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
Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach

SECOND EDITION

EDITED BY

David L. Hayes, M.D.
Chair, Division of Cardiovascular Diseases, Mayo Clinic, Rochester, Minnesota, USA
Professor of Medicine, College of Medicine, Mayo Clinic

Paul A. Friedman, M.D.
Consultant, Division of Cardiovascular Diseases, Mayo Clinic, Rochester, Minnesota, USA
Professor of Medicine, College of Medicine, Mayo Clinic

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## Contents

Contributors, vii

Preface, ix

1 Clinically Relevant Basics of Pacing and Defibrillation, 1
   - T. Jared Bunch, David L. Hayes, Paul A. Friedman

2 Hemodynamics of Device Therapy, 43
   - David L. Hayes, Paul A. Friedman, Samuel J. Asirvatham

3 Indications for Pacemakers, ICDs and CRT, 82
   - Apoor S. Gami, David L. Hayes, Paul A. Friedman

4 Generator and Lead Selection, 121
   - Samuel J. Asirvatham, David L. Hayes, Paul A. Friedman

5 Implantation and Extraction Techniques, 144
   - David L. Hayes, Paul A. Friedman, Samuel J. Asirvatham

6 Implantation-related Complications, 202
   - David L. Hayes, Paul A. Friedman

7 Pacemaker and Cardiac Resynchronization Timing Cycles and Electrocardiography, 234
   - David L. Hayes, Paul J. Wang, Samuel J. Asirvatham, Paul A. Friedman

8 Programming, 300
   - David L. Hayes, Charles D. Swerdlow, Paul A. Friedman

9 Rate-adaptive Pacing, 380
   - David L. Hayes, Samuel J. Asirvatham

10 Troubleshooting, 401
   - Paul A. Friedman, Charles D. Swerdlow, David L. Hayes

11 Pacemaker, ICD and CRT Radiography, 517
   - David L. Hayes, Paul A. Friedman

12 Electromagnetic Interference and Implantable Devices, 550
   - David L. Hayes, Paul A. Friedman

13 Follow-up, 572
   - David L. Hayes, Niloufar Tabatabaei, Michael Glikson, Paul A. Friedman

Index, 617
Contributors

Samuel J. Asirvatham, MD
Consultant, Cardiac Electrophysiology, Mayo Clinic, Rochester, Minnesota, USA
Associate Professor of Medicine, College of Medicine, Mayo Clinic

T. Jared Bunch, MD.
Fellow in Electrophysiology, Mayo School of Graduate Medical Education, College of Medicine, Mayo Clinic, Rochester, Minnesota, USA

Paul A. Friedman, MD
Consultant, Division of Cardiovascular Diseases, Mayo Clinic, Rochester, Minnesota, USA
Professor of Medicine, College of Medicine, Mayo Clinic

Apoor S. Gami, MD
Fellow in Electrophysiology, Mayo School of Graduate Medical Education, Mayo Clinic, Rochester, Minnesota, USA
Assistant Professor of Medicine, College of Medicine, Mayo Clinic

Michael Glikson, MD
Associate Clinical Professor of Cardiology
Director - Electrophysiology and Pacing Unit
Heart Institute, Sheba Medical Center and Tel Aviv University, Tel–Hashomer, Israel

David L. Hayes, MD
Chair, Division of Cardiovascular Diseases, Mayo Clinic, Rochester, Minnesota, USA
Professor of Medicine, College of Medicine, Mayo Clinic

Charles D. Swerdlow, MD
Cedars Sinai Medical Center, Los Angeles
Clinical Professor of Medicine
David Geffen School of Medicine at UCLA
Los Angeles, California, USA

Niloufar Tabatabaei, MD
Fellow in Cardiovascular Diseases, Mayo School of Graduate Medical Education, College of Medicine, Mayo Clinic, Rochester, Minnesota, USA

Paul J. Wang, MD
Professor of Medicine
Director of Cardiac Arrhythmia Service and Cardiac Electrophysiology
Stanford University School of Medicine, Stanford, California, USA
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 multi-authored 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 Tabatabaei, 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,
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.

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 mem-
branous 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. 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 (Na⁺) and potassium (K⁺) ions; Na⁺ ions predominate extracellularly and 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. 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 Na⁺ ions into the myocyte, creating phase 0 of the action potential and rapidly raising the transmembrane potential to approximately +20 mV.\(^\text{18}\)\(^\text{19}\)

![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.)](image-url)
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 (Ca\(^{2+}\)) and Na\(^{+}\) currents balanced against outward K\(^{+}\) currents.\(^8\)\(^\text{-10}\) 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 ion channels differ between the components of the specialized cardiac conduction system.

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.\(^1\)\(^1\)\(^\text{-}2\) 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

\[
E = \frac{V^2}{R} \times t
\]

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.
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. 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.

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.14

“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 (Na+ and H2O+) to the cathode; the layer of positively charged ions is then surrounded by a layer of negatively charged ions (Cl–, HPO4 2–, and 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.15 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.16 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 24 h 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”17,18. 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.19,20

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.17 This effect has been at-

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**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.)
tributed 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 macrophages 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.21,22 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.23,24

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 fibrosis 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.19,25 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.17,26 Exit block, a consequence of lead maturation, results from the progressive rise in thresholds over time.17,26 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, VO0 devices. All contemporary devices offer demand 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.

Intrinsic cardiac electrical signals are produced by the wave of electrical current through the myocardium (Fig. 1.8). As the wavefront of electrical energy approaches an endocardial electrode, the electrode becomes positively charged relative to the depolarized region, recorded as a positive deflection in the intracardiac electrogram. As the wavefront passes directly under the electrode, the outside of the cell abruptly becomes negatively charged, and a sharp negative deflection is recorded, which is referred to as the intrinsic deflection.27 It is considered to occur at about the moment the advancing wavefront passes directly underneath the electrode. Smaller positive and negative deflections preceding and following the intrinsic deflection represent activation of surrounding myocardium. Ventricular electrograms typically are much larger than atrial electrograms because the ventricular mass is greater. The maximum frequency densities of atrial and ventricular electrograms have generally been found to be in the range of 80–100 Hz in the atrium and 10–30 Hz in the ventricles (these frequencies may differ slightly with newer leads/technologies). Based on these frequencies, filtering systems of pulse generators were designed to attenuate signals outside these ranges. Filtering and use of blanking and refractory periods have markedly reduced unwanted sensing, although myopotential frequencies (ranging from 10 to 200 Hz) considerably overlap with those generated by atrial and ventricular depolarization and are difficult to filter out, especially during sensing in a unipolar configuration.26-31 Shortening of the tip-to-ring spacing has also improved atrial sensing and rejection of far-field R waves.

Another component of the intracardiac electrogram is the slew rate, i.e. the peak slope of the }
The slew rate represents the maximal rate of change of the electrical potential between the sensing electrodes and is the first derivative of the electrogram ($dV/dt$). An acceptable slew rate should be at least 0.5 V/s in both the atrium and the ventricle. In general, the higher the slew rate, the higher the frequency content and the more likely the signal will be sensed. Slow, broad signals, such as those generated by the T wave, are much less likely to be sensed because of a low slew rate and lower frequency density.

Polarization also affects sensing function. After termination of the pulse stimulus, an excess of positive charge surrounds the cathode, which then decays until electrically neutral. Afterpotentials can be sensed with inappropriate inhibition or delay of the subsequent pacing pulse (Fig. 1.10). The amplitude of afterpotentials is directly related to both the amplitude and the duration of the pacing pulse; thus, they are most likely to be sensed when the pacemaker is programmed to high voltage and long pulse duration in combination with the pacing stimulus.
with maximal sensitivity. The use of programmable sensing refractory and blanking periods has helped to prevent the pacemaker from reacting to afterpotentials, although in dual-chamber systems, atrial afterpotentials of sufficient strength and duration to be sensed by the ventricular channel may result in inappropriate ventricular inhibition (crosstalk), especially in unipolar systems. Afterpotentials may be a source of problems in devices with automatic threshold measurement and capture detection; the use of leads designed to minimize afterpotentials may increase the effectiveness of such algorithms.

"Source impedance" is a term used to describe the voltage drop that occurs from the site of the origin of the intracardiac electrogram to the proximal portion of the lead. Components include the resistance between the electrode and the myocardium, the resistance of the lead conductor material, and the effects of polarization. The resistance between the electrode and the myocardium, as well as polarization, is inversely related to the surface area of the electrode; thus, the effects of both can be minimized by a large electrode surface area. The electrogram actually seen by the pulse generator is determined by the ratio between the sensing amplifier (input impedance) and the lead (source impedance). Less attenuation of the signal from the myocardium occurs when there is a greater ratio of input impedance to source impedance. Clinically, impedance mismatch is seen with insulation or conductor failure, which results in sensing abnormalities or failure.

**Lead design**

Pacing lead components include the electrode and fixation device, the conductor, the insulation, and the connector pin (Figs 1.11 and 1.12). Leads function in a harsh environment in vivo. They must be constructed of materials that provide both mechanical stability and flexibility; they must have satisfactory electrical conductive and resistive properties; the insulating material must be durable but ideally have a low friction coefficient to facilitate implantation; and the electrode must provide good mechanical and electrical contact with the myocardium. Industry continues to modify and improve lead design, but the "ideal" lead remains a constant goal.

As previously discussed, optimal stimulation and sensing thresholds favor an electrode with a small radius and a large surface area. Electrode shape and surface composition have evolved over time. Early models utilized a round spherical shape with a smooth metal surface. Electrodes with an irregular, textured surface allow for increased surface area without an increase in electrode radius. To achieve increased electrode surface area, manufacturers have used a variety of designs, including microscopic pores, coatings of microspheres, and wire filament mesh.

Unfortunately, relatively few conductive materials have proven to be satisfactory for use in pacing electrodes. Ideally, electrodes are biologically inert, resist degradation over time, and do not elicit a marked tissue reaction at the myocardium-electrode interface. Certain metals, such as zinc, copper, mercury, nickel, lead and silver, are associated with toxic reactions with the myocardium. Stainless steel alloys are susceptible to corrosion. Titanium, tantalum, platinum and iridium oxide acquire a surface coating of...
oxides that impedes current transfer. Materials currently in use are platinum-iridium, platinized titanium-coated platinum, iridium oxide, and platinum (Fig. 1.13). Carbon electrodes seem to be least susceptible to corrosion; they have also been improved by a process known as activation, which roughens the surface to increase the surface area and allow for tissue ingrowth.37

Lead fixation may be active or passive. Passive fixation endocardial leads usually incorporate tines at the tip that become ensnared in trabeculated tissue in the right atrium or ventricle, providing lead stability. Leads designed for coronary venous placement usually incorporate a design that wedges the lead against the wall of the coronary vein. Active fixation leads almost exclusively utilize screw mechanisms to embed in the myocardium to provide lead stability. Some leads incorporate screws that are electrically inactive, and in others the screw is electrically active. There are advantages and disadvantages to each design, and the clinical situation and preference of the operator are important considerations when a lead is chosen. Considerable myocardial and fibrous tissue enveloping the tip typically develops with both active and passive fixation leads. However, the encasement of the tines of a passive fixation lead by fibrous tissue often makes the extraction of passive fixation leads more difficult than that of active fixation leads. Active fixation leads are
often preferable in patients with distorted anatomy, such as those with congenital cardiac defects or those with surgically amputated atrial appendages. Active fixation leads are also preferable in patients with high right-sided pressures. As alternative site pacing has evolved, i.e., the placements of leads outside the right atrial appendage and right ventricular apex, screw-in leads have become more popular and necessary for long-term stability.

There are various types of mechanism used to keep the screw unexposed until it is placed in an optimal site for fixation. One example is a system in which the screw is extendable and retractable from the pacemaker lead tip. This allows the operator to designate the precise location and timing to extend the screw from the tip. Another example involves covering a fixed helix screw in a material that dissolves in the blood stream in a time period that is advantageous for lead positioning. For example, screws can be covered by a mannitol compound that dissolves over time in the blood stream. Since the mannitol covers the screw, it prevents it from catching on tissue, allowing easier lead placement.

New technologies have emerged to assist in the placement of leads to targeted anatomical sites. Catheter-delivered systems use a deflectable catheter that is braided to allow the simultaneous ability to torque the catheter. A second technology developed to reach difficult anatomical targets is to use an over-the-wire lead delivery system, mainly used with placement of coronary venous leads for left ventricular stimulation. With this system the lead can be advanced to a stable position, a guidewire then being advanced to navigate tortuous regions similar to techniques used extensively for coronary angiography, followed by advancement of the lead over the wire. This approach not only improves access to target sites, but decreases injury to coronary venous structures. By combining these technologies, access to target sites has improved greatly, in particular, coronary vein subselection for left ventricular lead placement.

Conductors are commonly of a multifilament design to facilitate tensile strength and reduce resistance to metal fatigue (Fig. 1.14). Alloys such as MP35N (cobalt, nickel, chromium and molybdenum) and nickel-silver are typically used in modern pacing leads. Bipolar leads may be of coaxial design, with an inner coil extending to the distal electrode and an outer coil terminating at the proximal electrode (Fig. 1.15). This design requires that the conductor coils be separated by a layer of inner insulation. Coaxial designs remain commonly used in the treatment of bradyarrhythmias. Some bipolar leads are coradial, or “parallel-wound”; that is, two insulated coils are wound next to each other. Leads may also be constructed with the conductor coils parallel to each other (multiluminal), again separated by insulating material (Fig. 1.16). This type of design is typically used for tachyarrhythmia leads. Additionally, leads may use a combination of coils and cables. The coil facilitates the passage of a stylet for lead implantation, and the cable allows a smaller lead body.

Two materials have predominated in lead insulation: silicone and polyurethane. Each has its respective advantages and disadvantages, but the overall performance of both materials has been excellent.38 Table 4.2 in
Chapter 4 compares the advantages and disadvantages of these two insulating materials.

The two grades of polyurethane that have had the widest use are Pellathane 80A and Pellathane 55D. Early after the introduction of polyurethane as an insulating material, it became clear that clinical failure rates with specific leads were higher than acceptable; further investigation revealed that the failures were occurring primarily in leads insulated with the P80A polymer. Microscopic cracks developed in the P80A polymer, initially occurring as the heated polymer cooled during manufacture; with additional environmental stress, these cracks propagated deeper into the insulation, resulting in failure of the lead insulation.

Polyurethane may also undergo oxidative stress in contact with conductors containing cobalt and silver chloride, resulting in degradation of the lead from the inside and subsequent lead failure. Some current leads use silicone with a polyurethane coating, incorporating the strength and durability of silicone with the ease of handling of polyurethane while maintaining a satisfactory external lead diameter. Silicone rubber is well known to be susceptible to abrasion wear, cold flow due to cyclic compression, and wear from lead-to-lead and lead-to-can contact. Current silicone leads have surface modifications that improve lubricity and reduce friction in blood. Second, preliminary studies have suggested that a hybrid coating of silicone and polyurethane may offer improved wear. Despite lead improvements, laboratory testing and premarketing, clinical trials have been inadequate to predict the long-term performance of leads, so that clinicians implanting the devices or performing follow-up in patients with pacing systems must vigilantly monitor lead status.

Contemporary leads and connectors are standardized to conform to international guidelines (IS-1 Standard), which mandate that leads have a 3.2-mm diameter in-line bipolar connector pin. These standards were established many years ago because some leads and connector blocks were incompatible, requiring the development of multiple adaptors. Some patients who have functioning leads of the older 5- or 6-mm diameter unipolar design require lead adaptors when the pulse generator is replaced.

Coronary venous lead connectors were initially developed to accommodate patients with heart failure who had previously implanted pacemakers for other reasons and were considered eligible for an upgrade to biventricular pacing. For these patients, the ventricular output of the pacemaker generator was divided via a “Y” connector from one bipolar output to two separate outputs (usually a unipolar left ventricle and a bipolar right ventricle or a bipolar left ventricle and a bipolar right ventricle) to accommodate the left ventricular lead. However, this approach can lead to atrial oversensing, improper measurement of left ventricular thresholds, and inappropriate shocks. Currently, most left ventricular leads are connected to the pacemaker independently. The left ventricular leads are either bipolar or unipolar with a steroid eluding tip.

**Bipolar vs. unipolar pacing and sensing**

In unipolar pacing systems, the lead tip functions as the cathode and the pulse generator functions as the anode (Fig. 1.16). In bipolar systems, the lead tip functions as the cathode and the lead ring functions as the anode (Fig. 1.16). Unipolar leads are of simpler design and have a smaller external diameter. Unipolar leads have historically demonstrated greater durability than bipolar leads. In recent years the difference in durability has been less distinct. Unipolar leads do not offer the option of bipolar function. Although unipolar and bipolar leads are readily available, present usage of transvenous leads is almost exclusively bipolar in the USA. This is in contrast to epicardial leads, of which there is a lower percentage of bipolar leads in use. Bipolar leads may function in the unipolar mode if the pace-
maker is so programmed. They are available in several designs, generally coaxial or multiluminal. Regardless of design, the external diameter of a bipolar lead is usually greater than that of unipolar leads because each coil must be electrically separated by insulating material. Bipolar pacing and sensing are preferred over unipolar because bipolar pacing cannot cause extracardiac stimulation at the pulse generator, which may occasionally occur with unipolar pacing due to current returning to the generator. Also, bipolar sensing is less likely to detect myopotentials, far-field signals and electromagnetic interference.44

There are long-standing controversies regarding unipolar vs. bipolar pacing and sensing configuration and which, if either, are superior.44 Advocates of unipolar configuration argue that improvements in sensing circuitry and pacemaker filtering capabilities have minimized unipolar oversensing of extracardiac signals. The design of unipolar leads is often more simple and thereby the lead size may be less. They also argue that bipolar leads have a historically higher failure rate than unipolar leads. Although this is true, if the specific failures of Pellathane 80A and 55D that occurred many years ago are removed from the analysis, the failure rate between unipolar and bipolar lead designs does not differ significantly and varies between different manufacturers.45 Unipolar leads are often considered safer because they do not short circuit significantly when there are insulation breaches, although they may be susceptible to significant external interference. Nevertheless, a lead that is malfunctioning in the bipolar mode may function satisfactorily when programmed to the unipolar configuration (see Chapter 8: Programming).

Most pulse generators offer independently programmable pacing and sensing in each channel; however, bipolar programming of a device attached to a unipolar lead results in no output. Bipolar leads can function in the unipolar mode; the converse is not true.

**Left ventricular leads**
Cardiac resynchronization therapy with biventricular pacing is an established treatment for patients with severe congestive heart failure, low left ventricular ejection fraction, and New York Heart Association class III or IV heart failure.46 In order to pace the left ventricle, a pacing lead is implanted transvenously through the coronary sinus and coronary vein to stimulate the left ventricular free wall. Resynchronization is obtained by stimulating both ventricles to contract with minimal intraventricular delay, thereby improving the left ventricular performance.47 Modifications of the tip geometry have improved the stability of the passive lead over time. Tissue ingrowth can be a major impediment to the removal of defibrillation leads implanted in the coronary sinus. Coating these leads with polytetrafluoroethylene and backfilling the coil with medical adhesive facilitates transvenous lead removal.48

**Pulse generators**
All pulse generators include a power source, an output circuit, a sensing circuit, a timing circuit, and a header connector for communication with the external world. The most common power sources are lithium iodine cells, lithium-silver-oxide-vanadium cells, and lithium-silver oxide. The battery voltage of the cell depends on the chemical composition of the cell; at the beginning of life for the lithium iodine battery, the cell generates approximately 2.8 V, which decreases to 2.4 V when approximately 90% of the useable battery life has been reached. The voltage then exponentially declines to 1.8 V as the battery reaches end-of-life. However, the voltage at which the cell reaches a certain depth of discharge is load dependent. The elective replacement indicated voltages were chosen based on the shape of the discharge curves under expected operating conditions. When the battery is at end-of-service, most devices lose telemetry and programming capabilities, frequently reverting to a fixed high-output mode to attempt to maintain...
The pulse duration. The voltage drop is also dependent on the capacitance value of the capacitor and the time of longer pulse duration.

The output waveform is followed by a low-amplitude wave of opposite polarity, the afterpotential. The afterpotential is determined by the polarization of the electrode at the electrode–tissue interface; formation is due to electrode characteristics as well as to pulse amplitude and duration. The sensing circuit may sense afterpotentials of sufficient amplitude, especially if the sensitivity threshold is low. Newer pacemakers use the output circuit to discharge the afterpotential quickly, thus lowering the incidence of afterpotential sensing.

The afterpotential also helps to prevent electrode corrosion.

The intracardiac electrogram is conducted from the myocardium to the sensing circuit via the pacing leads, where it is then amplified and filtered. As noted above, the input impedance must be significantly larger than the sensing impedance to minimize attenuation of the electrogram. A bandpass filter attenuates signals on either side of a center frequency, which varies among manufacturers (generally ranging from 20 to 40 Hz). After filtering, the electrogram signal is compared with a reference voltage, the sensitivity setting: signals with an amplitude of this reference voltage or higher are sensed as true intracardiac events and are forwarded to the timing circuitry, whereas signals with an amplitude below the reference amplitude are categorized as noise, extracardiac or other cardiac signal, such as T waves.

