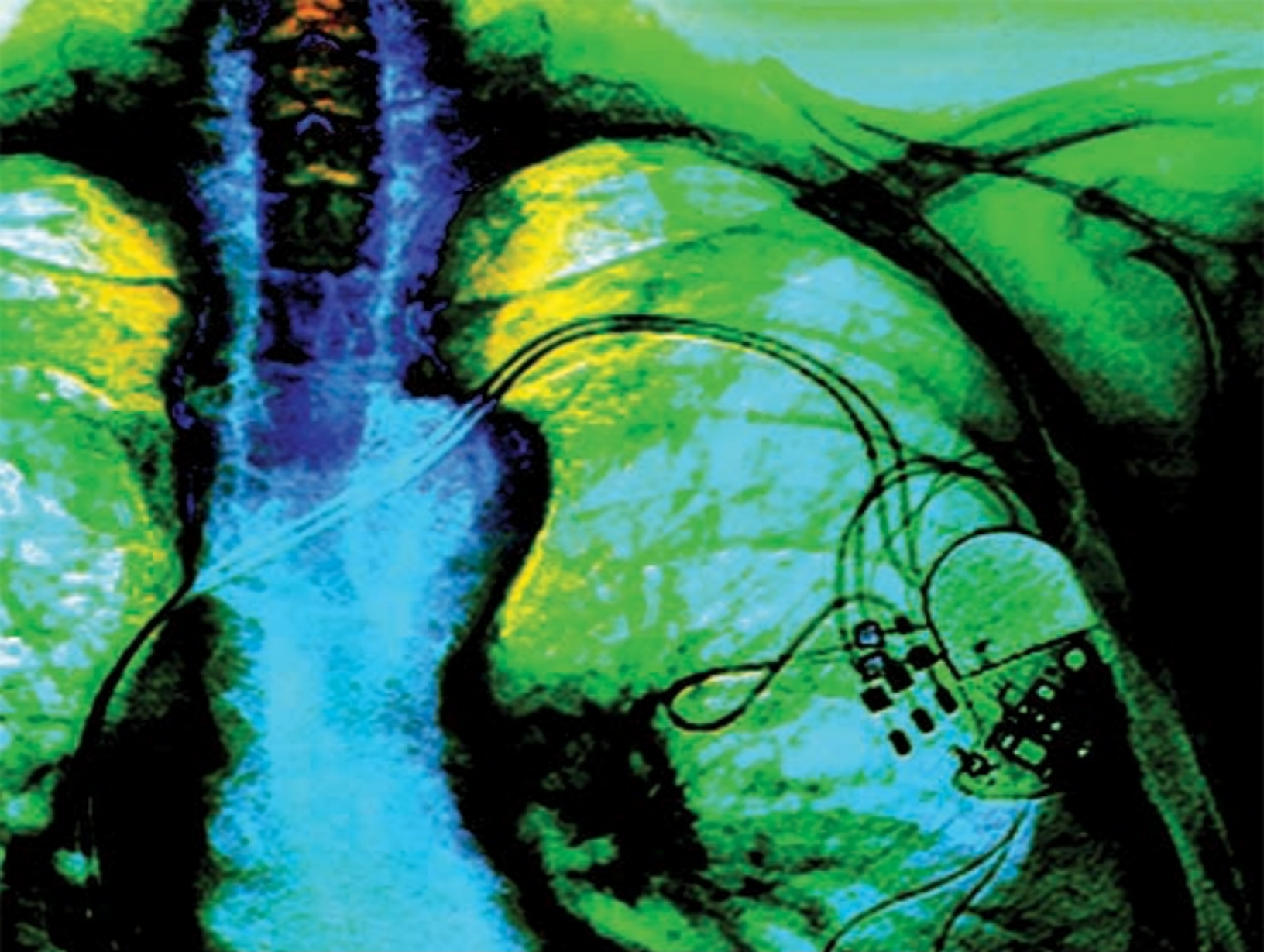




SECOND EDITION

# Cardiac Pacing, Defibrillation and Resynchronization

*A Clinical Approach*

David L. Hayes and Paul A. Friedman

 WILEY-  
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# Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach

