current stimulation threshold remains relatively constant for pulse durations >0.5 ms. In contrast, a pulse duration <0.2 ms results in a marked elevation of the stimulation threshold with the level approximating to infinity as the pulse duration approaches zero. For most electrodes, the most effective pulse duration range for maximum energy efficiency is from 0.25 to 0.6 ms. Because current or voltage stimulation threshold may rise following lead implantation, the acute and chronic strength–duration curves for the same lead will differ. Therefore, strength–duration curves differ between electrode designs as well as implant times. There are two reference points on a strength–duration curve that are important in determining the quality of a pacing electrode. The first is the lowest point on the curve, called the \textit{rheobase}. The rheobase is by definition the lowest voltage or current that results in myocardial depolarization at infinitely long pulse duration. In practice, the rheobase is almost never determined, because it is rare to measure the stimulation threshold at pulse durations >2.0 ms. Consequently the rheobase usually quoted in the literature is one that is extrapolated from a modified strength–duration curve and can be referred to as an \textit{apparent rheobase}.

The other reference point is the pulse duration time, called the \textit{chronaxie}, which is derived from the rheobase. By definition, the chronaxie is the threshold pulse duration at twice the rheobase voltage or current. The chronaxie can also be referred to as \textit{apparent chronaxie} because the rheobase cannot be accurately determined.

There are a number of lessons to be learnt from the inspection of a strength–duration curve. The ideal pulse duration should be greater than the chronaxie time. When a stimulation threshold is measured or recommended, whether it is voltage or pulse duration, the other determinant of the strength–duration curve must also be quoted. Although this appears obvious, it is not uncommon to try to overcome high threshold exit block by increasing the pulse duration. If the voltage output remains less than the rheobase, then no increase of the pulse duration from normal values will be effective for myocardial stimulation.

Another practical application involves the safety margin discussed earlier. Although 2:1 voltage and 3:1 pulse duration safety margins are usually quoted, this should be used in a practical sense only after inspection of the strength–duration curve. For instance, it may be recommended that for a voltage stimulation threshold of 2.0 V at pulse duration of 0.35 ms, the pulse generator be programmed to 2.0 V and the pulse duration 1.0 ms. However, after inspection of the exponential strength–duration curve, it may be wiser to keep the pulse duration at 0.35 ms and program the voltage to 4.0 V.

As mentioned earlier, there are two controversial derived measurements of lead function that are occasionally used for lead studies (Fig 1.7). The first is the threshold \textit{energy}, which is related to current, voltage, and pulse duration by the formula:

\[
\text{Energy (\(\mu\)J)} = \text{Voltage (V)} \times \text{Current (mA)} \times \text{Pulse Duration (PD in ms)}.
\]

Substituting current as in Ohms Law \((I = V/R)\) into the energy formula:

\[
E = V^2 \frac{PD}{R}.
\]

Like voltage and current, a threshold energy strength–duration curve can be created. Referring to the energy formula, it is not surprising that threshold energy
rises with increasing pulse duration. However, with low pulse durations, the exponential rise in voltage threshold results in very marked elevations of energy consumption because the voltage is squared. Knowing this it can be determined that the ideal pacing energy losses occur with a pulse duration of about 0.5 ms.

Because voltage is squared in the energy formula, the voltage safety margin is only 2:1 compared to 3:1 for pulse duration. Similarly, doubling the voltage output of a pulse generator from 1.0 to 2.0 V results in correspondingly smaller energy losses than from 2.5 to 5.0 V. This has important implications when considering pulse generator power source longevity. It is extremely important to measure stimulation thresholds and where possible, lower voltage outputs to 2.5 V or less. However, further energy savings become insignificant with voltage outputs below 1.5 V.

Another derived and equally controversial threshold measurement of electrode function is the charge delivered by the power source of the pulse generator. By definition, this is the current or the number of electrons delivered per unit time and is measured in microcoulombs (µC).

\[
\text{Charge (µC)} = \text{Current (mA)} \times \text{Pulse Duration (ms)}.
\]

Charge threshold is useful in predicting pulse generator power source longevity. This is because a power source is essentially a limited fuel tank of electrons and the rate at which these are used per unit time, which is essentially charge, identifies the time when the tank will become depleted. On the strength–duration curve, the threshold charge, being the product of current and pulse duration increases in a straight line. The continuing fall in charge threshold with reducing pulse duration is important, although this will plateau as the rising current threshold offsets the effect of the short pulse duration.