Sensing circuitry also incorporates noise reversion circuits that cause the pacemaker to revert to a noise reversion mode (asynchronous pacing) whenever the rate of signal received by the sensing circuit exceeds the noise reversion rate. This feature is incorporated to prevent inhibition of pacing when the device is exposed to electromagnetic interference. Pulse generators also use Zener diodes designed to protect the circuitry from high external voltages, which may occur, for example, with defibrillation. When the input voltage presented to the pacemaker exceeds the Zener voltage, the excess voltage is shunted back through the leads to the myocardium.

The timing circuit of the pacemaker is a crystal oscillator that regulates the pacing cycle length, refractory periods, blanking periods and AV intervals with extreme accuracy. The output from the oscillator (as well as signals from the sensing circuitry) is sent to a timing and logic control board that operates the inter-
Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach

Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach

Nal clocks, which in turn regulate all the various timing cycles of the pulse generator. The timing and logic control circuitry also contains an absolute maximal upper rate cut-off to prevent “runaway pacing” in the event of random component failure.55,56

Each new generation of pacemakers contains more microprocessor capability. The circuitry contains a combination of read-only memory (ROM) and random-access memory (RAM). ROM is used to operate the sensing and output functions of the device, and RAM is used in diagnostic functions. Larger RAM capability has allowed devices to store increased amounts of retrievable diagnostic information, with the potential to allow downloading of new features externally into an implanted device.

External telemetry is included in all implantable devices. The pulse generator can receive information from the programmer and send information back by radiofrequency signals. Each manufacturer’s programmer and pulse generator operate on an exclusive radiofrequency, preventing the use of one manufacturer’s programmer with a pacemaker from another manufacturer. Through telemetry, the programmer can retrieve both diagnostic information and real-time information on battery status, lead impedance, current, pulse amplitude and pulse duration. Real-time electrograms and marker channels can also be obtained with most devices. The device can also be directed to operate within certain limits and to store specific types of diagnostic information via the programmer.

The most recent change in telemetry is that of “remote” capability. Information exchange has traditionally occurred by placing and leaving the programming ‘head’ of the programmer over the pulse generator for the duration of the interrogation and programming changes. New telemetry designs allow the programming ‘head’ or ‘wand’ to be placed briefly over the pulse generator to establish identity of the specific model and pulse generator and then complete the bi-directional informational exchange at a distance, i.e. the ‘wand’ does not need to be kept in a position directly over the pulse generator. Finally, even the use of a wand for certain pulse generators is not required for remote programming.

**Pacemaker nomenclature**

A lettered code to describe the basic function of pacing devices, initially developed by the American Heart Association and the American College of Cardiology, has since been modified and updated by the members of the North American Society of Pacing and Electrophysiology and the British Pacing and Electrophysiology Group (currently the Heart Rhythm Society).57 This code has five positions to describe basic pacemaker function, although it obviously cannot incorporate all of the various special features available on modern devices (Table 1.1).

The first position describes the chamber or chambers in which electrical stimulation occurs. A reflects pacing in the atrium, V implies pacing in the ventricle, D signifies pacing in both the atrium and the ventricle, and O is used when the device has antitachycardia pacing (ATP) or cardioversion-defibrillation capability but no bradycardia pacing capability.

The second position describes the chamber or chambers in which sensing occurs. The letter code

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<thead>
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<th><em><em>Table 1.1 NBG</em> code</em>*</th>
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<tr>
<td><strong>I</strong></td>
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<tr>
<td>Chamber(s) paced</td>
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<tr>
<td>O = None</td>
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<tr>
<td>A = Atrium</td>
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<tr>
<td>V = Ventricle</td>
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<td>D = Dual (A + V)</td>
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CHAPTER 1 Clinically Relevant Basics of Pacing and Defibrillation

15

the same as that in the first position, except that an O in this position represents lack of sensing in any chamber, i.e. fixed-rate pacing. (Manufacturers may use an S in both the first and the second positions to indicate single-chamber capability that can be used in either the atrium or the ventricle.)

The third position designates the mode of sensing, i.e. how the device responds to a sensed event. I indicates that the device inhibits output when an intrinsic event is sensed and starts a new timing interval. T implies that an output pulse is triggered in response to a sensed event. D indicates that the device is capable of dual modes of response (applicable only in dual-chamber systems).

The fourth position reflects both programmability and rate modulation. O indicates that none of the pacemaker settings can be changed by noninvasive programming, P suggests “simple” programmability (i.e. one or two variables can be modified), M indicates multiprogrammability (three or more variables can be modified) and C indicates that the device has telemetry capability and can communicate noninvasively with the programmer (which also implies multiprogrammability). Finally, an R in the fourth position designates rate-responsive capability. This means that the pacemaker has some type of sensor to modulate the heart rate independent of the intrinsic heart rate. All modern devices are multiprogrammable and have telemetry capability; therefore, the R to designate rate-responsive capability is the most commonly used currently.

The fifth position was originally used to identify antitachycardia treatment functions. However, this has been changed, and antitachycardia options are no longer included in the nomenclature. The fifth position now indicates whether multisite pacing is not present (O), or present in the atrium (A), ventricle (V) or both (D). Multisite pacing is defined for this purpose as stimulation sites in both atria, both ventricles, more than one stimulation site in any single chamber, or any combination of these.

All pacemaker functions (whether single- or dual-chamber) are based on timing cycles. Even the function of the most complex devices can be readily understood by applying the principles of pacemaker timing intervals. This understanding is critical to accurate interpretation of pacemaker electrocardiograms, especially during troubleshooting. Pacemaker timing cycles are described in detail in Chapter 7: Timing Cycles.

Defibrillation basics

In 1899, Prevost and Battelli noted that the “fibrillatory tremulations produced in the dog” could be arrested with the reestablishment of the normal heartbeat if one submitted the animal “to passages of current of high voltage.” Despite these early observations, decades elapsed before broad clinical applicability fueled interest in more widespread investigation of the mechanism underlying defibrillation. With the development of internal defibrillators in the late 1970s came a greater need to quantify defibrillation effectiveness, to understand the factors governing waveform and lead design, and to determine the effect of pharmacological agents on defibrillation. Remarkably, much of this work was done without a complete understanding of the fundamental mechanism of defibrillation.

This section reviews the emerging insights to the electrophysiological effects of shocks and how they are related to defibrillation. It also reviews the means of assessing the efficacy of defibrillation (the “defibrillation threshold”) and the important effects of waveform, lead design and placement, and pharmacological agents on defibrillation, with an emphasis on those principles pertaining to clinical practice.

Electrophysiological effects of defibrillation shocks; antitachycardia pacing

Despite great strides made in understanding the technology required for defibrillation (e.g. lead design and position, waveform selection), the basic underlying mechanisms have not been definitively determined. A few contemporary theories accounting for how an electric shock terminates fibrillation coexist with some overlapping: critical mass, upper limit of vulnerability, progressive depolarization, and virtual electrode depolarization. These are discussed below in brief.

First, a brief review of the cardiac action potential will be useful to facilitate discussion of the effects of defibrillation. The surface electrocardiogram and intracardiac electrogram, common in clinical practice, are the result of extracellular potentials generated by myocardial action potential propagation. An action potential is the transmembrane voltage in a single myocyte over time (Fig. 1.17). The action potential upstroke (phase 0, or depolarization) is mediated by sodium ion flow through voltage-sensitive selective channels, and during ventricular activation it is registered on the sur-
face electrocardiogram as the QRS complex (Fig. 1.18). Repolarization (phase 3) of ventricular myocardium generates the surface electrophysiographic T wave. In its resting state, the myocardium is excitable, and a pacing stimulus, or current injected by the depolarization of a neighboring myocyte, can bring the membrane potential to a threshold value, above which a new action potential ensues. The ability of the action potential of a myocyte to depolarize adjacent myocardium results in propagation of electrical activity through cardiac tissue. Importantly, immediately after depolarization, the myocardium is refractory and cannot be stimulated to produce another action potential until it has recovered excitability (Fig. 1.19). The interval immediately after an action potential, during which another action potential cannot be elicited by a pacing stimulus, is referred to as the “refractory period.”

Ventricular fibrillation (VF) is the most common cause of sudden death. VF results when an electrical wavebreak induces re-entry and results in a cascade of new wavebreaks. In patients with a structurally abnormal or diseased heart, the underlying tissue heterogeneity results in a predisposition to wavebreak, then re-entry, and finally fibrillation.59 These wandering wavelets are self-sustaining once initiated. In the 1940s, Gurvich and Yuniev60 predicted that electric shocks led to premature tissue stimulation in advance of propagating wavefronts, preventing continued progression of the wavefront. This concept of defibrillation as a large-scale stimulation remains a central tenet of many of the currently held theories of defibrillation.

Critical mass
The critical mass theory proposed that shocks need only eliminate fibrillatory wavelets in a critical amount of myocardium to extinguish the arrhythmia. Experiments in canine models found that injection of potassium chloride (which depolarizes myocardium, rendering it unavailable for fibrillation) into the right coronary artery or the left circumflex artery failed to terminate VF as often as injection into both the left circumflex and the left anterior descending arteries together. Similarly, electrical shocks of equal magnitude terminated fibrillation most frequently when the electrodes were
positioned at the right ventricular apex and the posterior left ventricle, as opposed to two right ventricular electrodes. Thus, it was concluded that if a “critical mass” of myocardium was rendered unavailable for VF either by potassium injection or by defibrillatory shock, the remaining excitable tissue was insufficient to support the wandering wavelets, and the arrhythmia terminated. However, it was not critical to depolarize every ventricular cell to terminate fibrillation.

**Upper limit of vulnerability**

Studies mapping electrical activation after failed shocks led to several observations not accounted for by the critical mass hypothesis, giving rise to the upper limit of vulnerability theory. First, an isoelectric interval (an electrical pause) was seen after failed shocks before resumption of fibrillation. The relatively long pause suggested that VF was terminated by the shock and then secondarily regenerated by it (Fig. 1.20). The concept that failed shocks are unsuccessful because they give rise to a new focus of fibrillation rather than because they fail to halt continuing wavelets was further buttressed by a second observation—that postshock conduction patterns were not the continuation of preshock wavefronts. If a failed shock resulted from the inability to halt continuing fibrillation, the assumption was that the postshock wavefronts should be a continuation of the propagating wavefronts present before shock delivery and that new wavefronts at sites remote from the preshock wavefronts would not be expected. Furthermore, VF was frequently reinitiated in the regions of lowest shock intensity, suggesting that these low-intensity regions were responsible for reinitiating fibrillation.

Elegant mapping studies demonstrated that shocks with potential gradients less than a minimum critical value—termed the upper limit of vulnerability (ULV) (6V/cm for monophasic shocks, 4V/cm for biphasic shocks)—could induce fibrillation when applied to myocardium during its vulnerable period. Low-energy shocks did so by creating regions of functional block in vulnerable myocardium at “critical points” that initiated re-entry and subsequent fibrillation. Figure 1.21 depicts the vulnerable zone during normal sinus rhythm. In sinus rhythm, low-energy shocks delivered during the T wave induce VF; higher energy shocks—with energy above the ULV—do not. Since at any given time during fibrillation a number of myocardial regions are repolarizing and thus vulnerable, a shock with a potential gradient below the ULV may create a
critical point and reinitiate fibrillation. Conversely, a shock with a gradient above the ULV across the entire myocardium does not reinduce VF and should therefore succeed. During defibrillator testing, shocks are intentionally delivered in the vulnerable zone to induce fibrillation (Fig. 1.22), and the zone of vulnerability has been defined in humans. The fact that the vulnerable zone exists and that the ULV has been correlated with the defibrillation threshold supports the ULV hypothesis as a mechanism of defibrillation.

Progressive depolarization
A third theory of defibrillation, the progressive depolarization theory (also referred to as the “refractory period extension theory”) incorporates some elements of both critical mass and ULV theories. Using voltage-sensitive optical dyes, Dillon and Kwaku have demonstrated that shocks of sufficient strength were able to elicit active responses, even from supposedly refractory myocardium. Thus, as seen in Fig. 1.23A, the duration of an action potential can be prolonged (and the refractory period extended) despite refractory myocardium when a sufficiently strong shock is applied. This phenomenon may result from sodium channel reactivation by the shock. The degree of additional depolarization time is a function of both shock intensity and shock timing. Since the shock stimulates new action potentials in myocardium that is late in repolarization and produces additional depolarization time when the myocardium is already depolarized, myocardial resynchronization occurs. This is manifested by myocardial repolarization at a constant time after the shock (second dashed line in Fig. 1.23, labeled “constant repolarization time”). Thus, the shock that defibrillates extends overall ventricular refractoriness, limiting the excitable tissue available for fibrillation. It thus extinguishes continuing wavelets and resynchronizes repolarization, so that distant regions of myocardium become excitable simultaneously, preventing dispersion of refractoriness and renewed re-entry. Experimental evidence has demonstrated that shocks with a potential gradient above the ULV result in time-dependent extension of the refractory period. In contrast, lower-energy shocks may result in a graded response that could create transient block and a critical point, thereby reinducing fibrillation.

Virtual electrode depolarization
More recently, optical signal measurements of trans-
membrane potentials have demonstrated the concept of the “virtual electrode.” The virtual electrode effect makes the defibrillation electrode effectively much larger than the physical electrode. In the virtual electrode, the anode cells are brought close to their resting potential, increasing their responsiveness to stimulation. More importantly, the region of depolarization or hyperpolarization near the physical electrode is surrounded by regions with opposite polarity. Anodal shocking produces a wavefront which begins at the boundary of positively charged regions and then spreads in the direction of the negatively charged region of physical anode. This produces “collapsing” wavefronts that frequently collide and neutralize one another and thereby are less likely to result in a sustained arrhythmia (Fig. 1.24). This theory incorporates many aspects of the above-mentioned mechanisms.

To summarize and to put defibrillation theory into clinical perspective, the effects of the application of a voltage gradient across myocardium are a function of field strength and timing. Although the biological effects of shocks may overlap, this concept is summarized in Fig. 1.25. Extremely low energy pulses may have no effect on the myocardium. Stronger pulses (in the microjoule range), such as those used for cardiac pacing, result in action potential generation in non-refractory myocardium, which leads to a propagating impulse. With increasing electric field strength (to the 1-J area), VF can be induced with shocks delivered during the vulnerable period. Increasing the shock strength above the ULV (and above the defibrillation threshold) puts the shock in the defibrillation zone. Very high-energy shocks can lead to toxic effects, including disruption of cell membranes, postshock block, mechanical dysfunction and new tachyarrhythmias.

**Antitachycardia pacing**
The concepts of basic myocardial function also explain the mechanism of arrhythmia termination with ATP.
As an example, in monomorphic ventricular tachycardia (VT) late after myocardial infarction, a re-entrant circuit utilizing abnormal tissue adjacent to an infarct is responsible for the arrhythmia (Fig. 1.26). For the re-entrant circuit to perpetuate itself, the tissue immediately in front of the leading edge of the wavefront must have recovered excitability so that it can be depolarized (Fig. 1.26). ATP—delivered as a short burst of pacing impulses at a rate slightly greater than the tachycardia rate—can terminate VT by depolarizing the tissue in the excitable gap, so that the tissue in front of the advancing VT wavefront becomes refractory, preventing further arrhythmia propagation (Fig. 1.26B). The ability of a train of impulses to travel to the site of the re-entrant circuit and interrupt VT depends on several factors,
including the site of pacing (the closer to the circuit entrance, the greater the likelihood of circuit penetration and termination), the length of the tachycardia cycle, and the size of the excitable gap. With delivery of ATP, faster and more remote circuits with smaller excitable gaps are generally more difficult to terminate and have a greater risk of degeneration to less organized tachyarrhythmias, including fibrillation.

To treat VT, ATP is delivered through a right ventricular lead in ICDs. ATP has been applied successfully to treat slow VT (<188–200 bpm, success rate 78–91%)23, and recently fast VT (200–250 bpm, success rate 72–81%)23. These therapy success rates are reinforced by the observation that ATP did not result in an increased risk of acceleration of the arrhythmia, syncope, or mortality in comparison with patients who receive defibrillation shocks only.24 Patients with ATP, rather than those programmed to defibrillation shocks only, also report statistically higher quality of life scores. If ATP fails, or if the frequency of the VT is too high to apply ATP, the device diverts immediately to deliver a defibrillation shock. The use of ATP in the ventricle is important in limiting shocks, and is further discussed in Chapter 8. This chapter will also address the empiric use of ATP that may directly impact future appropriate shock therapies.
In addition to VT, atrial fibrillation and tachycardia occur frequently in patients with cardiac dysfunction, ventricular arrhythmias, and in patients with sinus node dysfunction. ATP for atrial arrhythmias is also successful, with atrial tachycardia termination rates from 40 to 50%. In addition to termination of the arrhythmia episode, ATP is also associated with an overall reduction in atrial tachycardia/atrial fibrillation burden. Due to the absence of studies demonstrating clinically significant improvements with atrial ATP, its adoption in clinical practice has been modest. This may evolve with further studies.

The mechanisms underlying the success and failure of ATP are not fully understood. One theory is that ATP failure may occur when the pacing electrode is located too far from the re-entry core, and therefore unable to terminate the arrhythmia orthodromic wavefront. However, this failure mechanism remains controversial. For example, a comparison of left vs. right ventricular ATP in induced VT showed both sites were equally effective, which raises questions regarding a location-dependent limitation. Nevertheless, a recent study examined the potential therapy modification of biventricular anti-tachycardia pacing rather than right ventricular anti-tachycardia pacing to see if spatially distributed leads would advance the orthodromic wavefront and increase the likelihood of arrhythmia termination. Although biventricular anti-tachycardia pacing was found to be superior in a rabbit model in terminating VT, there was also a theoretical increased risk of VT acceleration. This risk was not observed clinically in a study of patients who underwent cardiac resynchronization and ICD implantation, in which a significantly higher number of successful VT termination episodes were observed when biventricular ATP was used. Next, the MIRACLE ICD trial showed that biventricular ATP improved VT termination, including those VTs that were classified as fast. Finally, the ADVANCE CRT-D trial is an ongoing prospective trial that will examine the efficacy of right ventricular vs. biventricular ATP to terminate all types of VT.

A promising new approach that is founded on the concepts of ATP is to deliver a low-voltage shock to "unpin" re-entry from its stationary core. The method relies on the effect of virtual electrode polarization, which predicts hyperpolarization and depolarization on opposite sides of functional or anatomical heterogeneity that can result in secondary sources of excitation. When a low voltage shock is properly timed, all possible re-entry cores are simultaneously excited, which effectively destabilizes and unpinns a re-entrant arrhythmia. In an experimental model using rabbits, the unpinning method terminated VT in all preparations, in comparison with 63% of preparations treated with standard ATP only. Although 35% of the preparations treated with unpinning first also required ATP, the study data suggested that this potential therapy was as effective as or potentially more effective than ATP for terminating stable, pinned re-entrant arrhythmias. Although promising, the role of unpinning in clinical practice is not yet established.

Measuring the efficacy of defibrillation

Threshold and dose–response curve
At the time of defibrillator insertion, it is important to determine whether the system implanted can successfully terminate fibrillation. A measure frequently used to assess the ability of a system to terminate VF is the defibrillation threshold (DFT). The term “threshold” suggests that there is a threshold energy above which defibrillation is uniformly successful and below which shocks fail (Fig. 1.27A). The multitude of factors that affect whether a shock will succeed—patient characteristics, fibrillation duration, degree of ischemia and potassium accumulation, distribution of electrical activation at the time of the shock, circulating pharmacological agents, and others—result in defibrillation behavior that is best modeled as a random variable, with a calculable probability of success for any given shock strength. Thus, defibrillation is more accurately described by a dose–response curve, with an increasing probability of success as the defibrillation energy increases (Fig. 1.27B). The curve can be characterized by its slope and intercept, and specific points on the curve can be identified, such as \( D_{50} \), the energy dose with a 50% likelihood of success. Factors adversely affecting defibrillation shift the curve to the right, so that a higher dose of energy is required to achieve a 50% likelihood of success, and improvements in defibrillation (such as superior lead position and improved waveforms or lead design) shift the curve to the left (Fig. 1.28). Because of the large number of fibrillation episodes required to define a curve (30–40 inductions), the dose–response curve is not determined in clinical practice, but it remains a useful research tool and conceptual framework.
Relationship between defibrillation threshold and dose–response curve

If defibrillation is best described as a dose–response curve, where on the curve does the DFT exist (i.e. what is the probability of successful defibrillation at the clinically used DFT energy)? The probability of successful defibrillation at the DFT energy depends on the steps taken to define the threshold. Consider a step-down to failure DFT, in which shocks are delivered beginning at a relatively high energy (e.g. energy with a 99% success rate) and decremented by several joules with each VF induction until a shock fails (at which point a rescue shock is delivered). The DFT in this protocol is defined as the lowest energy shock that succeeds (Fig. 1.29).