SECOND EDITION

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*Dedication*

*For Sharonne, Sarah, Drew, and my parents, Audrey and  
Henry Hayes.*

*DLH*

*To my wife, Vicki, my daughters, Lindsay, Hannah, and  
Maddy, and my parents, Charles and Ety Friedman, for your  
love, support, patience, and encouragement.*

*PAF*

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# ***Preface***

In preparing this edition of *Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach*, our intention remained the same as for the previous edition, that is, to be a text that is uniformly written with sensible, matter-of-fact methods for understanding and caring for patients with permanent pacemakers, implantable cardioverter-defibrillators (ICDs), and, to a much greater extent in this edition, cardiac resynchronization therapy (CRT) devices. Once again, our intent was not to create an encyclopedic text. Instead, we want to provide practical clinical information for those involved in cardiac pacing, defibrillation and CRT. Several excellent multi-authored texts provide encyclopedic information.

Cardiac pacing, cardiac defibrillation, and, more recently CRT, have become fields unto themselves as the technology has proliferated and the devices have rapidly become increasingly more sophisticated. We have witnessed unbelievably rapid advances in the technology of implantable cardiac devices. With the first pacemaker implant in 1958, the first ICD implantation in 1980, and the first biventricular pacing report in 1994, few would have imagined the progress and improvements made in such a relatively short period of time. Having witnessed the continued improvements in pacemakers, ICDs and CRT in recent years, we would not underestimate the potential for future improvements of these devices and can see many opportunities for expansion of these therapies.

This text is meant to help the reader understand the technical capabilities of pacemakers, ICDs and CRT and how to apply this knowledge clinically. Whether the reader is new to these disciplines or sees patients with implantable devices every day, we hope that the information we have included will make clinical encounters easier.

We feel strongly that there is merit in a text written by a small number of contributors. Not to detract from the expertise of the contributors or editors of the excellent multiauthored texts available, limiting the number of authors allows a connection from chapter to chapter and a consistent writing style. Our hope is that this choice will make reading and comprehension easier. We (DLH and PAF) are involved in all of the chapters. However, we need to thank a number of colleagues who have contributed to the preparation of this edition of the text. First, we acknowledge our colleague Dr Margaret (Peg) Lloyd, MD, an author on the first edition of this text. We have built upon her prior contributions in specific chapters and greatly appreciate the foundations she helped lay. Our special thanks to our colleague Samuel J. Asirvatham, MD, who became progressively more involved as this edition moved forward. We genuinely appreciate his expertise, tireless efforts, and counsel. Our friend Dr Charles (Chuck) D. Swerdlow, MD, is an innovator in the field who enriched the chapters on Troubleshooting and Programming with significant contributions. Others who have contributed include Apoor Gami, MD, Jared Bunch, MD, and Niloufar Ta-batabaei, MD - many thanks for your energy and contributions. We have enjoyed long and fruitful collaborations with Michael Glikson, MD, and Paul J. Wang, MD,, and appreciate their contribution to this effort.

The text has also been influenced by and we have been given incredible assistance from friends and colleagues in industry. A special thanks to Paul Levine, MD, who has graciously allowed us to use a number of examples from his personal collection and who remains one of the “giants” in the field. Others who have responded to many questions, and have reviewed sections of text to ensure its technical accuracy; we are grateful for their efforts. They include: Doug Welter and Mario Bradley (Biotronik, Inc.); Jodie Alwin,

Dan Heffron, Tom Ermis, and Jim Gilkerson (Boston Scientific Company); Nancy Magnotto, Gregg Deutsch, Jay Wilcox, Jim Glover, Jeff Gilberg, Nancy Magnotto, Preface and Dave Furland (Medtronic, Inc.); Jim Gerrity and Marcel Limousin (Sorin Group); Leslie Meyer, Daryel Davis, Dan Hecker, and Steve Heinrich (St Jude Medical).