Why then, is there a controversy about the use of derived threshold parameters for strength–duration curves? On the surface, the use of energy or charge seems reasonable. The major criticism in using energy or charge is that it is not possible to program these parameters directly with current programmable pulse generators. Being derived parameters, the calculations must take into consideration other parameters such as impedance. As stated earlier, the polarization impedance rises during delivery of current; thus, where the value is measured can be critical. It is essential to measure the voltage before energy can be used in a meaningful way, because the voltage used in any energy calculation must be greater than rheobase for cardiac depolarization to occur. It would be easy to accept what appears to be an appropriate energy value, but because of an inappropriate pulse duration and impedance, the programmed voltage or current could be less than rheobase; thus it would fail to pace the heart. In a practical sense, pacemaker physicians and technologists think and program in terms of voltage and pulse duration. Energy and charge are not used in day-to-day management.

Despite the limitations of the derived parameters, they do help in understanding the concepts of safety margins and energy conservation. It is obvious to see that at very low pulse thresholds, the charge is low, but the energy requirements are high because of elevated current and voltage stimulation thresholds. At pulse durations of 0.4–0.6 ms, all threshold parameters appear ideal. At high pulse durations, the voltage and current requirements may be low, but the energy and charge values are unacceptable. A summary of the optimal pacing outputs is provided in Table 1.1.
Besides providing a method for myocardial stimulation, the pacemaker lead is also responsible for retrograde conduction of intrinsic intracardiac signals from the heart back to the pulse generator. The ability of a modern pacing system to adequately sense these signals and respond appropriately depends on both cardiac and pacemaker factors. Cardiac factors determine the quality of these signals, which in turn is dependent on the electrophysiological and anatomical properties of the surrounding myocardium. Once detected, the transfer of an intracardiac signal, without significant attenuation to the pulse generator-sensing circuit is dependent on the shape and materials of the electrode as well as the conductive and insulative properties of the lead. The signal must be amplified and analyzed by the sensing circuitry once it reaches the pulse generator. False or inappropriate signals – caused by extracorporeal interference, skeletal myopotentials and far-field events – must be accurately identified by the sensing circuitry and differentiated from intrinsic cardiac activation.

The foundation of intracardiac sensing involves the ability of the lead system with its cathode and anode to successfully detect spontaneous intracardiac depolarization from within the cardiac chamber it is primarily designed to pace. During Phase 4 or the resting phase of the cardiac action potential, individual myocytes have an electrical gradient of about −90 mV across the cell membrane. As all cardiac cells have a similar gradient; there is, therefore, no potential difference detected between the cathode and anode of a pacing system. The strength–duration curve must be taken into account when determining the optimal type of safety margin programming.

Table 1.1 Optimal programming of pacing outputs.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Optimal range</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Voltage</td>
<td>1.5–2.5 V</td>
<td>Longevity is markedly reduced when the output is greater than 2.5 V. Voltagles less than 1.5 V are not associated with significant increases in longevity.</td>
</tr>
<tr>
<td>Pulse duration</td>
<td>0.4–0.6 ms</td>
<td>Pulse durations of 0.4–0.6 ms correspond with the nadir of the threshold energy strength–duration curve (Fig. 1.7).</td>
</tr>
<tr>
<td>Safety margin</td>
<td>2:1 voltage 3:1 pulse duration</td>
<td>The strength–duration curve must be taken into account when determining the optimal type of safety margin programming.</td>
</tr>
</tbody>
</table>

Sensing

Besides providing a method for myocardial stimulation, the pacemaker lead is also responsible for retrograde conduction of intrinsic intracardiac signals from the heart back to the pulse generator. The ability of a modern pacing system to adequately sense these signals and respond appropriately depends on both cardiac and pacemaker factors. Cardiac factors determine the quality of these signals, which in turn is dependent on the electrophysiological and anatomical properties of the surrounding myocardium. Once detected, the transfer of an intracardiac signal, without significant attenuation to the pulse generator-sensing circuit is dependent on the shape and materials of the electrode as well as the conductive and insulative properties of the lead. The signal must be amplified and analyzed by the sensing circuitry once it reaches the pulse generator. False or inappropriate signals – caused by extracorporeal interference, skeletal myopotentials and far-field events – must be accurately identified by the sensing circuitry and differentiated from intrinsic cardiac activation.

The foundation of intracardiac sensing involves the ability of the lead system with its cathode and anode to successfully detect spontaneous intracardiac depolarization from within the cardiac chamber it is primarily designed to pace. During Phase 4 or the resting phase of the cardiac action potential, individual myocytes have an electrical gradient of about −90 mV across the cell membrane. As all cardiac cells have a similar gradient; there is, therefore, no potential difference detected between the cathode and anode of a pacing lead during this phase and thus signal is neither conducted to, nor registered in the sensing circuit.