Since the initial energies tested are at the upper end of the dose–response curve, successive shocks may have a 98%, 95%, 88%, 85% (and so on) likelihood of success, depending on the starting energy and size of the steps taken. Despite the fairly high likelihood of success for each shock individually, the sheer number of shocks delivered in this range on average result in a shock failing (thus defining the DFT) at a relatively high point on the curve. If this process is repeated many times, a population of DFTs is created, with a mean and expected range. In humans, step-down to failure algorithms

![Fig. 1.27 Defibrillation “threshold.” (A) The expected response to shock if a true threshold value existed. In reality, the likelihood of success is a sigmoidal dose–response curve, as shown in (B). The ED$_{50}$ is the energy dose with a 50% likelihood of success, and so on.](image1)

![Fig. 1.28 Use of dose–response curve to measure effects of an intervention on defibrillation efficacy. The graph shows the effect of thoracotomy on defibrillation in a canine model. The “immediate” group had defibrillation threshold testing done immediately after thoracotomy. Note that the curve is shifted to the right and that the energy with a 50% probability of success is 27 J, compared with 15 J for the “delayed” group, which was allowed 48–72 h of recovery before defibrillation testing. Defibrillation is more effective in the “delayed” group because the probability of success at a given energy is higher in this group. Thus, the curves graphically display diminished defibrillation efficacy immediately after thoracotomy. (From Friedman PA, Stanton MS. Thoracotomy elevates the defibrillation threshold and modifies the defibrillation dose–response curve. J Cardiovasc Electrophysiol 1997; 8:68–73. By permission of Futura Publishing Company.)](image2)
have a mean DFT with likelihood of success near 70%, but with a standard deviation near 25%. Thus, the likelihood of success of a shock delivered at the DFT energy of 10 J is 70%. Now, if the DFT process were repeated, it is possible that the second shock might fail on one occasion (defining the DFT as 10 J) or that all four shocks might succeed on another occasion (and that a lower energy shock would fail to define the DFT), and so on. Thus, repeating the DFT determinations may result in different values for the DFT with each determination. However, if enough repetitions were performed, a population of DFTs, as shown in (B), would be created. The most commonly observed DFT in this example would be 10 J, which has a 70% likelihood of success. Further details in text.

Fig. 1.29 Step-down to failure defibrillation threshold (DFT) testing. In this hypothetical example (A), four shocks are required to define the DFT. The first shock is delivered at 20 J and is successful (S). The next shock, delivered at 15 J, also succeeds. A 10 J shock succeeds, and a 5 J shock fails (F), defining the DFT at 10 J (the lowest successful energy). Note from the curve that the likelihood of success at the DFT energy (10 J) is 70%. Now, if the DFT process were repeated, it is possible that the second shock might fail on one occasion (defining the DFT as 20 J) or that all four shocks might succeed on another occasion (and that a lower energy shock would fail to define the DFT), and so on. Thus, repeating the DFT determinations may result in different values for the DFT with each determination. However, if enough repetitions were performed, a population of DFTs, as shown in (B), would be created. The most commonly observed DFT in this example would be 10 J, which has a 70% likelihood of success. Further details in text.

Defining an implantation safety margin
Given that a DFT determination is an estimated point on the dose–response curve and that the probability of successful defibrillation at the DFT is approximately 70% with the commonly used step-down protocol, a safety margin must be added to the DFT energy to increase the odds of success. Although all device shocks could be programmed to deliver the maximum available energy, using a lower energy that can consistently terminate fibrillation has advantages. These include faster charge time and more prompt delivery of therapy (with reduced chance of syncope), battery preservation, diminished risk of AV block, decreased myocardial damage in the regions with the highest voltage gradient, and diminished risk of impaired postshock sensing. These benefits must be weighed against the morbidity accrued by the requirement of a second shock and consequences of an extended VF with incremental doses of energy until a first success occurs, which defines the DFT. In this case, despite the fairly low likelihood of success at each low-energy shock, if enough shocks are delivered, one is likely to succeed, defining the DFT. With this protocol, the mean DFT has a likelihood of success near 30%. Iterative increment–decrement DFT or binary search algorithms that begin in the middle zone of the curve have been shown to approximate the ED50, the energy with a 50% probability of success. In this type of protocol, if the first shock defibrillates the heart, the first shock of the next fibrillation episode uses a lower energy. If the first shock does not defibrillate the heart, a second shock at a higher energy is delivered.

Regardless of the DFT protocol, a DFT determination is best conceptualized as a means of approximating a point on the dose–response curve, with the specific point estimated being a function of the DFT algorithm chosen. DFT determinations can be very useful tools for assessing defibrillation efficacy. Triplicate DFT measurements, which can be performed with fewer than 10 fibrillation episodes, have been demonstrated to be as reproducible as the true logistic regression model of the dose–response curve and to have less variability than other models used to estimate dose–response curves. Thus, determination of a DFT before and after an intervention (such as initiation of a drug or movement of a lead to a new position) can determine whether defibrillation efficacy is enhanced or impaired by the intervention.
period of ventricular tachyarrhythmia. Thus, the energy programmed should be a value high enough above the DFT to ensure that the shock is on the “plateau” of the dose–response curve, where success rates exceed 90%, but not necessarily at maximum output. In humans, adding 10 J to the DFT has been shown to result in first-shock success rates of 99.5±4.3%.\(^{22,23}\) If one shock fails, two of three successful shocks at a 10-J safety margin have been shown to predict an annual rate of sudden death of <1%.\(^{24}\) Strategies using one defibrillation shock, or using no VF inductions, are emerging and increasingly used in practice, and discussed below.

### Defibrillation testing at implantation

With the information known about the human defibrillation dose–response curve and defibrillation models, a practical approach to implantation testing can be used. Step-down to failure DFT testing can be done with three or four episodes of fibrillation. However, given the high likelihood of successful implant with modern active can, biphasic, implantable systems, strategies using fewer shocks to assess the safety margin are increasingly popular. In our practice, we typically employ an approach that requires two VF inductions (discussed below).

For step-down to failure testing, external defibrillation pads are placed before the surgical implantation procedure begins. Testing is done with the device in the surgical pocket and with leads connected. The high-voltage lead impedances are measured to insure appropriate lead connections. Standard ICDs can deliver programmable energies up to 30–35 J. Higher-energy devices, with outputs as high as 40 J, are also available. The first-shock energy is programmed to 10 J less than the maximum output of the device, and fibrillation is induced. If the test shock is successful, the first-shock energy is lowered by 5 or 6 J, and after a delay of 3–5 min fibrillation is induced again and the new energy tested. This iterative decremental process is continued until the first shock fails or until an energy of 5 or 6 J succeeds (at which point the DFT is often defined as ≤5 or 6 J). The lowest successful energy is taken as the DFT, and the first shock of the device is chronically programmed to the DFT energy plus 10 J. Often during testing, the second defibrillator shock is programmed to an energy equal to the last successful shock energy plus 10 J, and rescue is performed by the defibrillator (rather than externally). Thus, after a 15-J shock is successful, the first shock is programmed to 10 J for the next induction, and the second device shock is programmed to 25 J [which is the current lower boundary for the DFT (15 J) plus a 10-J safety margin].

Although step-down to failure testing is still occasionally used in our practice, we more commonly employ a technique utilizing two VF inductions. The first shock is set to 10 J less than the maximum device output. If successful, rather than stepping down by 5–6 J, for the second induction the first shock is programmed to 14 or 15 J, and the second shock is programmed to the same as the first shock. If the first shock succeeded, the approximate “DFT” is said to be ≤15 J, and if the second shock succeeds, the DFT is defined as that energy (typically 25 J). In our experience, patients with an active can, pectoral, biphasic DFT <15 J have a very low risk of subsequent inadequate defibrillation, and no additional testing is performed until the time of pulse generator change out.\(^{26}\) In patients in whom the DFT approximation is higher, additional testing may be done at implant or, more commonly, annually until a chronically stable DFT is confirmed. Two successes at an energy 10 J less than the maximum device output are required to achieve a 10-J safety margin. If these are not achieved, system modification is required, as discussed below.

Some experts recommend a second strategy that emerged from the low energy safety study (LESS) trial.\(^{26}\) In a substudy of the LESS trial, Higgins et al.\(^{97}\) reported that a single conversion success at 14 J on the first ventricular induction yielded a similar positive predictive accuracy (91%) as the commonly accepted approach of two successes at 17 J or 21 J in determining a successful outcome with a device that provided 31 J. However, an approach that utilized successes at 21 J provided the highest combination of positive and negative predictive accuracy (98.8% and 100%, respectively). Nonetheless, a subset analysis by Gold et al.\(^{98}\) showed that the results were durable, in that those patients in whom the VF induction test was successful with a first 14-J shock at implantation, regardless of additional induction tests, had similar long-term VF conversion success rates as all ICD recipients when the device was programmed to provide 31 J.

If an adequate safety margin is not demonstrated, a common next step is to reverse the shock polarity (waveform and polarity are discussed in greater detail below). In Table 1.2, potential options are provided if
an adequate safety margin is not demonstrated. Currently available devices also allow the programming of multiple potential configurations to alter the shock vector [e.g. exclusion of the superior vena cava coil, or of the can (particularly if it is placed in the right chest), etc.]. If implantation criteria are still not met, some devices permit waveform pulse width adjustment (discussed below). Alternatively, a high-output device is used, leads are repositioned if it is thought that lead position can be improved, or an additional endovascular lead is added in systems that permit it. If these approaches fail, a subcutaneous lead is added (see Chapter 5 for implantation technique). Using biphasic waveforms, we have found that subcutaneous leads are required in only 3.7% of devices implanted.99

In a single-center observational study of three types of subcutaneous leads (single-element subcutaneous array electrode, three-finger electrodes, subcutaneous patch electrodes), all types performed well without a significant change in defibrillation threshold observed.100 Although there was no significant difference in complications, 7.3—9.5% of patients developed a major complication (predominantly lead fracture). Therefore, with use of a subcutaneous ICD lead, patients require close follow-up with routine chest radiographs.

There are many factors that may result in elevated defibrillation threshold. These include drug therapy, underlying cardiac disease, the size, configuration and number of defibrillating leads, the time that VF persists before shock delivery, ischemia, hypoxia, amplitude of the VF waveform, temperature, heart weight, body weight, direction of the delivered shock and waveform, and chronicity of lead implantation.101 In patients with inherited channelopathies, such as Brugada syndrome, high defibrillation thresholds may be prevalent and problematic.102 In one series of patients who received a high-output generator for an elevated defibrillation threshold, the majority had underlying coronary artery disease, with reduced left ventricular function, and were on amiodarone.103 An important finding in this study was that in patients with high defibrillation thresholds who receive an ICD, arrhythmia death remained a significant long-term risk (42% of the deaths were arrhythmia related).

An interesting observation is that there is a circadian variation in the defibrillation threshold. The defibrillation threshold has a morning peak that is 16% higher than that measured after noon.103 In addition, the first failed shock rate is more often in the morning compared with other times during the day. This variability in defibrillation threshold is clinically important in patients with high thresholds, in whom a 10-J safety margin becomes more difficult to achieve.

Finally, with the advent of newer ICD technologies that utilize different waveforms and allow various shock configurations, some investigators have begun to ask whether DFT testing is required. In general the likelihood of a high DFT is low. However, in one contemporary observation study > 6% of patients required modification of their ICD system due to an inadequate safety margin.104 In some patients DFT testing is postponed due to other comorbid illness, such as an individual with atrial fibrillation and not on anticoagulation. These patients may account for up to 5% of patients who undergo ICD implantation. In considering the decision to perform DFT testing, other potential benefits of the study must be considered. For example, DFT testing may identify lead dysfunction, demonstrates appropriate sensing and charging of the device, and in general test the complete system integrity.105 These benefits must be

### Table 1.2 Options in a patient with high energy requirements or an inadequate safety margin at defibrillation threshold testing.

<table>
<thead>
<tr>
<th>Option</th>
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<tr>
<td>Reverse the shock polarity</td>
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<tr>
<td>Change shock configuration (example tip-to-generator, ring-to-generator, tip-to-coil)</td>
</tr>
<tr>
<td>Waveform modification if available with the generator</td>
</tr>
<tr>
<td>Exchange the generator to a “high-output” device</td>
</tr>
<tr>
<td>Exclude if possible drugs that increase the defibrillation threshold</td>
</tr>
<tr>
<td>Add a superior vena cava coil</td>
</tr>
<tr>
<td>Add a subcutaneous array or patch</td>
</tr>
<tr>
<td>Move the generator to a left pectoral position if located on the right</td>
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weighed against the risks of the procedure. Contraindications to ICD implant testing have been published, and are listed in Table 1.3.

**Upper limit of vulnerability to assess safety margin**

As previously discussed, the ULV is the lowest energy above which shocks delivered during the vulnerable period do not induce fibrillation. Numerous studies have demonstrated that the DFT and ULV are strongly linked. Since the DFT and ULV are correlated, some investigators have suggested that ULV determinations could be performed to assess defibrillation efficacy with one or no fibrillation episode. During sinus rhythm, test shocks are delivered at the peak of the T wave at initially high energies, with the energy level subsequently decreased in steps until fibrillation is induced, defining a shock that is below the ULV. Since the ULV may be dependent on the coupling interval, energies are also delivered at various intervals before the T-wave peak to "scan" repolarization. For conventional biphasic waveforms, the ULV corresponds to a 90% successful energy level, and it has been used to provide adequate safety margins at cardioverter defibrillator implantations and for long-term follow-up in clinical protocols. Furthermore, since ULV assessment is an indirect measure of defibrillation efficacy, the relationship between the ULV and the DFT may be affected by numerous factors, including electrode configuration, pharmacological agents, and the protocol used to determine the ULV. Important in the patient population that receive an ICD, acute ischemia may reduce the ULV. This phenomenon is felt to be due in part to conduction failure during acute ischemia. In some situations, the changes in ULV may not accurately reflect defibrillation efficacy. Since ULV may result in device testing with no VF induction, the R wave should be $\geq 7 \text{mV}$ to insure adequate sensing of VF. In the small subset of patients with ULV $> 20 \text{J}$, some experts advocate performing DFT testing at implant. Because of the indirect nature of the ULV--DFT relationship and the large body of clinical and experimental data based on DFTs, ULV testing has only been adopted as routine clinical practice in a few centers. If future ICDs adopt automatic ULV testing (in which the device would scan the T wave and determine appropriate shock timing), this technique may become more widespread due to its ability to assess defibrillation efficacy without VF inductions in many patients and the possibility of automated testing by the ICD.

**The importance of waveform**

The shape of a defibrillating waveform can dramatically affect its defibrillation efficacy. In the canine model, for example, Schuder et al. demonstrated that for trans-
In thoracic defibrillation, an ascending ramp waveform has a much higher success rate with the same delivered current than does a descending ramp (Fig. 1.30). This has been confirmed in a human study of 63 patients in which a 7-ms ascending ramp waveform significantly reduced delivered energy (18%) and voltage (24%) at DFT. However, because of the importance of using physically small circuits for implantable devices, a capacitor discharge, which more closely resembles the descending ramp is employed in devices.

Creating the defibrillation waveform
As in pacing, the battery serves as the source of electrical charge for cardiac stimulation in defibrillation. Before a high-energy shock can be delivered, the electrical charge must be accumulated in a capacitor, because a battery cannot deliver the amount of required charge in the short time of a defibrillation shock. A capacitor stores charge by means of two large surface area conductors separated by a dielectric (poorly conducting) material, and capacitor size is an important determinant of implantable defibrillator volume, typically accounting for approximately 30% of device size. If fluid analogies are used for electricity—voltage as water pressure and current as water flow (i.e. liters per minute)—the capacitor is analogous to a water balloon, which has a compliance defined by the ratio of volume to pressure. To increase the amount of water put into the balloon, one can increase the pressure or, alternatively, use a balloon with a greater compliance (more stretch for a given amount of pressure). Similarly, the charge stored can be increased by increasing capacitance or by applying greater voltage. The trend in implantable devices has been toward smaller capacitors to create smaller devices.

The charge stored by a capacitor is defined by

\[ \text{Charge} = \text{capacitance} \times \text{voltage} \]

The voltage waveform of a capacitor discharged into a fixed-resistance load (Fig. 1.31A) is determined by

\[ V(t) = V_i \cdot e^{-t/RC} \]

and the energy associated with the waveform is given by

\[ \text{Energy} = 0.5 CV^2 \]

Since the “tail” of the waveform in longer pulses (≥ 10 ms) refibrillates the ventricle (most likely accounting for the superiority of the ascending ramp seen by Schuder et al.), truncated waveforms have been used clinically. The classic monophasic truncated waveform is shown in Fig. 1.31B. The waveform is characterized by the initial voltage (\(V_i\)), the final voltage (\(V_f\)), and the pulse width or tilt. Tilt is an expression of the percentage decay of the initial voltage. The tilt of a waveform is a function of the size of the capacitor used, the resistance of the leads and tissues through which current passes, and the duration of the pulse. Tilt is defined by the percentage decrease of the initial voltage:

\[ \text{Tilt} = (V_i - V_f)/V_i \times 100\% \]

As shown in Fig. 1.31, tilt can have an important effect on defibrillation efficacy, with progressive improve-
ment in defibrillation efficacy with decreasing tilt, for a trapezoidal waveform of constant duration. For monophasic waveforms formerly used clinically, the optimal tilt was 50–80%.

**Biphasic waveforms**

Appropriately characterized biphasic shocks can result in significant improvement in defibrillation efficacy, with reductions in DFTs of 30–50%. All currently available commercial defibrillators use biphasic waveforms; a typical biphasic waveform is shown in Fig. 1.31 C. Biphasic waveforms have numerous clinical advantages, all stemming from their improved defibrillation efficacy. Biphasic waveforms have been shown to result in higher implantation success rates due to their lower DFTs, which are associated with higher safety margins. Since safety margins are increased, most patients do not require high-energy shocks, and smaller devices can be designed. The improved efficacy of biphasic waveforms permits a greater tolerance in electrode positioning than that required for monophasic waveforms, facilitating the implanting procedure. Additionally, biphasic shocks have been shown to result in faster postshock recurrence of sinus rhythm and to have greater efficacy than monophasic shocks at terminating VF of long duration.

With the development of biphasic defibrillation waveforms the energy required for defibrillation has been reduced. Simultaneously, advances in capacitor and battery technology have allowed for a reduction of pulse generator size. Further advances that will reduce the generator size will occur when the energy required for defibrillation is reduced.
Phase duration and tilt
In most commercially available ICDs, pulse duration and tilt are pre-set to values found to be optimal based on experimental evidence (Figs 1.32 and 1.33). Some devices do permit individualization of the pulse widths, based on the concept that individual variations in cellular time constants result in varying optimal pulse durations. Anecdotal observations and small studies support pulse width optimization in high DFT patients. With few studies that specifically address this concept, individualized variation for optimization in patients with a high DFT requires further study.

Polarity and biphasic waveforms
Polarity is an important determinant of monophasic defibrillation, with lower DFTs found for transvenous systems when the right ventricular electrode is the anode (+). The results of studies of biphasic polarity are less uniform, with some reports showing an effect of biphasic polarity and others indicating no effect. However, all studies demonstrating a polarity effect have found that waveforms with a first phase in which the right ventricular electrode is the anode (+) are more effective. Additionally, biphasic polarity has the greatest effect on patients with elevated DFTs. In a study of 60 patients, use of biphasic waveforms with a right ventricular anodal first phase resulted in a 31% reduction in DFT in patients with DFT ≥15 J, whereas polarity made no difference in patients with DFTs < 15 J. Despite the fairly uniform population improvement in DFT with a ventricular anodal first phase polarity among studies in which an effect was seen, there is clearly individual variability, so that if an adequate safety margin cannot be found in a patient, a trial of the opposite polarity is reasonable, regardless of the initial polarity tested.
Mechanism of improved efficacy with biphasic waveforms

Several theories have been proposed to explain the observed superiority of biphasic over monophasic waveforms. None provides a complete explanation for the benefits seen, and the fundamental mechanism remains to be determined. However, important basic observations have been made.