Many others have influenced this project. All our physician and nursing colleagues in the Heart Rhythm Services group at Mayo Clinic Rochester have had either a direct or an indirect influence on portions of this text. Our intention is not to officially represent our entire practice of pacing and electrophysiology with this text. However, given a significant consistency in the way we practice and how we approach patients, we would expect general agreement with the clinical management strategies put forward in this text.

Although the content of this text is patterned after the first edition, we have reorganized some sections, expanded ICD information and added CRT discussions to almost all chapters, to reflect the evolution of device practice. Internet-based remote monitoring has become a reality, “recalls” must be addressed, and new insights have revised how device therapy can prolong life and improve its quality; these important topics are extensively addressed. We have attempted to provide a logical progression from a description of device indications to selection of hardware, implantation, complications, programming, troubleshooting, and follow-up. It is our deepest hope that this effort will enhance the care of patients with arrhythmias.

*David L. Hayes, MD & Paul A. Friedman, MD*

# ***CHAPTER 1***

## ***Clinically Relevant Basics of Pacing and Defibrillation***

***T. Jared Bunch, David L. Hayes, Paul A.  
Friedman***

### **Anatomy and physiology of the cardiac conduction system**

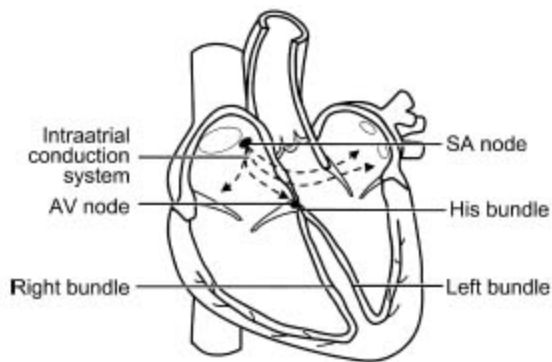
The cardiac conduction system consists of specialized tissue involved in the generation and conduction of electrical impulses throughout the heart. In this book, we review how device therapy can be optimally utilized for various forms of conduction system disturbances, tachyarrhythmias, and for heart failure. Knowledge of the normal anatomy and physiology of the cardiac conduction system is critical to understanding appropriate utilization of device therapy.

The sinoatrial (SA) node, located at the junction of the right atrium and the superior vena cava, is normally the site of impulse generation ([Fig. 1.1](#)). The SA node is composed of a dense collagen matrix containing a variety of cells. The large, centrally located P cells are thought to be the origin of electrical impulses in the SA node, which is surrounded by transitional cells and fiber tracts extending through the perinodal area into the right atrium proper. The SA node is richly innervated by the autonomic nervous system, which has a key function in heart rate regulation. Specialized fibers, such as Bachmann's bundle, conduct the impulse



throughout the right and left atria. The SA node has the highest rate of spontaneous depolarization and under normal circumstances is responsible for generating most impulses.

**Fig. 1.1** Drawing of the cardiac conduction system. AV, atrioventricular; SA, sinoatrial. See text for details.



Atrial conduction fibers converge, forming multiple inputs into the atrioventricular (AV) node, a small subendocardial structure located within the interatrial septum ([Fig. 1.1](#)). The AV node likewise receives abundant autonomic innervation, and it is histologically similar to the SA node because it is composed of a loose collagen matrix in which P cells and transitional cells are located. Additionally, Purkinje cells and myocardial contractile fibers may be found. The AV node allows for physiological delay between atrial and ventricular contraction, resulting in optimal cardiac hemodynamic function. It can also function as a subsidiary “pacemaker” should the SA node fail. Finally, the AV node functions (albeit typically suboptimally) to regulate the number of impulses eventually reaching the ventricle in instances of atrial tachyarrhythmia.

Purkinje fibers emerge from the distal AV node to form the bundle of His, which runs through the membranous septum to the crest of the muscular septum, where it divides into the various bundle branches. The bundle branch system exhibits significant individual variation and is invariably complex. The right bundle is typically a discrete structure

running along the right side of the interventricular septum to the anterior papillary muscle, where it divides. The left bundle is usually a large band of fibers fanning out over the left ventricle, sometimes forming functional fascicles. Both bundles eventually terminate in individual Purkinje fibers interdigitating with myocardial contractile fibers. The His-Purkinje system has little in the way of autonomic innervation.