As with a pacing wave of depolarization, the spontaneous cardiac wave of depolarization results from a complex and sophisticated movement of ions across individual cell membranes particularly during Phase 0 of the action potential. The result is an electrical event in both atria and ventricles created by a cascade of depolarizing cells. A potential difference is registered depending on the position of an individual depolarizing myocyte relative to the pacing electrode dipole. Thus, a wave of depolarization throughout the chamber involving millions of cells records a significant difference in electrical potential between the anode and cathode of the pacing system. This results
in a biphasic wave, which on the surface electrocardiogram is represented as the P wave for atrial depolarization or the QRS complex for ventricular depolarization.

The shape and voltage of this characteristic wave, which is called the electrogram is dependent on the position of the dipole and the number of cells depolarized. The movement of the wave of depolarization relative to the orientation of the dipole of the electrodes can have a marked effect on the eventual recorded electrogram (Fig. 1.8). For instance, if the inter-electrode dipole is parallel to the wave of depolarization, then the wavefront passes one pole first and then the other, recording the maximum potential with the highest slew rate or voltage change per unit time. The opposite occurs if the electrode dipole is oriented perpendicularly. In this situation, both electrodes record the same potential change simultaneously; thus, there is no relative change in potential between electrodes, despite a normal wave of depolarization in the heart. No electrogram would be recorded in this theoretical situation.

In clinical practice, a generous ventricular electrogram is usually recorded at the time of lead implantation. An atrial or ventricular lead rarely needs to be repositioned, because of failure to sense (Fig. 1.9). The usual cause of a poor voltage ventricular electrogram is loss of ventricular muscle mass such as following infarction. Atrial electrograms are generally smaller than ventricular electrograms, but modern pulse generators can be programmed to high sensitivity to overcome this potential problem.
The pacing lead not only delivers the electrogram to the sensing circuit of the pulse generator, but is also critical in ensuring that the delivered product is not attenuated below a programmed sub-threshold value. For a lead to deliver an electrogram to the sensing circuit it must pass through two impedance barriers placed in parallel (which should not be confused with the system impedance discussed earlier). The first is the \textit{sensing or source impedance}. This is effectively the sensing impedance of the conductor and electrode–tissue interface that presents itself to a cardiac signal. This was very important in early electrode design, because theoretically the smaller the electrode, the higher the sensing impedance. In turn the higher the sensing impedance, the more markedly the electrogram signal is attenuated. The second impedance barrier is the \textit{input amplifier impedance} within the pulse generator. In this case, the higher the resistance value of the amplifier, the superior the delivered signal to the sensing circuit. Because these impedance barriers lie in parallel, they significantly influence each other. This is demonstrated in Fig. 1.10, where the attenuation of the electrogram signal is plotted against the sensing impedance for different input impedances. Early pulse generators had input amplifier impedances of 1 K\(\Omega\). With high sensing impedances, the size of the signal could fall to 20\% of that initially seen at the electrodes. However, once the input amplifier impedance was raised to 15 K\(\Omega\) or greater, the attenuation was only 75\% of the initial value. This allowed the introduction of small cathode electrodes.

Ohm’s law can also explain this signal attenuation. If a ventricular electrogram signal of 10 mV presents itself to a sensing impedance of 5 K\(\Omega\) and a very low input amplifier impedance of 1 K\(\Omega\), then the total impedance (in parallel) is 6 K\(\Omega\).

\[
\text{Current flow (I)} = \frac{\text{Voltage (V)}}{\text{Total impedance (R)}} = \frac{10}{6} = 1.67 \text{ mA}.
\]

The voltage that eventually presents itself to the sensing circuit is thus:

\[
V = I \times \text{Input impedance} = 1.67 \times 1 = 1.67 \text{ mV}.
\]

---

\textbf{Fig. 1.9} Atrial and ventricular electrograms (EGM) as obtained through a Medtronic Pacing System Analyser Model 5311. \textit{Ventricular:} Above: A typical rS configuration with a minor current of injury pattern. Below: The current of injury pattern is more pronounced. \textit{Atrial:} Above: A typical rS pattern. Below: Approximately 150 ms after the atrial electrogram lies a large far-field ventricular electrogram.
This is 17% of the original signal. However, if the input impedance is increased to 15 KΩ, then the final signal voltage will be:

\[
\frac{10 \text{mV}}{(15 + 5 \text{kΩ})} \times 15 \text{KΩ} = 7.5 \text{mV}.
\]

In this case the signal improves to 75% of the original signal. In a practical sense, this was very important, when low input impedance sensing circuits were used with small surface area electrodes. With modern pulse generators, this is not a problem as the attenuation is minimal.