Ascending ramp waveform

Mathematical models that predict defibrillation efficacy suggest that use of an ascending ramp waveform may improve efficacy of defibrillation. The waveform uses an ascending ramp phase over a predetermined time interval in the first phase.\textsuperscript{116,131-133} In animal models, ascending ramp waveforms were more effective than truncated exponential waveforms.\textsuperscript{134} In a recent randomized trial, patients were divided into two groups, one with a 12-ms ascending first phase and the other with a 7-ms ascending first phase. In those patients randomized to the 7-ms ascending first phase, the energy and voltage required at DFT were significantly reduced in comparison with the other group.\textsuperscript{116}

First phase as “conditioning” pulse

Successful defibrillation requires sodium channel activation at a time when cells are ordinarily not receptive to physiological stimulation. The first phase of a pulse may serve to hyperpolarize tissue near the anode, thereby reactivating otherwise inactive sodium channels. This conditioning pulse facilitates excitation by the following pulse.\textsuperscript{135}

Refractory period shortening

The first phase of a biphasic pulse may shorten the refractory period of myocardial cells. This transient shortening may then facilitate the effective recruitment of sodium channels by the second phase of the pulse. This ultimately extends the duration of the action potential and the refractory period, important putative mechanisms for defibrillation.\textsuperscript{136}

Membrane stabilization

In addition to being more effective and requiring a lower potential gradient for defibrillation, biphasic waveforms are less toxic than monophasic waveforms. In higher voltage gradient regions, membrane disruption and myocardial damage may result from the shock. However, higher voltage gradients are required to produce these toxic effects with biphasic waveforms than with monophasic waveforms. Deleterious postshock effects may be due to membrane microlesions, which permit indiscriminate exchange of ions. The reversal of polarity during the shock may expedite membrane reorientation and repair, decreasing postshock dysfunction.\textsuperscript{137}

Measuring shock dose

All the discussion to this point has described the shock dose in terms of energy (joules). As noted above, the shape of the waveform is a function of the initial voltage, the size of the capacitor, and the resistance of the load. If a smaller capacitor is used to diminish device size, a larger initial voltage may be needed to deliver an equivalent amount of charge into the fibrillating tissue. Thus, two waveforms may have different leading edge voltages, but the same energy if there are differences in capacitance (Fig. 1.34). Therefore, the question of how to determine the “dose” of a shock arises. It is clearly important, because shocks of insufficient dose fail to terminate fibrillation and excessively strong shocks can lead to proarrhythmia or myocardial injury. The “dose” of defibrillation is usually given in units of energy (joules) on the basis of tradition and ease of measurement. Physiologically, however, energy has little bearing on defibrillation; the voltage gradient is the factor that affects membrane channel conductance, and at the tissue level several decades of animal and human research have shown current to be the most important factor for generating action potentials and for defibrillation.\textsuperscript{69} To add to the complexity, energy
can be described as the stored energy—the amount of energy stored in the capacitor before shock delivery—or the energy delivered. Since the waveforms are truncated, usually around 10% of the stored energy is not delivered. Additionally, although the term is used clinically, “delivered energy” is highly variable, depending on where the delivery is recorded; energy delivered at the lead surface is not the same as energy delivered only a few millimeters into the tissue. Some device manufacturers, in fact, simply report an arbitrary percentage of the stored energy as the delivered energy. Stored energy, although not a direct indicator of the factors responsible for biological defibrillation, indicates the size of the device necessary to generate a given energy shock. Over the range of clinically utilized capacitor size and biological tissue resistance in a given system, a change in energy up or down is reflected by a similar change in voltage and current. In practice, “energy” is the most commonly used term to indicate shock dose.

**Use of waveform theory in clinical practice**

The optimal biphasic waveform is specific to the device, lead, and patient. In many commercially available devices, the only programmable option is the polarity. Therefore, if a patient undergoing implantable defibrillator insertion does not have an adequate defibrillation safety margin, a logical next step is reversal of polarity. If an adequate safety margin is still not met, a lead is often added (discussed below). Tilt or duration can also be modified as an alternative next step in systems that offer this feature.

**Lead system and defibrillation**

The most efficient lead system is one that evenly distributes the shock over the myocardium and minimizes the difference in potential between high-gradient and low-gradient zones. This is best accomplished with large contoured epicardial patches positioned so that an imaginary line connecting the centers of the electrodes passes through the ventricular center of mass. However, since epicardial leads require thoracotomy for placement, they are typically used after other approaches have been exhausted.

Although intrinsically less efficient, transvenous lead systems can now be used almost universally because of the adoption of biphasic waveforms (discussed above) and the introduction of defibrillators in which the pulse generator shell is an active electrode. Because the surface area of the pulse generator is large, the addition of the generator shell as an active electrode reduces the biphasic endocardial DFT by 30% compared with that of a dual-coil defibrillation lead alone. When an active can system with a single distal defibrillation coil is used, addition of a proximal coil has further lowered the DFT in some, but not all, studies. Nevertheless, if implantation safety margins cannot be achieved despite waveform modification (reversal of polarity and, if available, adjustment of pulse width), adding a second lead with the electrode positioned near the junction of the right atrium and superior vena cava is a logical next step. Alternatively, since most leads in use today have two coils, in a subset of patients defibrillation is improved when the proximal coil is removed from the defibrillation circuit. This observation probably results from individual anatomical variations such that the proximal coil may lessen the field strength over the left ventricle. Anecdotally, use of three right ventricular coils (placement of a dual coil lead in the apex, and use of adapter to place a single coil lead in the outflow tract, with passage of shock from the two distal coils to the proximal coil and can) may help in high DFT situations, although this has not been validated. If adequate safety margins cannot be achieved despite optimal deployment of endovascular leads, subcutaneous patches or arrays, which further significantly increase defibrillation electrode surface area and can favorably direct greater current through the ventricles, can lead to successful implantation. With biphasic active-electrode pulse generators, the addition of subcutaneous leads is required in only 3.7% of patients (Fig. 1.33). When they are required, arrays may be more effective than patches, though we found that this benefit was blunted in biphasic systems.

As noted above, defibrillation efficacy is improved with optimal lead positions, although the effectiveness of biphasic waveforms, the large surface area of the pulse generator, and programmability that has allowed multiple potential vectors to be used that take advantage of the geometry of the leads and can, have permitted tolerance of less than perfect positions. Generally, defibrillation effectiveness diminishes as the right ventricular electrode is placed in a progressively proximal position, toward the tricuspid valve. Therefore, this lead should be placed as apically as possible. Additionally, a septal location, to direct as much of the electrical field over the left ventricular mass as possible, is desir-
able. Active pulse generator shell permits independent positioning of a proximal defibrillation coil, and the proximal lead position can be near the superior vena cava, near its junction with the right atrium, or in the left subclavian vein (Fig. 1.36).

Since in nearly all commercially available defibrillators the pulse generator shell serves as an electrode, its position can also affect defibrillation efficacy. Implantable defibrillators are most commonly placed in the left pectoral region, typically in the pectoral (subcutaneous) plane. However, the site of pulse generator placement and vascular access is influenced by multiple factors, including patient and physician preference, anatomical anomalies, previous operations, integrity of the vascular system, and whether a preexisting permanent pacing system is present. In addition to factors specific to the patient, choice of the implantation site can affect ease of technical insertion, defibrillation effectiveness, and long-term rates of lead failure.

Fig. 1.35 Effect of waveform on frequency of subcutaneous (SQ) lead use. On the ordinate is the frequency of subcutaneous lead usage, and on the abscissa are the subgroups analyzed. In 45 of 94 (48%) patients with monophasic systems, subcutaneous leads were required to meet implantation criteria. In contrast, only 17 of 460 (3.7%) biphasic systems required subcutaneous leads to meet implantation criteria. (From Trusty et al. By permission of Futura Publishing Company.)

Fig. 1.36 Chest radiographs depict active pulse generator shell system with an added proximal defibrillation coil to optimize defibrillation threshold.
Right pectoral implantation may be considered in left-handed persons, hunters who place the rifle butt on the left shoulder, and patients with previous mastectomy, other surgical procedures, or anatomy that precludes left-sided insertion. In systems with both distal and proximal defibrillation coils, the proximal coil is either shifted toward the right hemithorax (if both coils are on the same lead) or, often, advanced to a lower superior vena cava position for greater cardiac proximity (in two-lead systems) with right-sided placement. With active can pulse generators, the largest defibrillation lead surface, the device shell, is shifted away from the ventricular myocardium (Fig. 1.37). These unfavorable restrictions on lead position decrease defibrillation effectiveness.\textsuperscript{143,144} With biphasic waveforms, we found that right-sided implantation results in a 6-J increase in DFT compared with left-sided placement (11.3±5.3 J, left-sided; 17.0±4.9 J, right-sided; \textit{P}<0.0001).\textsuperscript{143} Even with the increase, right-sided devices were successfully placed in 19 of 20 patients; in one patient, an acceptable right-sided threshold could not be achieved and that approach was abandoned. Despite the concern that a right-sided active can might be detrimental by diverting a significant portion of the electrical field away from the ventricles, the large surface area of the shell compensates for this, so that when right-sided implantation is required, active can devices are preferable (Fig. 1.38).\textsuperscript{143}

In general, however, left-sided insertion is superior to right-sided placement and is used if there are no compelling factors against it.

An alternative site for device placement is the abdomen, but this site is only rarely used. Although not as effective for defibrillation as the left pectoral position, the abdomen appears superior to the right pectoral lo-

Fig. 1.37 (A) Posteroanterior and lateral chest radiographs from a patient with a left-sided defibrillator. Note that the proximal defibrillation lead is in the left subclavian vein. (B) Posteroanterior and lateral chest radiographs from a patient with right-sided defibrillator placement. Note that the proximal defibrillation lead is in the superior vena cava.
ciliation for active can placement. However, abdominal insertion is technically more challenging, requiring two incisions, lead tunneling, abdominal dissection (often necessitating surgical assistance), and general anesthesia. Additionally, because of the greater risk of infection, threat of peritoneal erosion and increased risk of lead fracture, even with totally transvenous systems this position is used only in rare circumstances.

**Drugs and defibrillators**

Antiarrhythmic agents are frequently used in patients with implantable defibrillators to treat supraventricular arrhythmias (particularly atrial fibrillation), suppress ventricular tachyarrhythmias, and slow VT to increase the responsiveness of antitachycardia pacing. In the implantable defibrillator trials, concomitant use of membrane-active agents (Vaughn-Williams class I or class III drugs) has ranged from 11% to 31%.

Several important device–drug interactions must be considered:

1. Detection. Most drugs slow VT. If slowed below the detection cut-off rate, VT is not detected by the device and remains untreated. Initiation of antiarrhythmic drugs in patients with VT is usually followed by device testing to assess detection of VT.
2. Pacing thresholds. Bradycardia and antitachycardia pacing thresholds may be affected by pharmacological agents, as discussed in Chapter 13.
3. Pacing requirements. Drugs may exacerbate conduction defects or slow the sinus rate, necessitating pacing for bradycardia.
5. Changes in DFT. Although it is well known that pharmacological agents can modulate defibrillation effectiveness, drug–defibrillation interactions are complex. Moreover, assessment of the influence of drugs on defibrillation is confounded by the effects of anesthetic agents, variability in lead systems and waveforms across studies, and heterogeneity in study subjects (i.e. human, canine and porcine). In general, however, agents that impede the fast inward sodium current (such as lidocaine) or calcium channel function (such as verapamil) increase the DFT, whereas agents that block repolarizing potassium currents (such as sotalol) lower the DFT. The effects of amiodarone are legion; clinically, long-term administration of amiodarone increases DFTs, whereas intravenous administration has little immediate effect. In addition to antiarrhythmic agents, other drugs have been shown to increase the defibrillation threshold, such as sildenafil, venlafaxine and alcohol.

Importantly, with current generation biphasic ICDs, the clinical effect of most drugs, including amiodarone, is modest. In general, then, ICD evaluation should be performed when administration of membrane active drugs that can increase the threshold (especially amiodarone) is initiated, particularly in patients with borderline DFTs. Drug effects on defibrillation are summarized in Table 1.4. In patients with a low DFT, testing for slow VTs or, less commonly, empirically lengthening the detection interval (to allow for VT slowing) is most important. As a general rule, ICD evaluation should be considered whenever administration of Vaughn-Williams class I or class III drugs is initiated or their dosage significantly increased. These drugs are listed.
in Table 1.5. Drug and defibrillator interactions are also discussed in Chapter 13.

It is equally important to remember that use of cardiovascular medications outside of membrane active drugs (i.e. use of ACE-inhibitors, angiotensin receptor blockers, β-blockers, statins, aspirin, warfarin, and other evidence-based medications have been shown to reduce mortality in various clinical situations) does not interact with ICD function in any clinically significant way, and should therefore be encouraged.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Class*</th>
<th>Effect on defibrillation threshold†</th>
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<tbody>
<tr>
<td>Quinidine</td>
<td>IA</td>
<td>Increase</td>
</tr>
<tr>
<td>Procainamide</td>
<td>IA</td>
<td>No change</td>
</tr>
<tr>
<td>N-acetylprocainamide</td>
<td>IA</td>
<td>Decrease</td>
</tr>
<tr>
<td>Disopyramide</td>
<td>IA</td>
<td>No change</td>
</tr>
<tr>
<td>Mexiletine</td>
<td>IB</td>
<td>Increase</td>
</tr>
<tr>
<td>Flecainide</td>
<td>IC</td>
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</tr>
<tr>
<td>Moricizine</td>
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<td>Increase</td>
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<td>Propafenone</td>
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<tr>
<td>Propranolol</td>
<td>II</td>
<td>Increase</td>
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<tr>
<td>Atenolol</td>
<td>II</td>
<td>No change</td>
</tr>
<tr>
<td>Isoproterenol</td>
<td></td>
<td>Decrease</td>
</tr>
<tr>
<td>Sotalol</td>
<td>III</td>
<td>Decrease</td>
</tr>
<tr>
<td>Ibutilide</td>
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<td>Decrease</td>
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<tr>
<td>Dofetilide</td>
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<tr>
<td>Intravenous</td>
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<td>Increase</td>
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<tr>
<td>Verapamil</td>
<td>IV</td>
<td>Increase</td>
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*Vaughn-Williams classification.
†If study results conflict, the most frequently reported effect is noted.
Modified from Carnes et al. By permission of Pharmacotherapy Publications.

### References


### Table 1.5 Membrane-active drugs*

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<tr>
<th>Vaughan-Williams classification</th>
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<tr>
<td>IA</td>
<td>Quinidine, procainamide, disopyramide</td>
</tr>
<tr>
<td>IB</td>
<td>Lidocaine, tocainide, phenytoin</td>
</tr>
<tr>
<td>IC</td>
<td>Flecainide, propafenone, encainide, moricizine</td>
</tr>
<tr>
<td>III</td>
<td>Sotalol, ibutilide, dofetilide, amiodarone</td>
</tr>
</tbody>
</table>

*These agents may significantly affect defibrillator function, often mandating device testing on initiation.
CHAPTER 1 Clinically Relevant Basics of Pacing and Defibrillation


Clinical Relevant Basics of Pacing and Defibrillation

90 Brady PA, Friedman PA, Stanton MS. Effect of failed defibrillation shocks on electrogram amplitude in a non-integrated transvenous defibrillation lead system. Am J Cardiol 1995; 76:580–4.


CHAPTER 1 Clinically Relevant Basics of Pacing and Defibrillation


Our understanding of the hemodynamic consequences of cardiac pacing has evolved dramatically over past decades. Dual-chamber pacing, rate-responsive pacing, rate-adaptive and differential atrioventricular (AV) intervals, alternative-site pacing, ventricular pacing avoidance algorithms, ventricular rate regularization and cardiac resynchronization therapy are all attempts to mimic and/or restore normal cardiac conduction and physiology.

Application of the appropriate pacing therapy first requires understanding of normal physiology, the various interrelated components contributing to the normally functioning cardiovascular system, and the effects of cardiac and noncardiac diseases on these individual components as well as on function of the whole. Given that our understanding of cardiac function in normal and abnormal conditions is incomplete and that current technology is imperfect, the goal to mimic perfectly the normal cardiovascular system under all conditions has yet to be met. Nevertheless, hemodynamic pacing continues to attract intense interest as technological advances bring us closer to that goal.

**Cardiovascular physiology**

Challenge to the cardiovascular system, such as exercise or emotion, usually results in an increase in cardiac output, which is determined by heart rate and stroke volume. The relative contribution of each is variable and in part determined by age, the type and intensity of activity, baseline cardiovascular conditioning, and whether there is underlying cardiac or noncardiac disease (Fig. 2.1).

The cardiovascular demands incurred with exercise are usually met primarily by an increase in heart rate and secondarily by increases in stroke volume. Aerobically trained athletes can increase stroke volume proportionally more, thus enabling them to reach the same cardiac output with a smaller increase in heart rate. Stroke volume is defined as the amount of blood ejected with each ventricular contraction, i.e. end-diastolic volume minus end-systolic volume. In the normal heart, end-diastolic volume depends on end-diastolic filling pressure, total blood volume, distribution of that blood volume, and atrial systole (preload). End-systolic volume depends on myocardial contractility and afterload. The Frank-Starling law relates the degree of left ventricular (LV) filling pressure to cardiac output at various degrees of contractility (Fig. 2.2).

All of these relationships are modulated by metabolic alterations, autonomic tone, pharmacological agents, and the cardiac rhythm. For example, the increase in sympathetic tone associated with an increase in heart rate decreases the AV interval. Antiarrhythmic drugs can increase or decrease the heart rate at rest and in response to exercise, either by a direct effect on the sinus node or by effects on the autonomic nervous system.

**Abnormal physiology**

A large segment of the pacing population has cardiac disease or other comorbidities that affects cardiac performance. These conditions can be characterized as those affecting heart rate, those affecting stroke volume, and those affecting both.

Chronotropic incompetence (an inadequate heart rate increase with exercise or stress) may be due to isolated sinus node dysfunction, autonomic dysfunction, or drugs. Individuals with normal LV function may be asymptomatic at rest but experience symptoms with activity, depending on activity level, comorbid conditions (such as pulmonary disease), and the severity of chronotropic incompetence. Patients with significant
LV dysfunction may be less tolerant of chronotropic incompetence because their impaired stroke volume makes them more dependent on heart rate to maintain cardiac output. Patients may be unaware of how symptomatic they are unless objectively evaluated.

Myocardial contractility may be impaired by coronary artery disease, myocardial infarction, non-ischemic cardiomyopathy, valvular disease, or pericardial disease. Patients with LV dysfunction regardless of cause are more dependent on preload and afterload to maintain optimal stroke volume. Many of these patients have associated conduction system disease, such as sinus node dysfunction, AV nodal disease, or His-Purkinje disease. Atrioventricular dissociation and inter- and intraventricular dyssynchrony can worsen already impaired myocardial performance. Interventricular dyssynchrony refers to activation of the right and left ventricles at different times; intraventricular dyssynchrony refers to temporal delay in mechanical contraction of different left ventricular segments. It has been the focus of extensive study, as it adversely impacts clinical heart failure. Metabolic abnormalities, such as chronic acidosis, hypoxia and hypercarbia, may depress cardiac performance. Patients with severe LV dysfunction may also have autonomic dysfunction that further limits the ability of the heart to increase heart rate and stroke volume with physiological stress.
Many individuals with LV dysfunction have a number of associated comorbidities, e.g. concomitant renal failure, diabetes mellitus, coronary artery disease, hypertension, chronic obstructive pulmonary disease and many others, all of which may affect indices of preload, afterload and autonomic function as well as directly impair myocardial contractility (Fig. 2.3). Drugs used in the treatment of these conditions, atrial fibrillation, and coronary artery disease may further affect these functions and directly suppress intrinsic conduction. Understandably, determining which patients will benefit from hemodynamic pacing techniques and which pacing technique will most benefit any individual patient is complex and incompletely understood.

**Basics of hemodynamic pacing**

**Chronotropic response**

Appropriate heart rate response during exercise, i.e. chronotropic competence, is the most important contributor to cardiac output, especially at moderate or extreme degrees of exercise (Fig. 2.4). At rest and at lower levels of activity, AV synchrony contributes significantly to achieving an appropriate cardiac output (Fig. 2.5). Because many paced patients are at the lower end of the activity curve most of the time and a significant proportion of these patients are also dependent on adequate preload because of decreased ventricular compliance, AV synchrony is perhaps just as important as rate responsiveness for achieving optimal cardiovascular hemodynamics in the typical patient. Restoration of both rate responsiveness and AV synchrony should be the goal of physiological pacing and should be viewed as complementary.

**Atrioventricular dissociation and ventriculoatrial conduction**

The earliest indication for pacing was complete heart block, and ventricular-only pacing was the only mode available. Establishing a stable ventricular rhythm was lifesaving and overshadowed the fact that normal car-
Cardiac function was not reestablished. However, some patients experienced hemodynamic decline with this mode of pacing. Later it was established that hemodynamic impairment could be caused by ventriculoatrial conduction and atrial contraction against a closed AV valve, which could result in pacemaker syndrome.5 Ventriculoatrial conduction can activate mechanical stretch receptors in the walls of the atria and pulmonary veins (Fig. 2.6). Vagal afferents transmit these impulses centrally, and reflex peripheral vasodilation results. In addition, various neurohormonal agents, such as atrial natriuretic peptide, are activated. Pacemaker syndrome may be manifested by a variety of symptoms and physical signs (Table 2.1 and Fig. 2.7). Pacemaker syndrome was initially identified as a complication of VVI pacing; however, it may occur with any pacing mode when there is AV dissociation. It may also occur in persons with a markedly prolonged AV delay, with atrial systole effectively occurring concurrently with or after ventricular systole.

The prevalence of pacemaker syndrome is difficult to determine and depends in part on how it is identified. Studies evaluating objective clinical impairment with pacing in a nontracking mode suggest that the incidence may be in the range of 7–10%.5 In a substudy of the MOST trial, investigators noted the development of ‘severe’ pacemaker syndrome in approximately 20% of patients paced in the VVIR mode. Improvement was
CHAPTER 2  Hemodynamics of Device Therapy

Table 2.1  Pacemaker syndrome

<table>
<thead>
<tr>
<th>Potential symptoms</th>
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<tr>
<td>Weakness</td>
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<tr>
<td>Chest pain</td>
</tr>
<tr>
<td>Syncpe or near-syncpe</td>
</tr>
<tr>
<td>Dyspnea</td>
</tr>
<tr>
<td>Cough</td>
</tr>
<tr>
<td>Neck pulsations</td>
</tr>
<tr>
<td>Apprehension</td>
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<td>Abdominal pulsations</td>
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</table>

<table>
<thead>
<tr>
<th>Potential physical findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypotension</td>
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<tr>
<td>Congestive heart failure</td>
</tr>
<tr>
<td>Cannon “a” waves</td>
</tr>
<tr>
<td>Blood pressure decline during ventricular pacing</td>
</tr>
<tr>
<td>Decrease in cardiac output and arterial pressure</td>
</tr>
<tr>
<td>Increase in peripheral vascular resistance during monitoring</td>
</tr>
</tbody>
</table>

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noted with reprogramming to a dual-chamber pacing mode. However, in an older crossover study of patients with pacing in each of the DDD and VVI modes for a week in randomized order, 83% of subjects experienced some degree of pacemaker syndrome with pacing in the VVI mode. This finding suggests that when patients have a basis for comparison, they are more aware of symptoms of pacemaker syndrome (Fig. 2.8).