Because of their key function and location, the SA and AV nodes are the most common sites of conduction system failure; it is therefore understandable that the most common indications for pacemaker implantation are SA node dysfunction and high-grade AV block. It should be noted, however, that conduction system disease is frequently diffuse and may involve the specialized conduction system at multiple sites.

Although the earliest pacemakers were designed to treat life-threatening ventricular bradyarrhythmias, indications have drastically expanded to include conditions that do not specifically involve intrinsic conduction system disease. Guidelines have been developed to provide uniform criteria for device implantation, but the importance of the patient's clinical status and any extenuating circumstances should also be considered.

## **Electrophysiology of myocardial stimulation**

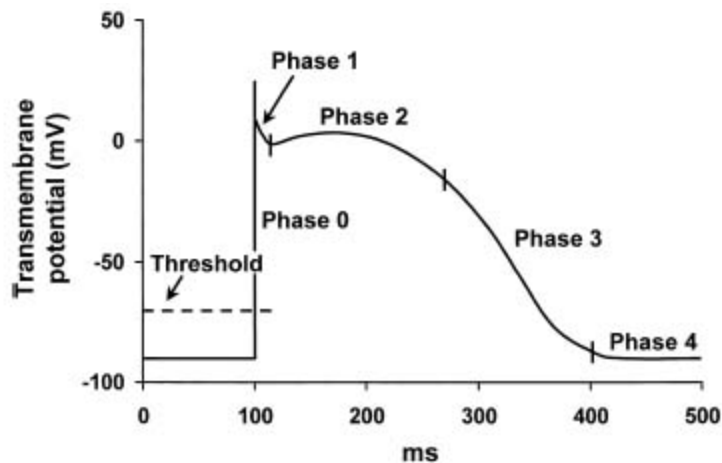
Stimulation of the myocardium by a pacemaker requires the initiation of a self-propagating wave of depolarization from the site of initial activation, whether from a native "pacemaker" or from an artificial stimulus. Myocardium exhibits a biological property referred to as "excitability," which is a response to a stimulus out of proportion to the

strength of that stimulus.<sup>1</sup> Excitability is maintained by separation of chemical charge, which results in an electrical transmembrane potential. In cardiac myocytes, this electrochemical gradient is created by differing intracellular and extracellular concentrations of sodium ( $\text{Na}^+$ ) and potassium ( $\text{K}^+$ ) ions;  $\text{Na}^+$  ions predominate extracellularly and  $\text{K}^+$  ions predominate intracellularly. Although this transmembrane gradient is maintained by the high chemical resistance intrinsic to the lipid bilayer of the cellular membrane, passive leakage of these ions occurs across the cellular membrane through ion channels. Passive leakage is offset by two active transport mechanisms, each transporting three positive charges out of the myocyte in exchange for two positive charges that are moved into the myocyte, producing cellular polarization.<sup>2,3</sup> These active transport mechanisms require energy and are susceptible to disruption when energy-generating processes are interrupted.

The chemical gradient has a key role in the generation of the transmembrane action potential ([Fig. 1.2](#)). The membrane potential of approximately -90 mV drifts upward to the threshold potential of approximately -70 to -60 mV. At this point, specialized membrane-bound channels modify their conformation from an inactive to an active state, which allows the abrupt influx of extracellular  $\text{Na}^+$  ions into the myocyte<sup>4,5</sup>, creating phase 0 of the action potential and rapidly raising the transmembrane potential to approximately +20 mV.<sup>6,7</sup> This rapid upstroke creates a short period of overshoot potential (phase 1), which is followed by a plateau period (phase 2) created by the inward calcium ( $\text{Ca}^{2+}$ ) and  $\text{Na}^+$  currents balanced against outward  $\text{K}^+$  currents.<sup>8-10</sup> During phase 3 of the action potential, the transmembrane potential returns to normal, and during

phase 4 the gradual upward drift in transmembrane potential repeats. The shape of the transmembrane potential and the relative distribution of the various membrane-bound channels differ between the components of the specialized cardiac conduction system.