The ventricular electrogram waveform as seen from the apex of the right ventricle is predominantly a negative deflection, although in a majority of cases it is biphasic. The classic pattern is a small initial positive wave followed by a negative deflection and a small positive terminal wave (Fig. 1.9). In about 10% of cases, the electrogram waveform is predominantly positive. Occasionally, a transient current of injury pattern may be seen at the terminal part of the wave. At the time of lead implantation, the amplitude of this deflection and the slew rate are measured by the pacing system analyser. In the ventricle, a bipolar amplitude of >4 mV is recommended for satisfactory sensing. For atrial leads, the amplitude should be >1.5 mV. However, the sensing characteristics of modern pulse generators can usually cater for lower voltages in both chambers. It may be valuable to repeat the measurements in the unipolar configuration as modern pulse generators have programmable sensing polarity.

The **slew rate** is the maximum rate of voltage change (dV/dt) in the electrogram and represents the steepness of the slope over the first derivative or 2-mV voltage excursion. Satisfactory ventricular electrogram slew rates are of the order of 1–4 V/s with the minimum generally accepted being 0.5 V/s. A desirable atrial slew rate is >0.5 V/s.
Lead Polarity

The vast majority of pacing leads implanted today in the atrium and to a lesser extent in the ventricle are bipolar (1). Compared to their unipolar counterparts, early bipolar leads were large, cumbersome and difficult to implant resulting in an early preference for unipolar systems (2). Today, with major technological and engineering advances, bipolar leads are the same as unipolar designs in size and ease of insertion. By definition, all pacemaker electrical circuits are bipolar. To complete the circuit, electrons flow from the cathode to the anode. When applied to transvenous leads, the terms “unipolar” and “bipolar” simply indicate the number of electrodes in contact with the heart (Fig. 1.6). A unipolar lead has only one electrode (the cathode), located at the tip. Current flows from the negatively charged cathode to the heart and returns to the anode (the pulse generator) to complete the circuit. In contrast, a bipolar lead has both electrodes, a short distance from each other at the distal end. The tip electrode is the cathode, and a ring electrode proximal to this, serves as the anode. In reality, the differences between unipolar and bipolar pacing are relatively minor. The principal differences between these two electrode configurations are discussed:

Size. The original bipolar lead designs had two parallel conductors individually encased in a stiff, dual-lumen insulated tube and a very bulky bifurcated connector. They were difficult to implant compared to unipolar models. In comparison modern bipolar pacing leads are as thin as their unipolar counterparts and thus size is no longer an issue.

Stimulation Threshold. Because of the larger anode, unipolar pacing has a marginally lower total system resistance compared to bipolar pacing. Unipolar pacing, therefore, should result in a slightly lower stimulation threshold compared with bipolar pacing. In reality, modern, small, low polarization cathodes play a much more important role in determining overall pacing impedance than the larger anode, resulting in essentially the same stimulation thresholds for both configurations.

Sensing of intracardiac electrograms. With its broad inter-electrode distance, the unipolar system “sees” more of the heart in which to detect a spontaneous intracardiac electrical event and thus it was assumed that unipolar was superior to bipolar sensing. In reality, the modern unipolar and bipolar pacing systems show comparable and usually excellent atrial and ventricular electrogram amplitudes and slew rates which usually exceed the standard limits of the sensing circuit by a comfortable margin.

Far-field sensing. The atrial sense amplifier of a dual chamber (or AAI) pacemaker may demonstrate apparent inappropriate sensing related to small amplitude far-field signals arising from the ventricles (Fig. 1.9). In the presence of high atrial sensitivity settings, such signals may be inappropriately interpreted as atrial depolarizations. Generally, this is more likely to occur if the tip of the atrial lead is positioned near the tricuspid valve. Since all leads sense the dipole between the anode and the cathode, a bipolar atrial lead with closely spaced electrodes is less likely to record far-field electrical signals than a bipolar lead with a wider inter-electrode distance or a unipolar lead (3). Sensed, far-field electrical signals may also result in inappropriate mode switching (4). Thus, bipolar leads have a clear advantage with respect to decreasing the size of far-field cardiac signals.