Atrioventricular synchrony

The contribution of AV synchrony to maintaining physiological cardiac performance is well established. AV synchrony is estimated to increase stroke volume by as much as 50% and in normal hearts may decrease left atrial pressure and increase cardiac index by as much as 25–30%. Although patients with normal ventricular function may have the greatest absolute degree of improvement with restoration of AV synchrony, a greater degree of relative improvement is typical in patients with severe left ventricular systolic dysfunction (Table 2.2). In these patients, any improvement derived from appropriately timed atrial systole may be beneficial.

Mitral valve closure and diastolic filling are influenced by the timing of atrial and ventricular contraction. Identifying the optimal AV interval for a given patient can be difficult. Regardless of whether the AV interval is programmed too long or too short, optimal AV interval timing leads to premature mitral closure. If the AV interval is too long, ventricular contraction does not immediately follow atrial emptying. Thus, the atrioventricular valves “float” back towards the atria, resulting in near closure of these valves prior to the onset of ventricular systole. This results in a soft first heart sound and, when extreme, regurgitation of blood through the atrioventricular valves during diastole (diastolic AV regurgitation). If the AV interval is unduly short, ventricular contraction and closure of the AV valves occurs before completion of atrial emptying. Since in patients with cardiomyopathy and heart failure there can be considerable differences in the timing of right and left ventricular contraction and relaxation, optimal AV closure for one side of the circulation may not be ideal for the other. Patients with severe diastolic dysfunction benefit even more with appropriately timed atrial systole, because dependence on optimal preload is even greater to maintain satisfactory cardiac output.

The influence of pacing mode on factors indirectly but importantly related to cardiovascular performance...
has also been studied. P-synchronous pacing has been shown to result not only in significantly higher cardiac outputs than VVI pacing, but also in lower systemic vascular resistance, lower serum lactate levels, smaller AV oxygen gradients, and lower levels of circulating vasoactive peptides and norepinephrine.

Mechanical AV delay varies between paced and sensed atrial beats because of the intrinsic delay in atrial
activation after atrial pacing, i.e. intra-atrial conduction. The absolute intra-atrial conduction delay varies significantly among patients and also depends on underlying conduction or myocardial disease (Fig. 2.9). The right intra-atrial conduction time is measured from the beginning of the P wave, or the intracardiac signal recorded in the upper right atrium, to the onset of atrial depolarization in the para-His bundle region. The normal right intra-atrial conduction time is usually between 30 and 60 ms. Interatrial conduction time, measured from the beginning of the P wave or depolarization in the upper right atrium to the onset of left atrial depolarization, is recorded at the level of the distal coronary sinus. The interatrial conduction time is generally between 60 and 85 ms. Taking the inter- and intra-atrial delay into consideration and programming the differential AV interval accordingly will result in improved hemodynamics.9

Since intra-atrial conduction delay varies from patient to patient and the relative timing of left and right atrial contraction depends on the actual site in the atrium of earliest activation, it can be very difficult to consistently predict when atrial systole will actually be complete for both atria. Added to this, the extent of intra-atrial conduction delay is dependent on the site of activation (pacing site within diseased/scarred area, etc.) during atrially paced complexes, further complicating the issue. Clues on the electrocardiogram that suggest echocardiographic atrioventricular optimization may be particularly helpful include a P wave duration > 120 ms, an absent or negative PR segment, and notching/isoelectric periods during the inscription of the P wave. In practice, with the exception of cardiac resynchronization devices, echocardiographic optimization is rarely performed. Programming the AV interval is discussed further below.

In addition to independently programmable paced and sensed AV intervals, dual-chamber pacemakers provide rate-adaptive AV intervals. Conduction time through the AV node normally decreases due to sympathetic nervous system activity with physiological increases in heart rate, resulting in shorter AV intervals at higher heart rates. Any variation in heart rate has been demonstrated to result in an immediate, precise and inversely proportional variation in the AV interval in normal hearts.10 A linear relationship exists between heart rate and the AV interval, independent of age or baseline PR interval11 (Fig. 2.10). In patients with conduction system disease or autonomic dysfunction, the AV delay may not shorten with heart rate increase. Rate-adaptive AV interval attempts to mirror normal physiology and allow a higher maximal tracking rate, and has been shown to improve hemodynamic indices during exercise compared with those with a fixed AV interval.10,12

Optimization of the AV interval has been a source of frustration for many years. When dual-chamber pacemakers were first introduced and only a fixed AV interval was possible, very simple programming guidelines were followed. In general, if the patient had intact

![Fig. 2.9 A differential atrioventricular interval (AVDI) attempts to correct for the timing differences between a paced and a sensed atrial event. When the atrium is paced, the atrioventricular interval (AVI) begins with delivery of the pacing artefact. However, there is latency between delivery of the pacing artefact and actual depolarization. Depending on interatrial conduction time, the paced-sensed difference can be great. In this diagram, the AVI is programmed to 200 ms for each event, but the effective AVI is 160 ms after the sensed atrial event and 240 ms after the paced atrial event. (From Janosik et al.7 By permission of the American College of Cardiology.)](image-url)
AV conduction, the AV interval was programmed long enough to allow intrinsic conduction. If the patient had AV block, the AV interval was programmed to mimic what was considered to be a normal PR interval, i.e. 150–200 ms. However, this outdated approach fails to account for the previously described mechanical intra-atrial delay from atrial pacing to atrial depolarization, the effect of any interatrial conduction delay and the deleterious effects of right ventricular (RV) apical pacing that may be avoided if a significantly longer AV interval allows intrinsic conduction to occur. In addition, optimization of the AV interval becomes a significantly more important hemodynamic issue with the introduction of cardiac resynchronization therapy.

**AV optimization**

Optimizing the AV interval serves to optimize left ventricular preload. Selection of the best AV interval often depends on echocardiographic or invasive hemodynamic measurement. Patients who require biventricular systems typically have abnormal and variable intra-atrial and intraventricular conduction. This makes the prediction of left-sided atrial ventricular mechanical delay difficult using right atrial pacing. Thus, a given AV interval will result in markedly different left-sided AV mechanical delays in different patients based on their individual intra-atrial and intraventricular conduction delay. The site of both ventricular and atrial leads also impacts optimization of AV timing. For example, an atrial lead placed on the intra-atrial septum with a left ventricular lead placed near the base of the left ventricle will give rise to a completely different AV mechanical interval than a right atrial appendage position used with LV apical pacing, even though the AV interval is set similarly.

The rationale for cardiac resynchronization therapy is primarily to normalize the ventricular activation sequence and coordinate septal and free wall contraction, thereby improving cardiac efficiency. Although this primary utility of ventricular resynchronization is independent of atrioventricular conduction and mechanical AV delays, an incremental benefit above that achieved with ventricular resynchronization has been demonstrated with a range of ideal AV delays, and others have demonstrated the effects of suboptimal AV delays in heart failure. With long AV delays, there is a suboptimal contribution of atrial systole. This gives rise to diastolic mitral regurgitation and limits the diastolic filling that occurs as a result of active atrial systole. Shortening the AV delay decreases the amount of diastolic mitral regurgitation and dilated cardiomyopathy, thereby decreasing pulmonary capillary wedge pressures, etc. Conversely, an AV delay that is too short gives rise to a suboptimally shortened filling period for the left ventricle and thus decreased preload and cardiac output.

The degree and extent of intra-atrial conduction delay in patients with heart failure and abnormal heart is highly variable. In the normal state, sinus impulses from the right atrial/superior vena caval junction reach the left atrium primarily through the roof of the atrium (Bachmann’s bundle) and secondarily through the fossa ovalis and the musculature of the coronary sinus. Once the impulse reaches the atria, a distinct sequence of activations involving predictable differences in activation of the posterior atrium, left atrial appendages, and pulmonary vein are observed. While this intra-atrial conduction is occurring, conduction via the AV node to the ventricle is also occurring. In patients without bundle branch block there is near-simultaneous activation of the right and left mid endocardial surfaces of the intraventricular septum. With AV pacing using a right atrial appendage lead and a right ventricular apical lead configuration, increased intra-atrial conduction delay may give rise to near-simultaneous left atrial and left ventricular activation, producing the equivalent of a left-sided pacemaker syndrome. On the other hand, placement of the atrial lead in the Bachmann’s bundle region in a patient with...
insignificant intra-atrial conduction delay, but with significant conduction delay from the right ventricular pacing site to the left ventricle, will cause marked prolongation of the left-sided mechanical AV interval. Intra-atrial conduction delay also affects the AV interval during atrial sensing. During atrial tracking, right atrial events are sensed after the onset of atrial depolarization. In some patients sinus activation occurs first on the septal side of the right atrial superior vena cava junction. There may be marked intra-atrial delay from this site to the right atrial appendage where the pacing lead is required. In these patients, by the time the atrial event is sensed, left atrial activation may be ongoing or even completed, giving rise to a very long left atrium to left ventricle mechanical delay with usual AV timing.

Thus, the location of the atrial, the right ventricular and the left ventricular leads, the magnitude and difference between intra-atrial conduction and intraventricular conduction delay and the lead function at any given time (atrial pacing vs. sensing) all impact LA/LV mechanical intervals in a manner that is difficult to predict.17

Left ventricular end-diastolic pressures may change significantly based on the actual AV delay, and this change is largely independent of the site of ventricular pacing.17 Left ventricular contractility as measured as dP/dt is also affected by the AV delay, and this effect is incremental to the benefit seen with left ventricular-based pacing over right ventricular pacing.19 In the PATH-CHF trial a hemodynamic benefit as seen with increased LV maximal dP/dt and increased impulse pressure was demonstrated with either left ventricle or biventricular pacing in comparison with right ventricular-based pacing. The effect, however, was seen best at atrial ventricular delays between 25 and 75% of the intrinsic PR interval.14 Butter has also shown a benefit of left ventricular free-wall pacing when compared with an anterior site in the left ventricle when measuring a percent increase in dP/dt. This effect was also optimal at AV delays between 50 and 100 ms and prolongation of the AV delay showed a decrease in this beneficial effect on contractility irrespective of the site of pacing.19

Principles of echocardiographic AV optimization

The AV or PR interval is usually simply measured from either the atrial pacemaker artifact or from the start of the P wave to the beginning of the QRS complex. As previously noted, the mechanically relevant AV interval is the time between mechanical atrial contraction and ventricular contraction. The echocardiographic parameter most useful in studying the filling characteristics of the left ventricle is the mitral valve inflow Doppler velocity. The mitral valve inflow pattern in sinus rhythm is biphasic. Distinct filling waves can be recognized (Fig. 2.11). The first is the early filling wave (E wave). This represents blood flow into the left ventricle during diastole. The velocity and magnitude of this flow are dependent primarily on the relaxation characteristics of the left ventricle. The second distinct wave is the A wave, which occurs only in sinus rhythm and is from active atrial contraction. Because of electromechanical delays in the atrium and the ventricle there is a distinct interval between the start of the P wave and the start of the A wave measured by mitral Doppler inflow. In fact, the QRS complex itself is usually inscribed typically before the start of the A wave. When the PR interval/AV interval is short, the QRS is inscribed early and this results in aortic ejection and mitral valve closure occurring (forced by ventricular contraction) before complete inscription of the atrial Doppler inflow (truncation of the A wave) (Fig. 2.12) Thus, with short AV delays, diastolic flow is limited and the full benefit of atrial contraction is not obtained. This is important in
all patients with congestive heart failure (CHF), but extremely important in patients with significant diastolic dysfunction/relaxation abnormality. In these patients, because of problems with ventricular relaxation, the E wave is limited and a greater portion of diastolic filling is from atrial systole and the A wave. Conversely, with a long AV interval (PR interval) the A wave is completed. However, a significant gap between the end of atrial filling (A wave) and the beginning of ventricular contraction and aortic ejection occurs. During this delay the mitral valve has passively closed (soft first heart sound) and diastolic mitral regurgitation may occur. Thus, in an optimal AV interval the atrial filling wave has completed and there is no excessive delay between this completion and the beginning of aortic ejection. Most methods of echocardiographic AV optimization use this principal. In one technique the mitral inflow Doppler velocity and aortic outflow are continuously monitored echocardiographically. During this monitoring the AV interval is first set at or about the patient’s intrinsic PR interval (long AV interval). The AV interval is then progressively shortened until truncation of the atrial and flow wave is just seen. This AV interval with or without a small positive offset is taken as the optimized AV interval. Using this principal that optimal AV delay results when spontaneous mitral valve closure occurs at about the same time as forced closure (ventricular contraction) and therefore aortic ejection, Ritter has described the following method.20 Initially a short AV delay (AV1) is programmed and the interval between the onset of the QRS to the end of the truncated A wave (QA1) is measured (Fig. 2.13). Next there is a long AV delay (AV2) that maintains ventricular pre-excitation but allowing mitral valve closure before aortic ejection is

**Fig. 2.12** The situation when the P-R interval is too short. Now ventricular systole (mechanical contraction of the ventricle) occurs even before complete emptying of the atrium (truncation of the A wave). Thus in a mechanical sense the patient is the equivalent of atrial fibrillation (no effective hemodynamic contribution of the atrium).


Fig. 2.13 Echocardiographic example for atrioventricular (AV) delay optimization. With the short AV delay, truncation of the A wave is seen. Gradually lengthening the AV delay takes away the truncation while further lengthening (long AV delay) allows diastolic mitral regurgitation (see text). (Data from Ritter P, Dib JC, Mahaux V, et al. PACE 1995; 18:855 (abstract). Reproduced with permission from Blackwell Publishing.)

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- Program short AV delay (AVshort), forcing closure of mitral valve
- Note premature shortening of A-wave, reducing diastolic filling prior to aortic out-flow (ao); QApre is time from QRS onset to the premature end of the A-wave

- Program long AV delay (AVlong), maintaining ventricular pre-excitation but allowing spontaneous MV closure
- Note delay between MV closure and start of systole marked by beginning of isovolumic contraction period; QA sport is the time from QRS onset to the spontaneous end of the A-wave
measured (Fig. 2.13). The interval between the start of the QRS to the end of the non-truncated A wave is noted (QA2). The optimal AV interval according to the Ritter calculation is AV\textsubscript{opt} = AV\textsubscript{short} + d where
\[ d = (AV_{\text{long}} - AV_{\text{short}}) - (QA_{\text{pre}} - QA_{\text{spont}}) \]
(Fig. 2.14). Thus, the greater the difference in the AV interval that allows complete inscription of the A wave and the short AV interval the larger the optimal AV interval will be. On the other hand, a large difference between the start of the QRS and the end of the A wave when allowing for complete AV inscription (long atrial filling) will result in the optimal AV interval being short. Echocardiographic images demonstrating use of the Ritter method are demonstrated in Fig. 2.15.

Ishikawa has proposed an alternative method for AV optimization\textsuperscript{21} (Fig. 2.16). In this somewhat simpler method, a slightly prolonged AV delay is set. From this number is subtracted the interval between the end of the A wave and complete closure of the mitral valve. This interval from the end of the A wave to the beginning of aortic ejection or complete closure of the mitral valve is the duration of diastolic mitral regurgitation. Thus, from a single long AV interval the optimal AV interval can be calculated. The steps for using the Ishikawa method would be to set a long AV interval and measure the mitral Doppler inflow. The interval between the end of the complete A wave and the beginning of aortic ejection can be measured. This measurement is subtracted from the long AV interval.

Several observations with regard to AV optimization in the PATH-CHF study also allow for relatively simpler AV interval optimization. Patients with a wide QRS complex demonstrated shorter optimal AV delay sensations with a narrow QRS complex. This is likely to be because intraventricular conduction delay is more prominent than interatrial conduction delay. The optimal average AV interval for patients with a QRS > 150 ms was 43% of the intrinsic AV interval. On the other hand, for patients with a normal or narrower QRS, optimal AV intervals were about 80% of the intrinsic AV interval. This, of course, will be affected by the position of the left ventricular lead. For example, if the left ventricular lead is located at a site causing early aortic ejection, then a longer AV interval despite the wider QRS will be required.

Acute hemodynamic studies suggest that optimal ventricular contractility is further enhanced in individual patients when a patient’s specific AV interval is programmed. It has been shown that there is a close correlation between impedance-measured AV interval and echo Doppler-derived AV interval and externally applied impedance signals are a relatively straightforward bedside method to adjust the AV interval.\textsuperscript{22-24}

A major weakness that remains is the inability to optimize the AV interval during exercise. Optimization of the AV interval at rest does not reflect the optimal AV interval during exercise. New technology is being assessed which has potential for AV interval optimization during stress in cardiac resynchronization systems.\textsuperscript{25}

While adaptive algorithms to optimize the AV interval during exercise are a step forward, several problems
Fig. 2.15 Echocardiographic images demonstrating the Ritter method for atrioventricular (AV) interval optimization. The upper left panel obtained with a short AV interval; upper right panel obtained with a long AV interval; lower panel demonstrates optimal separation of E and A waves.

Fig. 2.16 Ishikawa method for atrioventricular (AV) optimization (see text). (Reproduced from Ishikawa T et al.21 with permission from Blackwell Publishing.)
remain. The actual optimal AV interval during exercise may vary with loading conditions on the heart, e.g., when overdiuresis or incipient heart failure occur. Even with real-time physiological parameter monitoring as part of a feedback system, the optimal AV interval may be difficult to determine, as no single parameter may be representative of all factors that need to be considered. The optimal AV interval for diastolic function is difficult to determine; only measuring mitral inflow Doppler measurements and making sure that all of atrial contraction has contributed to diastole is probably too simplistic. Ventricular stretch, efficiency of ventricular relaxation, and optimization of early filling may involve different AV interval optimizing algorithms from systolic function optimization. Even if the AV interval has been carefully optimized, if radiofrequency ablation is performed in the atrium, antiarrhythmic drugs are used, or myocardial infarctions have occurred, a completely different relative atrial and ventricular timing may result. Another compounding variable is optimal RA-RV timing. The operator may perfectly optimize LA-IV timing, only to find that there is significant diastolic tricuspid regurgitation giving rise to hepatic engorgement and right-sided heart failure.

**Effect of pacing mode on morbidity and mortality**

An early study of the effect of pacing mode on morbidity and mortality\(^\text{26}\) paved the way for intense clinical interest in and subsequent clinical trials on the effect of pacing mode on morbidity and mortality.\(^\text{26}\) and the potential adverse effects of VVI pacing (Figs 2.17 and 2.18). In this early study, at 4 years of follow-up, atrial fibrillation had occurred in 47% of the patients receiving VVI pacing, but in only 7% of those receiving AAI pacing (\(P<0.0005\)); CHF occurred in 37% of the VVI group and in 15% of the AAI group (\(P<0.005\)); and mortality was 23% in the VVI group and 8% in the AAI group (\(P<0.025\)).

Many other investigators have performed retrospective reviews to assess the effect of pacing mode on mortality. Despite the inherent weaknesses of retrospective analyses, it is difficult to dismiss the similar finding among all the studies of significantly lower mortality with DDD or AAI pacing than with VVI pacing and significantly lower incidences of atrial fibrillation.\(^\text{27}\)

Survival was assessed in a large population of patients (20,948) with sinus node dysfunction.\(^\text{28}\) This random sample was from the complete United States cohort of Medicare patients receiving pacing for sinus node dysfunction in 1988 through 1990. The DDD/
DDDR pacing mode was an independent correlate of survival.

Prospective trials assessing the effect of pacing mode on morbidity and mortality are summarized in Table 2.3. Andersen et al. published the first prospective data on pacing mode and survival. (This trial is referred to as the Andersen Trial or the Danish Pacer-Tach Trial.) Among 225 patients (mean age 76 years) with sinus node dysfunction randomized to AAI or VVI pacing, the incidence of atrial fibrillation was higher in the VVI group (AAI group 14%; VVI group 23%; \( P = 0.12 \)) and the incidence of thromboembolism was also higher in the VVI group than in the AAI group (\( P = 0.0083 \)). Although no difference in mortality could be detected at the initial analysis at 3.3 years, subsequent analysis at 5.5 years showed improved survival and less heart failure in the AAI group. In addition, there was a persistent reduction in the incidence of atrial fibrillation and thromboembolic events. This trial stands alone in demonstrating lower mortality with physiological pacing. This may be explained by the fact that the physiological pacing mode implemented was AAI mode. With AAI pacing, the patient maintains intrinsic AV conduction; perhaps more important, this avoids the abnormal depolarization pattern of right ventricular pacing that would occur with VVI or DDD pacing.