**Fig. 1.2** Action potential of a typical Purkinje fiber, with the various phases of depolarization and repolarization (described in the text). (From Stokes KB, Kay GN. Artificial electric cardiac stimulation. In: Ellenbogen KA, Kay GN, Wilkoff BL, eds. *Clinical cardiac pacing*. Philadelphia: WB Saunders Co., 1995:3-37. By permission of the publisher.)



Depolarization of neighboring cells occurs as a result of passive conduction via low-resistance intercellular connections called “gap junctions,” with active regeneration along cellular membranes.<sup>11,12</sup> The velocity of depolarization throughout the myocardium depends on the speed of depolarization of the various cellular components of the myocardium and on the geometrical arrangement and orientation of the myocytes. Factors such as myocardial ischemia, electrolyte imbalance, metabolic abnormalities, and drugs may affect the depolarization and depolarization velocity.

# Pacing basics

## Stimulation threshold

Artificial pacing involves delivery of an electrical impulse from an electrode of sufficient strength to cause depolarization of the myocardium in contact with that electrode and propagation of that depolarization to the rest of the myocardium. The minimal amount of energy required to produce this depolarization is called the stimulation threshold. The components of the stimulus include the pulse amplitude (measured in volts) and the pulse duration (measured in milliseconds). An exponential relationship exists between the stimulus amplitude and the duration, resulting in a hyperbolic strength-duration curve. At short pulse durations, a small change in the pulse duration is associated with a significant change in the pulse amplitude required to achieve myocardial depolarization; conversely, at long pulse durations, a small change in pulse duration has relatively little effect on threshold amplitude ([Fig. 1.3](#)). Two points on the strength-duration curve should be noted ([Fig. 1.4](#)). The *rheobase* is defined as the smallest amplitude (voltage) that stimulates the myocardium at an infinitely long pulse duration (milliseconds). The *chronaxie* is the threshold pulse duration at twice the stimulus amplitude, which is twice the rheobase voltage. The chronaxie is important in the clinical practice of pacing because it approximates the point of minimum threshold energy (microjoules) required for myocardial depolarization.

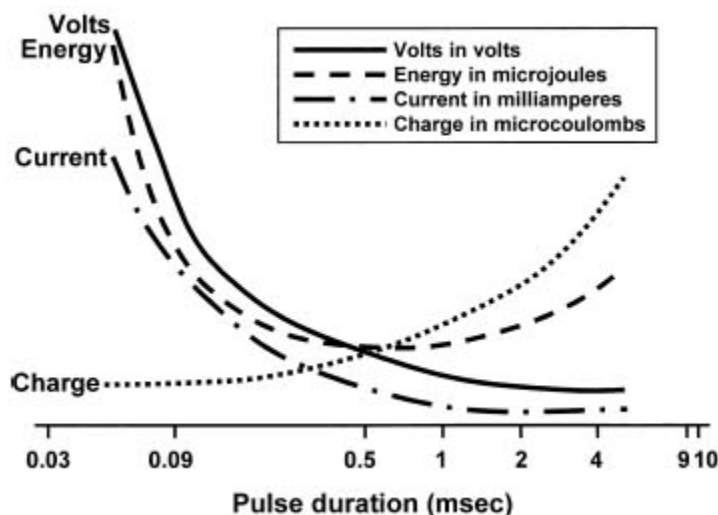
The relationship of voltage, current, and pulse duration to stimulus energy is described by the formula

$$E = V^2/R \times t$$

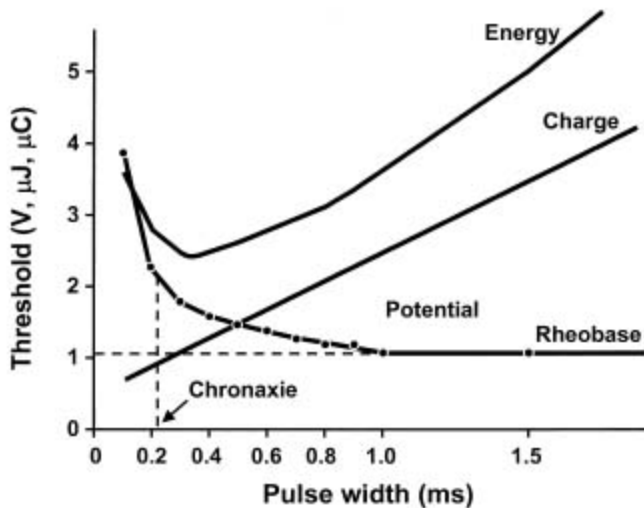
in which  $E$  is the stimulus energy,  $V$  is the voltage,  $R$  is the total pacing impedance, and  $t$  is the pulse duration. This

formula demonstrates the relative increase in energy with longer pulse durations. The energy increase due to duration is offset by a decrement in the needed voltage.

**Fig. 1.3** Relationship of charge, energy, voltage, and current to pulse duration. As the pulse duration is shortened, voltage and current requirements increase. Charge decreases as pulse duration shortens. At threshold, energy is lowest at a pulse duration of 0.5–1 .0ms and increases at pulse widths of shorter and longer duration. (Modified from Furman S. Basic concepts. In: Furman S, Hayes DL, Holmes DR Jr, eds. *A practice of cardiac pacing*. Mount Kisco, NY: Futura Publishing Co. By permission of the publisher.)

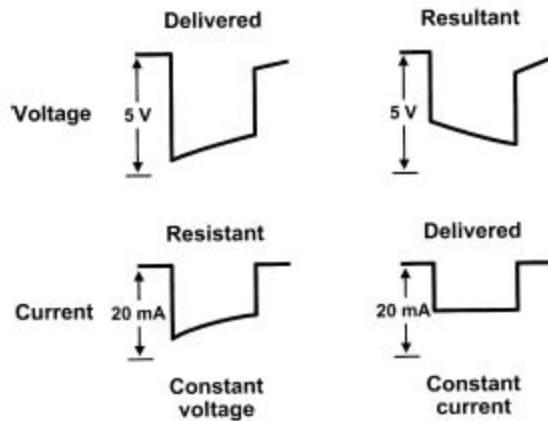


**Fig. 1.4** Relationships among chronic ventricular strength-duration curves from a canine, expressed as potential (V), charge ( $\mu\text{C}$ ), and energy ( $\mu\text{J}$ ). Rheobase is the threshold at infinitely long pulse duration. Chronaxie is the pulse duration at twice rheobase. (From Stokes K, Bornzin G. The electrode-biointerface stimulation. In: Barold SS, ed. *Modern cardiac pacing*. Mount Kisco, NY: Futura Publishing Co., 1985:33–77. By permission of the publisher.)



The strength-duration curve discussed thus far has been that of a constant voltage system, because contemporary permanent pacemakers are constant voltage systems. Constant current devices are no longer used ([Fig. 1.5](#)). It should be recognized, however, that constant current strength-duration curves can also be constructed.<sup>13</sup> These strength-duration curves, like constant voltage curves, are hyperbolic in shape, but they have a much more gradual decline in current requirements as the pulse width lengthens. Because of this gradual decline, chronaxie of a constant current system is significantly greater than that in a constant voltage system.

**Fig. 1.5** Diagrammatic representation of the delivered voltage and resultant current in a constant-voltage system compared with the delivered current and resultant voltage in a constant-current system. (Modified from Stokes K, Bornzin G. The electrode-biointerface stimulation. In: Barold SS, ed. *Modern cardiac pacing*. Mount Kisco, NY: Futura Publishing Co., 1985:33-77. By permission of the publisher.)