In a smaller trial, paroxysmal atrial fibrillation occurred more frequently with VVI pacing than with DDD pacing. However, the Pac-A-Tach trial found no significant difference in recurrence of atrial tachyarrhythmias by intention to treat at 1 year—48% in DDDR and 43% in VVI.

The Pacemaker Selection in the Elderly (PASE) trial, a prospective, randomized, single-blind trial, compared DDDR and VVIR pacing modes. There was no statistically significant difference in quality of life between DDDR and VVIR pacing modes, but there was a trend toward improved quality of life in patients with sinus node dysfunction randomized to dual-chamber pacing. Perhaps more significant was a crossover of 26% of patients from ventricular pacing to dual-chamber pacing because of pacemaker syndrome.

The Canadian Trial of Physiologic Pacing (CTOPP) compared VVIR with DDDR or AAIR and had primary end-points of overall mortality and cerebrovascular accidents and secondary end-points of atrial fibrillation, hospitalizations for CHF, and death due to a cardiac cause. CTOPP demonstrated that physiological pacing (DDDR/AAI) was associated with a reduced rate in the development of chronic atrial fibrillation, from 3.78% to 2.87% per year, at the 3-year analysis. No significant improvement in quality of life or mortality was demonstrated with dual-chamber pacing. However, there was a slight divergence of the mortality curves favoring dual-chamber pacing. In addition, quality of life was improved in subsets of patients. These included patients who were pacemaker-dependent and patients with severe diastolic or systolic dysfunction.

The United Kingdom Pacing and Cardiovascular Events (UKPACE) trial compared DDD with VVI pacing modes in patients ≥70 years old who required permanent pacing for second- or third-degree AV block. UKPACE demonstrated no significant difference between pacing modes in the primary end-point of all-cause mortality or in the composite secondary end-point of cardiovascular deaths, atrial fibrillation, heart failure hospitalizations, cerebrovascular accidents or thromboembolic events, and reoperation.

Of the major trials assessing the effect of pacing mode on morbidity and mortality, the Mode Selection Trial (MOST) and subsequent substudies have probably had the most profound effect on the practice of pacing. MOST randomized 2010 patients with sinus node dysfunction to either VVI or DDD pacing. The primary end-points were all causes of mortality and cerebrovascular accidents. This trial failed to demonstrate any difference in mortality, but did demonstrate a lower incidence of atrial fibrillation with physiological pacing and reduced the signs and symptoms of heart failure. Quality of life was slightly improved. The rates of hospitalization for heart failure and of death, stroke, or hospitalization for heart failure were not significant in unadjusted analyses, but with adjusted analyses became marginally significant. The study concluded that, overall, dual-chamber pacing offers significant improvement compared with ventricular pacing.

Substudies of the MOST trial and the DAVID (Dual Chamber and VVI Implantable Defibrillator) trial have demonstrated the adverse effects of right ventricular apical pacing when no ventricular pacing was required, i.e. the patient had intact AV conduction at some AR interval.

The DAVID trial was designed to assess the effect of dual-chamber pacing vs. back-up ventricular pacing in patients with an internal cardioverter-defibrillator (ICD) indication but no indication for anti-bradycardia pacing, no history of recurrent atrial arrhythmias...
### Table 2.3 Trials assessing the effect of pacing mode on morbidity and mortality

<table>
<thead>
<tr>
<th>Study</th>
<th>Patient inclusion criteria</th>
<th>End-points</th>
<th>Treatment arms</th>
<th>Key results</th>
</tr>
</thead>
</table>
| Danish study30   | Sick sinus syndrome requiring pacing | Mortality, CV death, AF, TE events, Heart failure, AV block | AAI pacing (n = 110) vs. VVI pacing (n = 115) | • Cumulative incidence of CV death, PAF, chronic AF, and TE events lower with AAI pacing  
• Less severe heart failure with AAI  
• Multivariate analysis: AAI associated with freedom from TE events, survival from CV death |
| PASE33           | Age ≥ 65 years Need for PPM to prevent or treat bradycardia | QOL, All-cause mortality, First nonfatal CVA or death, First hospitalization for CHF, AF, PM syndrome | Single-blind, randomized, controlled comparison; VVIR pacing vs. DDDR pacing | • QOL improved significantly, but no difference between pacing modes  
• 26% of patients with VVIR crossover to DDDR due to PM syndrome  
• Trends of borderline statistical significance in end-points favoring DDDR in patients with SND |
| CTOPP34          | Initial PM Life expectancy > 1 year Not in chronic AF | Cardiovascular mortality or stroke, Paroxysmal or chronic AF, Hospitalization for CHF, QOL 6-min walk | DDDR/R or AAI/R pacing vs. VVIR/R pacing | • No difference in QOL, VVI vs. DDD/AAI  
• No statistically significant difference in mortality or stroke  
• No difference in hospitalizations  
• 24% decrease in incidence of chronic or paroxysmal AF with DDD/AAI |
| MOST105          | SND requiring PM NSR or atrial standstill at time of implantation | Stroke, Health status, Cost effectiveness, Total mortality, CV mortality, AF, Heart failure score, PM syndrome | DDDR vs. VVIR | • Lower incidence of AF with DDDR  
• No difference in any other end-point |
| UKPACE36         | Age ≥ 70 years High-grade AV block requiring PPM | All-cause mortality | DDDR (50%) vs. VVIR (25%) vs. VVI (25%) | • No significant difference between pacing modes in the primary end-point of all-cause mortality or in the composite secondary end-point of cardiovascular deaths, atrial fibrillation, heart failure |
| DANPACE106       | Tachycardia-bradycardia syndrome with normal AV conduction | All-cause mortality, CV mortality, Incidence of AF and TE events, QOL, Cost-effectiveness | AAIR vs. DDDR | • In progress |

AF, atrial fibrillation; AV, atrioventricular; CHF, congestive heart failure; CTOPP, Canadian Trial of Physiologic Pacing; CV, cardiovascular; CVA, cardiovascular accident; DANPACE, Danish Pacing Trial; MOST, Mode Selection Trial; NSR, normal sinus rhythm; PAF, paroxysmal atrial fibrillation; PASE, Pacemaker Selection in the Elderly; PM, pacemaker; PPM, permanent pacemaker; QOL, quality of life; SND, sinus node dysfunction; TE, thromboembolic; UKPACE, United Kingdom Pacing and Cardiovascular Events.
and a LV ejection fraction of $\leq 40\%$. Patients were randomized to effectively no pacing, VVI-backup pacing at 40 bpm, or DDDR pacing with a lower rate of 70 bpm. Dual-chamber pacing offered no advantage and actually increased the combined end-point of death or hospitalization for heart failure (Fig. 2.19).

Investigators from the MOST trial demonstrated that in patients with a normal baseline QRS duration, cumulative percent of ventricular pacing is a strong predictor of hospitalization for heart failure, and the risk of atrial fibrillation also increased linearly with cumulative percent of ventricular pacing. This occurs even when AV synchrony is preserved (Figs 2.20 and 2.21).

These findings led to the development and widespread use of pacing algorithms that avoid ventricular pacing. Rather than placing a pacemaker capable only of atrial pacing and therefore not providing back-up ventricular pacing in the event of AV block, ventricular pacing avoidance algorithms may dynamically alter the AV interval to allow intrinsic ventricular depolarization, and other algorithms may allow one or more P waves to occur without a subsequent pacemaker output in an effort to promote intrinsic ventricular depolarization (Figs 2.22 and 2.23).

The potential advantage of extending RV pacing avoidance to cardiac resynchronization therapy (CRT) has also been studied. In one investigation, synchronized LV pacing, i.e. eliminating RV stimulation, produced acute LV and systemic hemodynamic benefits similar to biventricular pacing. This pacing configuration provided superior RV hemodynamics compared with biventricular pacing.}

![Figure 2.19 Survival in the DAVID trial. Survival was significantly improved in patients in whom ventricular pacing was avoided. DDDR mode vs. VVI mode and composite end-point of death or new/worsening heart failure hospitalization. In DDDR group, patient who survived to 3 months’ follow-up had worse 12-month event-free rates when % of RV pacing was >40% (P = 0.09). (Reprinted with permission from Wilkoff BL, Cook JR, Epstein AE et al. Dual-chamber pacing or ventricular backup pacing in patients with an implantable defibrillator: the Dual Chamber and VVI Implantable Defibrillator (DAVID) Trial. JAMA 2002; 288:3115–23. Copyrighted and used with permission of Mayo Foundation for Medical Education and Research.)](image)

![Figure 2.20 Sweeney and colleagues demonstrated a relationship between % ventricular pacing (VP) and subsequent hospitalization for heart failure and the occurrence of atrial fibrillation. DDDR mode, cumulative % VP and risk of first heart failure hospitalization (HFH). Risk of HFH increased between 0 and 40% with VP, but RR was level above 40% VP. Risk is reduced to about 2% if VP is minimized. (Reprinted with permission from Sweeney MO, Hellkamp AS, Ellenbogen KA et al.; MOde Selection Trial Investigators. Adverse effect of ventricular pacing on heart failure and atrial fibrillation among patients with normal baseline QRS duration in a clinical trial of pacemaker therapy for sinus node dysfunction. Circulation 2003; 107:2932–7, American Heart Association.)](image)
Optimal ventricular pacing sites

Given the potential adverse effects of RV apical pacing, significant attention has been given to other RV pacing sites that may avoid these adverse effects (Table 2.4). Although there is no definitive answer regarding optimal RV pacing site(s), there have been multiple studies assessing the efficacy and safety of different RV pacing sites. Table 2.4 presents a summary of these studies.

**Table 2.4** Clinical studies of the adverse effects of right ventricular (RV) apical pacing

<table>
<thead>
<tr>
<th>Trial</th>
<th>No. of patients</th>
<th>Mean age (years)</th>
<th>Mean FU (years)</th>
<th>LA diameter</th>
<th>LV function</th>
<th>CHF</th>
<th>AF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tantengco et al.</td>
<td>24</td>
<td>19.5</td>
<td>9.5</td>
<td>NA</td>
<td>↓</td>
<td>2 patients</td>
<td>NA</td>
</tr>
<tr>
<td>Karpawich et al.</td>
<td>14</td>
<td>15.5</td>
<td>5.5</td>
<td>NA</td>
<td>Altered histology</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Thambo et al.</td>
<td>23</td>
<td>24</td>
<td>10</td>
<td>NA</td>
<td>↓/DS</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Tse et al.</td>
<td>12</td>
<td>72</td>
<td>1.5</td>
<td>NA</td>
<td>↓/MPD</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Hamdan et al.</td>
<td>13</td>
<td>66</td>
<td>NA*</td>
<td>NA</td>
<td>↓/↑SNA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>DAVID</td>
<td>506</td>
<td>64</td>
<td>1</td>
<td>NA</td>
<td>↑</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>MADIT</td>
<td>567</td>
<td>64</td>
<td>1.7</td>
<td>NA</td>
<td>↑</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Substudy</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wonisch et al.</td>
<td>17</td>
<td>59</td>
<td>0.25</td>
<td>NA</td>
<td>NA</td>
<td>**</td>
<td>NA</td>
</tr>
<tr>
<td>Thackray et al.</td>
<td>307</td>
<td>72</td>
<td>5.2</td>
<td>NA</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>MOST</td>
<td>1,339</td>
<td>74</td>
<td>6</td>
<td>NA</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Nielsen et al.</td>
<td>177</td>
<td>74</td>
<td>2.9</td>
<td>↑</td>
<td>↓</td>
<td>NA</td>
<td>↑</td>
</tr>
<tr>
<td>O’Keefe et al.</td>
<td>59</td>
<td>69</td>
<td>1.5</td>
<td>NA</td>
<td>↓</td>
<td>NA</td>
<td>NA</td>
</tr>
</tbody>
</table>

AF, atrial fibrillation; CHF, congestive heart failure; DS, dyssynchrony; FU, follow-up; LA, left atrium; LBBB, left bundle branch block; LV, left ventricular; MPD, myocardial perfusion defects; NA, not available/not assessed; SNA, sympathetic nerve activity.

*Acute study.

**Permanent RV pacing significantly reduced exercise capacity and submaximal cardiorespiratory parameters.


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ies assessing different pacing configurations and their impact on hemodynamics. Tables 2.5 and 2.6 summarize some of the studies to date. Although a number of studies have now been published, many of the series are small, some involving long-term pacing and others short-term observation only, and overall there is no trend or concordance of the results.

One elegant study deserves more detail. In a study of 24 patients randomized to RV apical or RV outflow tract pacing, the mean QRS duration was significantly longer with apical pacing than during outflow tract pacing (151 ± 6 vs. 134 ± 4 ms, *P* = 0.03). At 18 months, the incidence of myocardial perfusion defects (83% vs. 33%) and regional wall motion abnormalities (75% vs. 33%) were higher and left ventricular ejection fraction (47 ± 3 vs. 56 ± 1%) was lower in those patients paced apically vs. those patients with outflow tract pacing (all *P* < 0.05). Investigators concluded that preserving synchronous ventricular activation with right ventricular outflow tract pacing prevented the long-term deleterious effects of RV apical pacing on LV function and perfusion.*" (Fig. 2.24).

In addition to investigations of alternate RV pacing sites, there have been multiple additional studies which have investigated the hemodynamics of LV pacing, multisite RV pacing and pacing the ventricles in three sites (Table 2.6). However, a definitive answer regarding the optimal ventricular pacing site(s) is still not available. Also, the literature is confusing because of the anatomical terms used to describe alternative RV pacing sites. The difficulty in comparing different ventricular pacing sites has led to proposed nomenclature to describe alternative RV pacing sites, i.e. other than the apical pacing site.*" The proposed sites are as follows (Fig. 2.25):

- **RV inlet septal pacing.** Pacing above, on or beneath the annulus of the septal-anterior tricuspid valve leaflets, yielding relatively normal QRS morphology and axis (A).
- **RV infundibular septal pacing.** Pacing proximal to the pulmonic valve distal to, or near, the crista supraventricularis, yielding left bundle branch block (LBBB) and a vertical axis (B).
- **RV outflow septal pacing.** Pacing most commonly referred to as RV outflow tract pacing near the septal-moderator band insertion at a midposition on the RV septum, yielding LBBB and a vertical axis (C).
- **RV apical septal pacing.** Pacing proximal to the septal-moderator band continuity that does not
typically produce a vertical QRS axis (D). The exact site of RV outflow tract pacing is critical to the pattern of LV activation. For example, cephalad to the crista supraventricularis on the free wall, the wavefront of activation will spread to the right ventricle significantly ahead of the intraventricular septum and left ventricle. On the other hand, a posterior pacing site above the crista supraventricularis may pre-excite the LV free wall because of the myocardial architecture and direction of spiral laminarization of the myocardium in this location. Further, the remnants of an extensive embryological infranodal Hisian conduction system (right superior fascicles) if present in a patient will result in more rapid and early conduction via the His-Purkinje system than in patients who do not have such remnants and thus rely entirely on intraventricular conduction for wave propagation.

Table 2.5 Studies comparing hemodynamic and/or clinical effects of right ventricular (RV) apical pacing and alternate site pacing in the RV outflow tract (RVOT) or RV septal (RVS) or left ventricular (LV) or biventricular (Biv) site

<table>
<thead>
<tr>
<th>Study</th>
<th>No. of patients</th>
<th>Mean age (years)</th>
<th>Hemodynamic/clinical variables</th>
<th>Improved results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cowell et al.</td>
<td>15/120</td>
<td>59</td>
<td>CO (Cath)</td>
<td>RVS, NA</td>
</tr>
<tr>
<td>Giudici et al.</td>
<td>89/121</td>
<td>68</td>
<td>CO (Echo)</td>
<td>RVOT, NA*</td>
</tr>
<tr>
<td>Buckingham et al.</td>
<td>11/12</td>
<td>48</td>
<td>CO (Echo)</td>
<td>RVOT, NA</td>
</tr>
<tr>
<td>Karpawich and Mital</td>
<td>22/122</td>
<td>10</td>
<td>LVEDP (Cath)</td>
<td>RVS, NA</td>
</tr>
<tr>
<td>Blanc et al.</td>
<td>23/123</td>
<td>66</td>
<td>PWP (Cath)</td>
<td>LV/BIV, NA</td>
</tr>
<tr>
<td>De Cock et al.</td>
<td>17/125</td>
<td>58</td>
<td>CO (Echo)</td>
<td>RVOT, NA</td>
</tr>
<tr>
<td>Mera et al.</td>
<td>12/124</td>
<td>68</td>
<td>FS/EF (Echo/RNV)</td>
<td>NA, RVS</td>
</tr>
<tr>
<td>Buckingham et al.</td>
<td>14/127</td>
<td>55</td>
<td>12 (Echo/Cath)</td>
<td>RVOT/BF, NA</td>
</tr>
<tr>
<td>Victor et al.</td>
<td>16/128</td>
<td>69</td>
<td>4 (Echo/RNV)</td>
<td>NA, None</td>
</tr>
<tr>
<td>Schwaab et al.</td>
<td>14/129</td>
<td>71</td>
<td>EF (RV)</td>
<td>RVS, NA</td>
</tr>
<tr>
<td>Kolettis et al.</td>
<td>20/130</td>
<td>62</td>
<td>CO (Echo)</td>
<td>RVOT, NA</td>
</tr>
<tr>
<td>Bourke et al.</td>
<td>20/131</td>
<td>64</td>
<td>8 (RNV)</td>
<td>NA, RVOT</td>
</tr>
<tr>
<td>Tse et al.</td>
<td>24/132</td>
<td>75</td>
<td>WMA/EF (RNS/RNV)</td>
<td>NA, RVOT</td>
</tr>
<tr>
<td>Hamdan et al.</td>
<td>13/133</td>
<td>66</td>
<td>BP/CVP/SNA</td>
<td>LV/Biv, NA</td>
</tr>
<tr>
<td>Kass et al.</td>
<td>18/134</td>
<td>66</td>
<td>10 (Cath)</td>
<td>LV/Biv, NA</td>
</tr>
<tr>
<td>Yu et al.</td>
<td>33/135</td>
<td>66</td>
<td>14 (Echo)</td>
<td>Biv, NA</td>
</tr>
<tr>
<td>Leclercq et al.</td>
<td>37/136</td>
<td>63</td>
<td>6 (Clinical)</td>
<td>NA, Biv</td>
</tr>
<tr>
<td>Leon et al.</td>
<td>20/137</td>
<td>70</td>
<td>6 (Echo/Clinical)</td>
<td>NA, Biv</td>
</tr>
<tr>
<td>Leclercq et al.</td>
<td>56/138</td>
<td>73</td>
<td>5 (Clinical)</td>
<td>NA, Biv</td>
</tr>
<tr>
<td>ROVA**</td>
<td>103/139</td>
<td>69.5</td>
<td>6 (Clinical/Echo)</td>
<td>ND, ND</td>
</tr>
<tr>
<td>OPSITE**</td>
<td>56/140</td>
<td>70</td>
<td>QOL/exercise capacity</td>
<td>Biv/LV, NA</td>
</tr>
<tr>
<td>PAVE**</td>
<td>252/141</td>
<td>NA</td>
<td>4 (Clinical)</td>
<td>NA, Biv</td>
</tr>
</tbody>
</table>

**BF, bifocal; BP, arterial blood pressure; Cath, cardiac catheterization; CO, cardiac output; CVP, central venous pressure; Echo, echocardiography; EF, (left ventricular) ejection fraction; FS, (left ventricular) fractional shortening; LVEDP, left ventricular end-diastolic pressure; NA, not available/not assessed; ND, no difference; PWP, pulmonary wedge pressure; RNS, radionuclide scintigraphy; RNV, radionuclide ventriculography; SNA, sympathetic neural activity (measured by microneurography); WMA, wall motion abnormalities.

*Only five patients were evaluated at 6 months and demonstrated a similar improvement in cardiac output with RV outflow tract pacing compared with RV apical pacing.

**Randomized studies; ROVA compared quality of life between RV apical and RV outflow tract pacing only after 3 months of pacing in patients with heart failure and chronic atrial fibrillation; OPSITE: randomized, single-blind, 3-month crossover comparison between RV and LV pacing and between RV and Biv pacing in patients with atrial fibrillation and heart failure undergoing AV node ablation; PAVE: randomized study evaluating Biv vs. RV pacing in atrial fibrillation patients receiving ablate and pace therapy.


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Pacing in congestive heart failure

CHF affects a large population of patients in the USA, approximately 5 million, and is the most common diagnosis at hospital dismissal. Although medical therapy has been shown to improve survival and functional status in these patients, many remain symptomatic despite maximally tolerated doses. Patients who remain symptomatic with medical therapy have an observed annual mortality of 12–40%. After initial investigations for standard dual-chamber pacing as a therapy for refractory heart failure had proved ineffective in most patients, biventricular pacing was attempted and has rapidly grown into the well-established discipline of cardiac resynchronization therapy.

Dual-chamber pacing

In the early 1990s, right-sided AV sequential (dual-chamber) pacing with short AV delays was proposed as empirical therapy to relieve CHF symptoms in patients with severe LV dysfunction. In 1990, Hochleitner et al. reported improvement in LV ejection fraction and functional class with pacing at a short AV delay in patients with dilated cardiomyopathy. The 16 patients studied were critically ill and refractory to maximal medical management. The baseline PR interval was 200 ms (range 140–320 ms); patients received empirical pacing at an AV delay of 100 ms.