Impedance is the term applied to the impediment to current flow in the pacing system. Ohm's law describes the relationship among voltage, current, and resistance as

$$V = IR$$

in which  $V$  is the voltage,  $I$  is the current, and  $R$  is the resistance. Although Ohm's law is used for determining impedance, technically impedance and resistance are not interchangeable terms. Impedance implies inclusion of all factors that contribute to current flow impediment, including lead conductor resistance, electrode resistance, resistance due to electrode polarization, capacitance and inductance. Technically, the term "resistance" does not include the effects of capacitance (storage of charge) or inductance (storage of current flow) to impede current flow. Nevertheless, Ohm's law (substituting impedance for  $R$ ) is commonly used for calculating impedance. In constant voltage systems, the lower the pacing impedance, the greater the current flow; conversely, the higher the pacing impedance, the lower the current flow. Ideally, the lead conductor material would have a low resistance to minimize the generation of energy-wasting heat as the current flows along the lead, and the electrode would have a high resistance to minimize current flow and negligible electrode polarization. Decreasing the electrode radius minimizes current flow by providing greater electrode resistance and



increased current density, resulting in greater battery longevity and lower stimulation thresholds.<sup>14</sup>

“Polarization” refers to layers of oppositely charged ions that surround the electrode during the pulse stimulus. It is related to the movement of positively charged ions ( $\text{Na}^+$  and  $\text{H}_3\text{O}^+$ ) to the cathode; the layer of positively charged ions is then surrounded by a layer of negatively charged ions ( $\text{Cl}^-$ ,  $\text{HPO}_4^{2-}$ , and  $\text{OH}^-$ ). These layers of charge develop during the pulse stimulus, reaching peak formation at the termination of the pulse stimulus, after which they gradually dissipate. Polarization impedes the movement of charge from the electrode to the myocardium, resulting in a need for increased voltage. Since polarization develops with increasing pulse duration, one way to combat formation of polarization is to shorten the pulse duration. Electrode design has incorporated the use of materials that discourage polarization, such as platinum black, iridium oxide, titanium nitride, and activated carbon.<sup>15</sup> Finally, polarization is inversely related to the surface area of the electrode. To maximize the surface area (to reduce polarization) but minimize the radius (to increase electrode impedance), electrode design incorporates a small radius but a porous, irregular surface construction.<sup>16</sup> Leads designed to maximize these principles are considered “high-impedance” leads.

## **Variations in stimulation threshold**

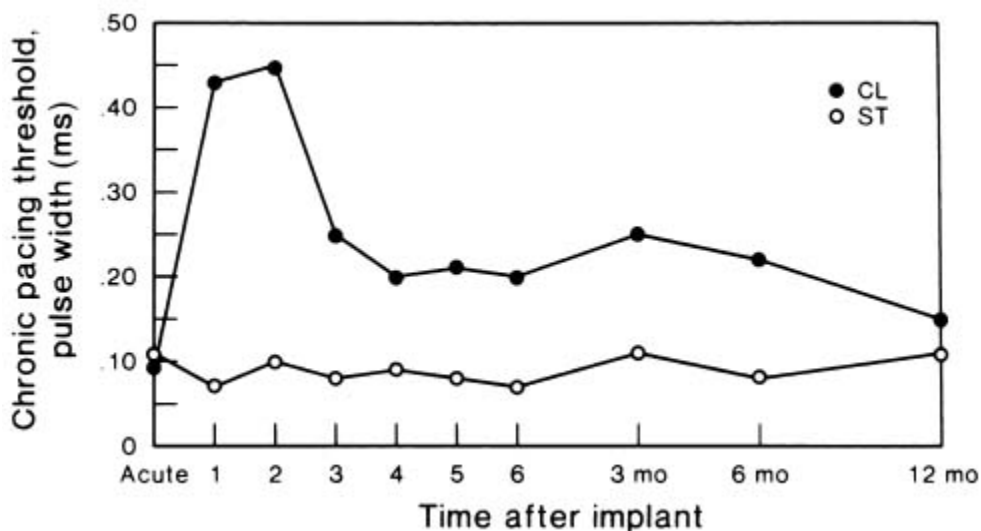
Myocardial thresholds typically fluctuate, occasionally dramatically, during the first weeks after implantation. After implantation of earlier generations of endocardial leads, the stimulation threshold would typically rise rapidly in the first 24h and then gradually increase to a peak at approximately 1 week ([Fig. 1.6](#)). Over the ensuing 6–8 weeks, the

stimulation threshold would usually decline to a level somewhat higher than that at implantation, but less than the peak threshold, known as the “chronic threshold”.<sup>17,18</sup> The magnitude and duration of this early increase in threshold is highly dependent on lead design, the interface between the electrode and the myocardium, and individual patient variation, but chronic thresholds would typically be reached by 3 months. The single most important lead design change to alter pacing threshold evolution was incorporation of steroid elution at the lead tip. With steroid elution there is a slight increase in thresholds post implantation, with subsequent reduction to almost that of acute thresholds.<sup>19,20</sup>

Transvenous pacing leads have used passive or active fixation mechanisms to provide a stable electrode-myocardium interface. Active fixation leads may have higher initial pacing thresholds at implantation, but frequently decline significantly within the first 5-30 min after placement.<sup>17</sup> This effect has been attributed to hyperacute injury due to advancement of the screw into the myocardium. On a cellular level, implantation of a transvenous pacing lead results in acute injury to cellular membranes, which is followed by the development of myocardial edema and coating of the electrode surface with platelets and fibrin. Subsequently, various chemotactic factors are released, and an acute inflammatory reaction develops, consisting of mononuclear cells and polymorphonuclear leukocytes. After the acute response, release of proteolytic enzymes and oxygen free radicals by invading macro-phages accelerates cellular injury. Finally, fibroblasts in the myocardium begin producing collagen, leading to production of the fibrotic capsule surrounding the electrode. This fibrous capsule ultimately increases the effective radius of the electrode, with a smaller increase in

surface area.<sup>21,22</sup> Steroid-eluting leads are believed to minimize fibrous capsule formation. In both atrial and ventricular active fixation leads, steroid elution results in long-term reduction in energy consumption with maintenance of stimulation thresholds, lead impedance values, and sensing thresholds.<sup>23,24</sup>

**Fig. 1.6** Long-term pacing thresholds from a conventional lead (no steroid elution) (CL; *closed circles*) and a steroid-eluting lead (ST; *open circles*). With the conventional lead, an early increase in threshold decreases to a plateau at approximately 4 weeks. The threshold for the steroid-eluting lead remains relatively flat, with no significant change from short-term threshold measurements. (From Furman S. Basic concepts. In: Furman S, Hayes DL, Holmes DR Jr, eds. *A practice of cardiac pacing*, second edn. Mount Kisco, NY: Futura Publishing Co., 1989:23-78. By permission of Mayo Foundation.)



The stimulation threshold typically has a circadian pattern, generally increasing during sleep and decreasing during the day, probably reflecting changes in autonomic tone. The stimulation threshold may also rise after eating; during hyperglycemia, hypoxemia or acute viral illnesses; or as a result of electrolyte fluctuations. These changes, as well as

the circadian variation in stimulation threshold, are usually minimal. Certain drugs used in patients with cardiac disease may also increase pacing thresholds (see Chapter 8: Programming).

The inflammatory reaction and subsequent fibro-sis that occur after lead implantation may act as an insulating shield around the electrode. These processes effectively increase the distance between the electrode and the excitable tissue, allowing the stimulus to disperse partially before reaching the excitable cells. These changes result in an increased threshold for stimulation and attenuate the amplitude and slew rate of the endocardial signal being sensed. This is a process termed “lead maturation.” Improvements in electrode design and materials have reduced the severity of the inflammatory reaction and thus improved lead maturation rates.<sup>19,25</sup> When the capture threshold exceeds the programmed output of the pacemaker, exit block will occur; loss of capture will result if the capture threshold exceeds the programmed output of the pacemaker.<sup>17,26</sup> Exit block, a consequence of lead maturation, results from the progressive rise in thresholds over time.<sup>17,26</sup> This phenomenon occurs despite initial satisfactory lead placement and implantation thresholds, often but not always occurs in parallel in the atrium and ventricle, and usually recurs with placement of subsequent leads. Steroid-eluting leads prevent exit block in most, but not all patients ([Fig. 1.7](#)).

## **Sensing**

The first pacemakers functioned as fixed-rate, VOO devices. All contemporary devices offer demand-mode pacing, which pace only when the intrinsic rate is below the programmed rate. For such devices to function as programmed, accurate and consistent sensing of the native rhythm was essential.