In subsequent investigations, hemodynamic improvement by dual-chamber pacing was shown to be related to optimal synchronization of atrial and ven-
tricular contractions. However, only patients with CHF who have prolonged PR intervals, in which atrial contraction occurs so prematurely that the atrial “kick” to ventricular contraction is lost, appear to derive benefit from dual-chamber pacing. Conversely, in patients with normal or short AV conduction, the diastolic filling period does not change and cardiac output decreases by 23%, most likely because of the systolic and diastolic dyssynergy induced by RV pacing. Overall, the clinical trials demonstrated that dual-chamber pacing had limited long-term efficacy as an adjunct to medical therapy in relieving CHF symptoms.

Left ventricular and biventricular pacing
Progressive QRS widening develops during the course of disease in 68% of patients with cardiomyopathy and baseline intraventricular conduction abnormality. Complete LBBB is present in >80% of patients with end-stage cardiomyopathy within 6 weeks before death. Similar to the hemodynamic effects of isolated LBBB, LBBB in cardiomyopathy increases isovolumic contraction and relaxation times, thereby increasing the duration of mitral regurgitation and shortening LV filling time, with the net effect of decreasing preload. The duration of mitral regurgitation is more sensitive to heart rate in patients with LBBB than in those without. The magnitude of these effects is proportional to the QRS duration. Regionally diminished myocardial function or disturbed temporal sequence of contraction secondary to abnormal electrical activation disproportionately worsens systolic dysfunction in cardiomyopathy, since the remaining myocardium cannot provide the compensatory increase in fiber shortening necessary to maintain stroke volume. Therefore, LV pacing and si-
multaneous biventricular RV and LV (multisite) pacing have recently been used to alter the ventricular electrical and mechanical activation sequence in patients with severe, symptomatic LV dysfunction and intraventricular conduction delay or LBBB.\textsuperscript{56–64}

Influence of pacing site
The site of latest LV activation during RV apical pacing is the posterior or posteroinferior base.\textsuperscript{65} Since the electrical activation sequence in LBBB is very similar to that in RV pacing, LV pacing at the posterior or posteroinferior base should have a normalizing effect on ventricular activation. Consequently, monoventricular LV pacing in patients with severe LV dysfunction and LBBB yields a hemodynamic response similar to that of biventricular pacing and significantly higher than that of RV pacing.\textsuperscript{56,64} Moreover, pacing of the midlateral area or posterior area of the left ventricle in this situation leads to greater improvement in pulse pressure and $dP/dt$ than pacing of anterior or apical LV sites.\textsuperscript{66}

When comparing patterns of LV activation during intrinsic rhythm and RV pacing, two factors are important. First, the location of the RV pacing site relative to the normal exit of the right bundle branch. When pacing exactly at the exit site of the right bundle branch, one would expect that LV activation would be identical to that seen with LBBB. With varying the RV pacing site, however, intrinsic conduction with LBBB may have either less or more disorganized activation when compared with RV pacing. Second, when considering LV pacing simultaneous with the RV pacing site and comparing this with intrinsic conduction, one must keep in mind that the normal left bundle exit (if it was conducting normally) would be on the left side of the intraventricular septum and not on the free wall. This important difference is among the reasons why LV free wall pacing may be considered in the future for CRT even in the absence of LBBB.\textsuperscript{58} The reduced hemodynamic effects of simultaneous biventricular compared with monoventricular LV pacing\textsuperscript{58,60,61} suggest that some delay between RV and LV stimulation may be beneficial given the degree of LV hypertrophy frequently present in patients with cardiomyopathy. The functional status of the myocardium in the paced segment also affects the hemodynamic results: in patients with coronary artery disease, pacing of ischemic myocardium is not as effective as pacing of myocardium with normal regional function\textsuperscript{67} and adversely affects the relationship of LV $dP/dt$ to end-diastolic volume.

Mechanisms underlying the benefits of left ventricular and biventricular pacing
The mechanisms by which LV and biventricular pacing improve mechanical LV function in patients with CHF and LBBB are not entirely understood. Up to four levels of dysynchrony have been described (Fig. 2.27). These include atrioventricular dysynchrony (discussed above), interventricular dysynchrony (between the right and left ventricles), intraventricular dysynchrony (within segment of the left ventricle), and intramural dysynchrony within the walls of the left ventricle (least well established).\textsuperscript{68} Intraventricular dysynchrony is probably the most significant clinically. Electrical resynchronization between the right ventricle and left ventricle should eliminate the adverse effects of LBBB-induced mechanical ventricular dysynchrony on regional LV systolic function. However, biventricular pacing in the absence of conduction system disease is hemodynamically superior to RV pacing, despite similarly paced QRS
duration. Also, hemodynamic improvement with LV pacing in LBBB is equivalent to, if not better than, that with biventricular pacing even though it does not shorten the QRS complex. Preliminary studies have shown that the improvement in systolic dP/dt by LV pacing in LBBB is proportional to the reduction of the electromechanical delay within the left ventricle. This finding implies that synchronization of LV wall motion is important in the improvement of systolic function. Therefore, for achieving benefit from pacing therapy,
electrical resynchronization evidenced by QRS narrowing may be less important than the LV pacing site and the associated change in LV contraction efficiency.66-68,69 The hemodynamic improvements in systolic LV function with pacing may not be due to mechanical factors alone. Systemic or intramyocardial release of catecholamines, reflex-mediated baroreceptor and autonomic nervous system activation, and release of vasodilatory substances, such as atrial natriuretic peptides, have all been demonstrated for dual-chamber pacing and may well have a role in biventricular and LV pacing.

**Ventricular timing optimization (V-V optimization)**

Once it was recognized that not all LV pacing sites were equivalent in a given patient in terms of providing symptomatic benefits, methods to optimize biventricular pacing began to be developed. Optimization may take the form of attempting various pacing sites in the coronary venous system at implant, placing the RV pacing lead at varying sites, varying the vector of pacing (bipolar vs. LV tip to RV ring, etc.), varying the timing of LV and RV pacing, and utilizing or avoiding anodal stimulation.

**Optimizing site of pacing (LV and/or RV)**

It is generally recognized that the ideal LV pacing site is on the midportion of the free wall of the left ventricle placed as far away as possible from the RV pacing lead when observed in the left anterior oblique (or lateral X-ray) image. There are obvious difficulties with this simplification, since some patients will have a lateral wall infarction or have an inordinate amount of exit delay related to the lateral pacing site, and the least dysynchrony-producing LV site may depend on the location of the RV pacing lead, etc.

**Varying the pacing vector**

With bipolar stimulation using closely spaced electrodes, the pacing site coincides with the location of the electrodes. The situation is more complex when pacing occurs between widely spaced electrodes on the LV pacing lead or between a LV electrode and a RV ring or coil electrode. The QRS morphology, activation pattern, and actual site of stimulation (cathode vs. anode) can vary quite markedly. There are no clear data in the literature in terms of impact on symptom improvement and long-term outcomes when the pacing vectors are changed.

**Impact of diseased myocardium proximate to the pacing electrodes**

At times, the LV pacing lead may be optimally located; however, because of prominent exit delay, only a small region of left ventricle is captured by the LV electrode, so that effectively, the left ventricle is predominantly activated via the RV pacing lead. During threshold testing at high outputs, because of recruitment of larger areas of myocardium, there is less capture latency and more rapid intraventricular conduction, with true LV stimulation during biventricular pacing. On the other hand, at lower output, despite LV capture, capture latency is prolonged and conduction slowed with effective loss of LV lead contribution to LV depolarization during simultaneous biventricular stimulation. In this situation, providing an offset (pacing the left ventricle earlier than the right ventricle) can remedy the situation by allow greater time for LV depolarization via the LV lead-initiated wavefront, prior to RV stimulation. Although increasing the LV pacing output is an alternative solution, the obvious limitation is shortened battery longevity.

**Electrical parameters for V-V optimization**

When optimizing the pacing site at implantation, the parameter used to identify the optimal site or pacing configuration must be determined. The QRS duration is the most easily obtained electrical parameter to use during implant procedures, as well as in follow-up to optimize ventricle to ventricle timing. Lecoq et al.69 studied whether electrocardiographic parameters can predict response from CRT. They found that of all the variables studied in predicting response to CRT, the amount of QRS shortening associated with biventricular stimulation was the only independent predictor. Notably, in this study, the RV implantation site was specifically manipulated to obtain the shortest possible QRS duration. However, contrary conclusions were drawn in other studies.70 There are clear limitations to the use of the QRS duration alone for optimization. First, isoelectric periods during QRS inscription may occur when the activation wavefront proceeds through areas of extremely slow conduction involving diseased myocardium. The location of the slow zones/abnormal tissue will determine whether the QRS appears wide or not. If a slow zone of myocardial conduction is the first or last site to be activated following stimulation, the QRS may appear normal (if normal portions not seen
in the routine 12-lead electrocardiogram). On the other hand, if the slow zone is mid-left ventricular and the pacing site is right ventricular or intrinsic rhythm with LBBB is present, a wide QRS results. Similarly, when optimizing the V-V timing or LV lead pacing site, a narrow QRS may result from true synchronous electrical activation of the ventricles or from activation of a slow zone at the beginning or end of the QRS inscription.

**QRS vector fusion**

The premise for this method to optimize biventricular devices is that the QRS morphology (vector) is specific for a given pacing site (Fig. 2.28). Thus, LV pacing from the lateral wall typically results in a QS complex in lead I, right bundle branch block patterns, and negative or isoelectric complexes in leads II, III and aVF; and RV apical pacing results in a tall R wave in lead I and a LBBB pattern. With biventricular stimulation, a fused vector, in which the QRS morphology is a hybrid between lone-RV and lone-LV pacing, is expected (Fig. 2.29) If, the biventricular paced QRS morphology matches the lone-RV pacing morphology despite a good LV threshold, then either prolonged capture latency or significant conduction delay from the LV pacing site is present. By progressively pacing the LV lead earlier than the RV lead while monitoring the QRS for fusion (hybrid QRS complexes) during biventricular stimulation, the V-V can be optimized (Fig. 2.30). In certain situations (septal infarcts) an offset may need to be programmed whereby the RV lead is paced earlier than the LV lead. Some studies have indicated that electromechanical delay as well as possible tethering of the infarct region to nearby myocardium may be more prominent in ischemic cardiomyopathy than in idiopathic dilated cardiomyopathy.

Therefore, when using the ECG to optimize V-V timing, the QRS duration and QRS vector fusion are analyzed, which may compensate for some the vagaries of capture latency and intraventricular conduction heterogeneity. Attention should also be paid to abrupt changes in QRS morphology which result from anodal stimulation or change in the pacing vector configuration. Anodal stimulation is discussed in detail in Chapter 10, Troubleshooting. Although QRS-based optimization is straightforward for use during implant, by definition it optimizes the surface

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**Fig. 2.28** Twelve-lead electrocardiogram obtained from a patient with a biventricular pacing system. The QRS demonstrates a right bundle branch block morphology suggesting left ventricular stimulation. To further localize the site of the left ventricular pacing lead we note that leads II, III and aVF are all negative, suggesting a posterior/inferiorly located pacing lead. However, we note further that lead I is negative, suggesting a lateral left ventricular location for the left ventricular pacing lead. An additional finding is that the degree of negativity (depth of QF complex) is deeper in lead II (left-sided lead) than in lead III (a right-sided lead). Each left ventricular lead pacing site has a specific signature electrocardiogram and this example is consistent with a left ventricular lead located in a posterolateral site.
As noted above, important electromechanical delay may be present despite optimization of the QRS. Thus, electrical synchronization, although feasible and usually quite straightforward, lacks the appeal of mechanical optimization of ventricular lead placement.

**Echocardiography for ventricular timing optimization**

Echocardiographic approaches used to identify the best LV lead placement site have included two-dimensional and M-mode analysis to find the sites of ventricular contraction that are delayed and occurring after aortic ejection is underway. This typically involves M-mode measurement through a short axis projection at the papillary muscle level comparing the septal and free-wall contractility. The imaging plane is scanned from apex to base and the site of maximal delay/dyssynchrony is targeted for lead placement. A further refinement over simple M-mode detection of delayed activation when compared with the onset of the QRS is the use of tissue Doppler velocity–derived variables. Others have used tissue Doppler velocity imaging to measure the interval from atrial filling to the actual onset of contraction of the left ventricle. This technique, described below, may take away the need to use surrogates for LV contraction.

One construct for predicting the ideal location to place the LV pacing lead is to aim for early stimulation at a location where, without pacing, the latest activation occurs. Using the QRS duration or morphology to identify this location is difficult. For example, if the latest sites of activation are within a diseased peri-infarct ventricular region, the fragmented local electrograms that result from the wavefront propagating to this region may not contribute (and hence be invisible in) the surface QRS. On the other hand, if the abnormally slow area of conduction is just proximal to the last site to be activated, a discernible deflection at the terminal portion of the QRS (as the wavefront exits the diseased region), possibly separated from the initial QRS by an isoelectric period (reflecting conduction through the diseased tissue), may result. Despite these ECG differences, the optimal pacing location, assuming it is the last site activated, would be very similar. Auricchio

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Fig. 2.29 EKG vector fusion. By permission of Mayo Foundation.
et al.\textsuperscript{73} found marked differences in endocardial activation despite a near identical LBBB morphology on the QRS complex. Endocardial activation depended on the site of intraventricular conduction delay as identified with electroanatomical and non-contact mapping. Because of these issues, mechanical parameters have been more frequently used to find the last site of ventricular activation. The important problem that remains even when an echocardiographic parameter (tissue velocity/M-mode contractility latest from the onset of the QRS, etc.) is used is that LV pacing at that location may not necessarily produce the maximal benefit. This is because one may simply change the pattern of dysynchrony with another location now being the latest to activate. Which pattern of dysynchrony is \textit{in toto} least conducive to normal hemodynamic is difficult, if not impossible, to predict. Algorithms that either simulate or predict resultant total LV dysynchrony following LV stimulation prior to placing the lead are being investigated.

![Fig. 2.30 The top panel shows the characteristic 12-lead electrocardiogram when pacing from a right ventricular apex. Note, in lead V1, the predominant morphology is that of left bundle branch block, but as is commonly observed in patients with enlarged left ventricles, a small R wave is noted in lead V1. The bottom panel shows the 12-lead electrocardiogram when pacing from a left free wall anterolateral location. Note, the deep negative QS complex in lead I, right bundle branch block configuration and the positive QRS complexes in leads II, III and aVF. The middle panel shows biventricular pacing with simultaneous left and right ventricular stimulation. Most of the leads depicted are more akin to LV pacing (bottom panel). This suggests that the patient may have a septal infarction such that the right ventricular pacing wavefront does not effectively depolarize a significant portion of the myocardium. An improvement in ventricular synchrony may be obtained by programming an offset for right ventricular stimulation to occur prior to left ventricular stimulation. This situation is exceptional, with most cases requiring an offset with left ventricular pre-excitation.\textsuperscript{81}]

RV pacing

BiV pacing

LV pacing
Clinical approaches to V-V optimization

With the option to program an offset between RV and LV stimulation, methods have been sought to optimize the V-V stimulus interval. Thus, QRS duration and QRS vector summation decreasing the site of maximal mechanical delay, delayed tissue Doppler-derived contraction sites and global indices improvement of tissue contraction have been studied with varying the V-V interval. Tissue Doppler acceleration is derived from the tissue Doppler velocity and represents the rate at which the tissue velocity changes. This parameter is less dependent on passive ventricular movement, and its timing relative to QRS onset and to tissue Doppler acceleration at other sites in the ventricle can be used in a similar manner. The frame rate or firing rate in case of M-mode imaging is of paramount importance when dealing with electrical and electromechanical phenomena. The frame rate has to be sufficiently high to track electrical conduction. Electrical conduction velocities, even in diseased myocardium, are sufficiently rapid that ideal frame rates are required in the range of 700–1200 frames/s. With transthoracic imaging, one is lucky to achieve frame rates between 100 and 150 frames/s. Thus, what is actually perceived as dyssynchrony between two segments is quite different when imaged with frame rates of 10/s or 1000/s. With the lower frame rates, two sites may be thought to activate simultaneously when in fact this is an artifact of the slow change in frame rate.

Sogaard published several of the early investigations to demonstrate the hemodynamic advantage of optimizing the V-V interval in patients with severe heart failure and LBBB. In one study, 3-D echocardiography and Doppler tissue imaging were performed before and after implant and re-examination at 3 months. Although simultaneous cardiac resynchronization did reduce the extent of myocardium displaying delayed longitudinal contraction (DLC, a Doppler measure of local mechanical activation) and improved the LV ejection frac-

Fig. 2.31 M mode echocardiogram obtained through a short axis view visualizing the septum and free wall of the left ventricle. In the upper panel, peak systolic contraction occurs on the free wall late after the QRS and is significantly preceded by peak contraction on the septum. In the lower panel, simultaneous peak contraction of both the septum and free wall occur soon after the QRS complex following biventricular pacing stimulation and signifies likely synchronous left ventricular contraction.
tion, there was an incremental benefit with sequential cardiac resynchronization. In this study, patients with idiopathic dilated cardiomyopathy had delayed longitudinal contraction in the lateral and posterior walls of the left ventricle. In contrast, DLC was more frequent in the septum and inferior walls in patients with ischemic cardiomyopathy. Preactivation of the LV lead was helpful in nine patients, whereas preactivation of the RV lead was superior in the remaining 11 patients. LV preactivation tended to be more beneficial in patients with delayed lateral wall contraction, i.e., patients with dilated cardiomyopathy. The degree of preactivation was surprisingly modest at about 20 ms. It is important to note that the actual area of mechanical asynchrony is not reflected by the LBBB pattern alone, but is specific based on the nature of the cardiomyopathy and location of ischemia and infarction. Also noted in this study was significant improvement in the 6-min hall walk test, where the walking distance doubled.

Pitzalis et al. assessed the role of septal-to-posterior wall motion delay (SPWMD) on the echocardiogram as a predictor of CRT response (Fig. 2.31). In a study of 60 patients followed over a 14-month period, multivariate analysis determined that a long SPWMD was significantly associated with a decreased risk of heart failure progression (hazard ratio 0.91; 95% confidence interval 0.83, 0.99; P < 0.05). In 79% of patients with a baseline SPWMD of ≥130 ms there was improvement in the LV ejection fraction, but in only 9% of those with an SPWMD of <130 ms (P < 0.0001). They concluded that the baseline SPWMD is a strong predictor of CRT response in patients with LBBB and severe heart failure. However, when data from patients from the CONTAK-CD trial were analyzed, the SPWMD did not correlate with clinical parameters of improvement. In summary, numerous studies have demonstrated improvement in markers such as ejection fraction when echocardiographically optimizing CRT. However, the prospective randomized clinical trials that provide the evidence base for CRT nearly universally used surface ECG criteria. At present, echocardiographic optimization is used to address the issue of non-responders; its role in patient selection and lead placement at implant is promising and the literature is rapidly expanding, but it has not yet been included as part of the accepted CRT selection criteria.

Other end-points for optimization

Although both atrioventricular and ventricle-to-ventricle optimization has primarily targeted improvements in mechanical function and, in some instances, its translation to effort tolerance, other end-points need to be considered also.

Mitrail regurgitation

The extent of mitral regurgitation has been shown to decrease with LV stimulation. It does not follow, however, that the ideal lead location, AV interval and VV interval to improve cardiac output or ventricular contractility will be the same as those that minimize mitral regurgitation the most.

Arrhythmogenesis

Certain pacing sites may be associated with an increased propensity for re-entrant ventricular arrhythmia, whereas other sites may actually serve to decrease the likelihood of a re-entrant circuit developing. Currently, it is not possible to predict which pacing site will be beneficial and which will be arrhythmogenic, as methods to reliably locate the arrhythmogenic slow zones necessary for re-entry are lacking. Coronary sinus lead placement pre-excites the epicardium relative to the endocardium, whereas the RV lead pre-excites the endocardium. Wedge preparations suggest epicardial pacing may be arrhythmogenic relative to endocardial pacing. Finally, the actual location of the RV pacing leads, even if non-arhythmogenic, would need to be located at a site likely to terminate ventricular tachycardia should it occur as part of defibrillator function (antitachycardia pacing).

Left atrial pacing

At times, it is impossible to provide optimal LA-LV synchrony while maintaining RA-RV synchrony. This is because a single atrial site with variable intra-atrial conduction is present, yet with two different intraventricular sites. Additionally, when the V-timing is varied, re-optimization of AV timing for both sides of the circulation may be required. In such cases, it becomes necessary to have the ability to pre-excite the left atrium if needed. Although this may be partially accomplished with Bachmann's bundle pacing or coronary sinus pacing, ideally, a separate left atrial pacing lead should be placed. This is typically done via the coronary sinus and utilizing a patent vein of Marshall (oblique vein of the left atrium). Although theoretically attractive, the clinical benefit of using a fourth (dedicated left atrial) pacing lead has not been established, and it is not routinely performed in practice.
The QT interval can also be used as a marker for ideal ventricular output. Ishikawa has shown a correlation between the QT interval and the cardiac output and the effect of these measures by varying PAV intervals. At optimal AV intervals, an increase in both the cardiac output and the QT interval is noted.

**Ventricular rate regulation**

Ventricular rate regulation (VRR) or ventricular rate stabilization algorithms are intended to minimize ventricular cycle length variation in patients with atrial fibrillation. In some patients, the use of a VRR algorithm will result in better tolerance of atrial fibrillation. It has been shown to stabilize effectively the ventricular rate without significantly increasing the pacing rate and may result in a more favorable autonomic balance and lead to improved rate recovery after exercise (Fig. 2.24).

In a multicenter trial, assessing the potential benefits of a ventricular pacing response algorithm did decrease the severity of atrial fibrillation-related symptoms. However, it did not improve the general quality of life assessed by the SF-36, functional capacity by 6-min hall walk, or performance of routine activities as assessed by the Duke Activity Status Index.

Although the overall impact of VRR on hemodynamic status will vary from patient to patient, it appears that this type of algorithm has potential hemodynamic advantage in patients with atrial fibrillation requiring permanent pacing.

Algorithms are also available to limit irregularities due to pauses after premature ventricular contractions. It had been hoped that these might prevent arrhythmias by mitigating arrhythmogenic short-long-short intervals that introduce variable repolarization and arrhythmogenesis. Although these might be attractive in subsets of patients (e.g., long QT syndrome), in a heterogeneous representative population of ICD recipients, these algorithms did not appear to prevent arrhythmias. Notably, the devices tested were standard ICDs, capable of pacing the ventricles only from the right ventricle.

Less common indications for pacing for hemodynamic improvement

**Pacing in hypertrophic obstructive cardiomyopathy**

The many subtypes and multiple clinical presentations of hypertrophic obstructive cardiomyopathy (HCM) have made it difficult to compare and identify optimal diagnostic and treatment modalities. Patients with HCM have various degrees of septal hypertrophy and outflow tract obstruction, and they may experience exertional dyspnea, angina, syncope, or sudden cardiac death. Symptoms may be due to massive hypertrophy and myocardial microischemia, mitral regurgitation from displacement of the mitral valve apparatus, outflow tract obstruction severe enough to cause hemodynamic embarrassment, or impaired diastolic function.

Treatment has traditionally been with β-adrenergic and calcium blocking agents and, in medically refractory cases, surgical septal myectomy (sometimes with concurrent mitral valve repair or replacement). Some patients have required postoperative pacing because of damage to the conduction system at the time of myectomy or pacing therapy for the severe bradycardia produced in some by medical therapy.

McDonald et al. described the use of dual-chamber pacing as a primary treatment for outflow tract obstruction in 1988. Subsequent investigators have evaluated both invasive and non-invasive hemodynamic values in both short-term and long-term pacing therapy.

Fananapazir et al. reported on 84 patients with HCM and drug-resistant symptoms who were treated by dual-chamber pacemakers programmed to the DDD mode with AV intervals short enough to fully activate the ventricle from the pacing site at the RV apex (according to electrocardiographic criteria). After a mean of 2.3 years, symptoms resolved (28 patients) or decreased (47 patients) in 89% of the patients. This outcome was associated with a significant improvement in mean New York Heart Association (NYHA) functional class, from 3.2 to 1.6, and a reduction in the LV outflow tract gradient from 96 to 27 mmHg in patients with significant outflow obstruction. These benefits persisted after cessation of pacing during normal sinus rhythm, as did some changes on the surface electrocardiogram (T-wave morphology) and the signal-averaged electrocardiogram. This study has led to hypotheses regarding improvement in hemodynamics post pacing and to multiple randomized clinical trials. The most widely accepted hypothesis to explain the improvement in hemodynamics that may occur during pacing in patients with HCM is that the altered septal activation caused by RV apical pacing may result in less narrowing of the LV outflow tract and a subsequent decrease in the Venturi effect, responsible for systolic anterior motion of the mitral valve (Fig. 2.32). However, the
persistence of improvement after cessation of pacing in some series and the observation that subjective and objective improvement may also be seen in some patients with LBBB suggest that the effect of long-term pacing cannot be attributed solely to alteration of the septal activation sequence by ventricular pacing. There are hypotheses that permanent pacing in patients with HCM may result in long-term remodeling of the left ventricle, but this has never been well established.

Pacing in HCM has been the subject of several randomized single-center and multicenter trials. A single-center, randomized, crossover trial demonstrated symptomatic improvement in 63% of patients with pacing in the DDD mode. However, 42% of patients had improvement with programming to a low pacing rate in the AAI mode, i.e., effectively no pacing, suggesting a significant placebo effect.

In the PIC (Pacing in Cardiomyopathy) study, a multicenter, randomized, crossover study, dual-chamber pacing resulted in a 50% reduction of the LV outflow tract gradient, a 21% increase in exercise duration, and improvement in NYHA functional class compared with baseline status. When clinical features, including chest pain, dyspnea, and subjective health status, were compared between DDD and back-up AAI pacing, there was no significant difference, again suggesting a significant placebo effect.

In a double-blind, crossover study, the M-PATHY (Multicenter Study of Pacing Therapy for Hypertrophic Cardiomyopathy) trial, patients were randomized to 3 months each of DDD or AAI pacing (rate, 30) in a crossover design. No significant differences were evident between pacing and no pacing, either subjectively or objectively, when exercise capacity, quality-of-life score, treadmill exercise time, and peak oxygen consumption were compared. Patients reported symptomatic improvement with pacing, a result suggesting a substantial placebo effect, and a small subset of patients > 65 years old had significant objective improvement, a suggestion that DDD pacing might be a viable option in these patients. The investigators concluded that pacing should not be considered a primary treatment for HCM and that subjective benefit without objective evidence of improvement should be interpreted cautiously.

As a result of these trials, pacing is now rarely used for the HCM patient. Device therapy in HCM patients is primarily ICD implantation for those HCM patients felt to be at significant risk for sudden cardiac death. Septal myotomy-myectomy remains the definitive therapy for outflow tract obstruction in HCM.

**Hemodynamic benefits of pacing in neurocardiogenic syndromes**

Hemodynamic considerations are important during pacing for neurocardiogenic syncope. Understanding the physiology involved is crucial to understanding the hemodynamics. The carotid sinus reflex is the physiological response to pressure exerted on the carotid sinus. Stimulation results in activation of baroreceptors within the wall of the carotid sinus, and they initiate an afferent response. Discharge from vagal efferents then results in cardiac slowing. Although this reflex is physiological, some persons have an exaggerated or even pathological response. This reflex has two components,
cardioinhibitory and vasodepressor. A cardioinhibitory response results from increased parasympathetic tone and may be manifested by sinus arrest, sinus bradycardia, PR prolongation, or advanced AV block. The vasodepressor response is due to sympathetic withdrawal and secondary hypotension. Although a pure cardioinhibitory or pure vasodepressor response can occur, a mixed response is most common.

Tilt-table testing can provide the physiological environment to reproduce vasovagal syncope (Fig. 2.33). With head-up tilt, susceptible patients have decreased venous return and subsequent decrease in LV filling. This response triggers stimulation of baroreceptors and adrenergic discharge, which can result in efferent vagal discharge and sympathetic withdrawal. Vasodilation and hypotension as well as cardiac slowing may result. It is important to document whether the predominant cause of symptoms is cardioinhibitory or vasodepressor, because therapy differs. Tilt-table testing is often helpful in determining the predominant cause.

A number of mechanical interventions, such as orthostatic training and support stockings, and medications including β-adrenergic blockers, serotonin uptake inhibitors, and α-receptor antagonists, are used in treatment. For refractory cases, although significant controversy persists, vasovagal syncope can be aborted or blunted by dual-chamber pacing, and even if syncope does occur, pacing can prolong consciousness to avoid injury.91

VVI pacing usually fails to ameliorate symptoms even if a bradycardic response prevails,96 because the absence of AV synchrony aggravates the peripheral vasodilation that generally accompanies this condition. However, existing data suggest that dual-chamber pacing may provide a beneficial effect. The results of randomized clinical trials have been inconsistent. In two trials of highly symptomatic patients with bradycardia, permanent pacing increased the time to first syncopal event. In one study the pacemaker was programmed to DDI at 80 bpm with hysteresis at 45 bpm vs. no pacing.97 In the other trial the pacemaker was programmed to β-blocker therapy or dual-chamber pacemaker with a sudden rate drop algorithm.98 In this study, pacemakers were more effective than β-blocker therapy in the prevention of recurrent syncope.

In the initial Vasovagal Pacemaker Study (VPS-1), the rate of recurrent syncope was 18.5% for pacemaker patients and 59.7% for control patients at 1 year.99 The same group of investigators subsequently performed VPS-2.100 In this double-blind randomized

Fig. 2.33 Electrocardiographic and arterial blood pressure recordings obtained during tilt testing. (A) Tilt with the patient asymptomatic demonstrates normal sinus rhythm at a heart rate (HR) of 115 bpm and baseline blood pressure (BP) of 136/67. (B) Tilt during syncope; the heart rate is 39 bpm and the blood pressure 54/30. (C) Subsequent tilt with atrioventricular (A-V) sequential pacing at a cycle length (PCL) of 700 ms (86 bpm). Significant vasodepression remains, but with the heart rate maintained, the patient experienced only presyncope. (Courtesy of Dr. W-K. Shen, Mayo Clinic.)
trial, in which all patients received a pacemaker and were randomized to pacing vs. no pacing, pacing therapy did not reduce the risk of recurrent syncopal events. This contrasts with VPS-1, in which patients were randomized to pacemaker implant vs. no pacemaker.

Pacing therapy is not considered first-line therapy for many patients with neurocardiogenic syncope. However, permanent pacing does have a role for some patients. In those patients who have little or no warning prior to their syncopal event, those with profound bradycardia or asystole during a documented event and those in whom other therapies have failed, a permanent pacemaker should be considered and may be effective in reducing symptoms if a significant cardioinhibitory component is felt to be contributing to the cause of the patient’s symptoms.

**Hemodynamic benefits of pacing in first-degree atrioventricular block**

Hemodynamic compromise due to marked first-degree AV block is well documented. It is unfortunate that the symptoms have been described as those of pacemaker syndrome. The hemodynamic compromise and symptoms in these patients are due to loss of optimal AV relationships (Fig. 2.27). Although loss of AV synchrony is a factor in pacemaker syndrome, as previously discussed, other adverse hemodynamic conditions, such as atrial stretch, contribute as well. Pacing therapy should not be limited to first-degree AV block. Patients with type I second-degree AV block, traditionally not an indication for pacing, who have hemodynamic compromise due to AV dysynchrony and not necessarily bradycardia, should also probably be considered for permanent pacing.

**Conclusions**

The goal of physiological pacing should be to restore normal physiology to the greatest extent possible. This includes restoration of rate responsiveness in all patients and restoration of AV synchrony in all patients with the exception of those with chronic atrial fibrillation. In CRT it also includes restoration of inter- and intraventricular synchrony.

The hemodynamic importance of optimizing the AV interval is well established, and differential AV intervals and rate-adaptive AV intervals are important considerations. Data from DAVID and MOST have also established the importance of avoiding ventricular pacing whenever possible.

Hemodynamic superiority of different pacing sites or multiple pacing sites has yet to be definitively proven.

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CHAPTER 3
Indications for Pacemakers, ICDs and CRT

Apoor S. Gami, David L. Hayes, Paul A. Friedman

Guidelines for the use of cardiac pacemakers and the internal cardioverter-defibrillator (ICD) were first established in 1984 by a task force formed jointly by the American College of Cardiology (ACC) and the American Heart Association (AHA). These were most recently updated in 2008 in conjunction with the North American Society of Pacing and Electrophysiology, now named the Heart Rhythm Society (HRS). In 2006, the ACC, AHA and European Society of Cardiology (ESC) published guidelines for prevention of sudden cardiac death, which included updated guidelines for ICD implantation. Based on the strength of available data and expert opinion, the indications have been divided into the following three classes:

Class I—There is evidence and/or general agreement that device implantation is beneficial, useful and effective.

Class IIa—There is conflicting evidence and/or a divergence of opinion, and the weight of the evidence or opinion is in favor of the usefulness or efficacy of device implantation.

Class IIb—There is conflicting evidence and/or a divergence of opinion, and the usefulness or efficacy of device implantation is less established.

Class III—There is evidence and/or general agreement that device implantation is not useful or effective and in some cases may be harmful. Device implantation is contraindicated.

Indications for permanent pacing

As the guidelines classification implies, some conduction disturbances are accepted as definite indications for permanent pacing, and for others there is general agreement that permanent pacing is not required. However, the precise criteria for the implantation of a permanent pacemaker vary by institution, and in a number of conduction disturbances the need for permanent pacing depends on the unique circumstances of the patient. Because of changes in diagnosis and therapy, the absolute indications for permanent pacing are constantly evolving.

Before concluding that permanent pacing is indicated, the physician must carefully assess whether it is in the best interest of the patient. This assessment should include the specifics of the cardiac rhythm disturbance, the patient’s general medical status, and the patient’s concerns and preferences.

Generally, indications for permanent pacing are categorized by the underlying conduction system disorder or disease process, including:

- Atrioventricular (AV) block
- Acute myocardial infarction
- Chronic bifascicular and trifascicular block
- Sinus node dysfunction
- Neuromediated syncope
- Tachyarrhythmias
- Hypertrophic cardiomyopathy
- Congestive heart failure
- Cardiac transplantation

The ACC/AHA/HRS guidelines also include a section on pacing indications for pediatric patients. In this chapter, specific pediatric considerations are included.
within the broader categories; for example, congenital AV block is included in the section on AV block.

**Atrioventricular block**

Atrioventricular block is the impairment of conduction of a cardiac impulse from the atrium to the ventricles. It can occur at different levels: proximal to the AV node, in the AV node, or in the His-Purkinje system. Indications for permanent pacing in patients with AV block are summarized in Table 3.1.

**Table 3.1 Indications for pacing in atrioventricular (AV) block**

<table>
<thead>
<tr>
<th>Class I</th>
</tr>
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<tbody>
<tr>
<td>1. Third-degree or advanced second-degree AV block at any anatomic level associated with any one of the following conditions:</td>
</tr>
<tr>
<td>a. Symptoms (including heart failure) attributable to AV block</td>
</tr>
<tr>
<td>b. Arrhythmias and other medical conditions that require drugs that result in symptomatic bradycardia</td>
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<tr>
<td>c. Documented periods of asystole &gt; 3.0 s, any escape rate ≤ 40 bpm, or any escape rhythm below the AV junction (e.g., a wide QRS morphology) in awake, asymptomatic patients in sinus rhythm</td>
</tr>
<tr>
<td>d. A documented period of asystole &gt; 5 sec in awake, asymptomatic patients in atrial fibrillation</td>
</tr>
<tr>
<td>e. After catheter ablation of the AV junction</td>
</tr>
<tr>
<td>f. Postoperative AV block that is not expected to resolve after cardiac surgery</td>
</tr>
<tr>
<td>g. Neuromuscular diseases, such as myotonic muscular dystrophy, Kearns-Sayre syndrome, Erb’s (limb-girdle) dystrophy, and peroneal muscular atrophy, with or without symptoms of bradycardia</td>
</tr>
<tr>
<td>2. Asymptomatic third-degree AV block at any anatomical site with an average awake ventricular rate &gt; 40 bpm in patients with cardiomegaly or left ventricular dysfunction</td>
</tr>
<tr>
<td>3. Second-degree or third-degree AV block during exercise in the absence of myocardial ischemia</td>
</tr>
<tr>
<td>4. Symptomatic second-degree AV block regardless of type or site of block</td>
</tr>
<tr>
<td>5. Congenital third-degree AV block with a wide QRS escape rhythm, complex ventricular ectopy, or ventricular dysfunction</td>
</tr>
<tr>
<td>6. Congenital third-degree AV block in an infant with a ventricular rate &lt; 50–55 bpm or with congenital heart disease and a ventricular rate &lt; 70 bpm.</td>
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</tbody>
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<table>
<thead>
<tr>
<th>Class IIa</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Advanced second-degree or third-degree AV block at any anatomical site with an average ventricular rate &gt; 40 bpm. in the absence of cardiomegaly or hypertrophy</td>
</tr>
<tr>
<td>2. Asymptomatic type I second-degree AV block at intra- or infra-His levels found at electrophysiological study</td>
</tr>
<tr>
<td>3. First-degree or second-degree AV block with symptoms similar to those of pacemaker syndrome</td>
</tr>
<tr>
<td>4. Congenital third-degree AV block after the first year of life with an average ventricular rate &lt; 50 bpm or abrupt pauses in ventricular rate that are two to three times the basic cycle length</td>
</tr>
<tr>
<td>5. Long QT syndrome with third-degree or advanced second-degree AV block</td>
</tr>
<tr>
<td>6. Congenital heart disease and loss of AV synchrony with impaired hemodynamics.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Class IIb</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Severe first-degree AV block (&gt; 0.30 s) in patients with ventricular dysfunction and symptoms of heart failure in whom a shorter AV interval results in hemodynamic improvement</td>
</tr>
<tr>
<td>2. AV block due to drug use or toxicity when the block is expected to recur even after withdrawal of the drug</td>
</tr>
<tr>
<td>3. Neuromuscular diseases, such as myotonic muscular dystrophy, Kearns-Sayre syndrome, Erb’s (limb-girdle) dystrophy, and peroneal muscular atrophy with any degree of AV block (including first-degree AV block), with or without symptoms of bradycardia</td>
</tr>
<tr>
<td>4. Pediatric patient with transient postoperative third-degree AV block that reverts to sinus rhythm with residual bifascicular block</td>
</tr>
<tr>
<td>5. Infant, child, adolescent, or young adult with asymptomatic congenital third-degree AV block, an acceptable rate, narrow QRS complex, and normal ventricular function.</td>
</tr>
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<thead>
<tr>
<th>Class III</th>
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</thead>
<tbody>
<tr>
<td>1. Asymptomatic first-degree AV block</td>
</tr>
<tr>
<td>2. Asymptomatic type I second-degree AV block at a site above the His (i.e., the AV node) or not known to be intra- or infra-Hisian by electrophysiology study</td>
</tr>
<tr>
<td>3. AV block expected to resolve and unlikely to recur (e.g., drug toxicity, Lyme disease, nocturnally in sleep apnea, early postoperative status, transient increases in vagal tone)</td>
</tr>
</tbody>
</table>
Electrocardiographically, AV block is divided into first-, second- and third-degree (complete) heart block. First-degree heart block is reflected by a prolonged PR interval with conduction to the ventricle. The normal PR interval is defined electrocardiographically as a range from 120 to 200 ms. First-degree AV block is usually secondary to a delay of impulse conduction through the atrium or AV node (Fig. 3.1).

Second-degree AV block occurs when an atrial impulse that should be conducted to the ventricle is not conducted. The blocked impulses may be intermittent or frequent, at regular or irregular intervals, and preceded by fixed or lengthening PR intervals. A distinguishing feature of second-degree heart block is that impulse conduction occurs in a recurrent pattern rather than randomly (i.e., there is a recognizable pattern in the relationship between P waves and QRS complexes). Second-degree AV block is further classified as Mobitz type I or Wenckebach block, Mobitz type II block, or advanced second-degree AV block. Typical type I second-degree AV block is characterized by progressive PR interval prolongation that culminates with a nonconducted P wave (Fig. 3.2). In type II second-degree AV block, the PR interval remains constant before the nonconducted P wave (Fig. 3.3). The AV block is intermittent and generally repetitive. Advanced second-degree AV block is characterized by more than one nonconducted P wave in a row, with the presence of some conducted beats. Type II second-degree AV block often precedes the development of higher grades of AV block, whereas type I second-degree AV block is usually a less severe conduction disturbance that does not consistently progress to more advanced AV block.

Type I second-degree AV block with a normal QRS complex usually occurs at the level of the AV node, proximal to the bundle of His. AV block that is 2:1 may be type I or type II second-degree AV block. If the QRS complex is narrow, the block is more likely to be type I and one should search for transition of the 2:1 block to 3:2 block, during which the PR interval lengthens in the second cardiac cycle (Fig. 3.4). If the QRS complex is wide, the level of block is more likely to be distal to the His bundle and the escape focus is usually less reliable (Figs. 3.5 and 3.6). If preexisting bundle branch block is

Fig. 3.1 First-degree atrioventricular block. Here, with a PR interval of 300 ms.

Fig. 3.2 Type I (Wenckebach) second-degree atrioventricular block.
present, it is difficult to distinguish whether the block is located in the AV node or the His-Purkinje system. An attempt to alter the AV conduction ratio, either by exercise or by pharmacological means (e.g., with atropine) may allow localization of the conduction abnormality and assist with diagnosis. AV block that develops or worsens during exercise reflects conduction disease in the His-Purkinje system, which warrants implantation.
of a permanent pacemaker. During exercise, increased adrenergic drive facilitates AV nodal conduction; a diseased distal conduction system is unable to accommodate the increased rate and block occurs. In contrast, block in the AV node is more often physiological, due to increased vagal tone. Exercise changes the autonomic balance, leading to diminished vagal tone and improved conduction. Pacing is generally not needed in this situation.

Third-degree AV block (complete heart block) is defined by lack of conduction of atrial impulses to the ventricle (Fig. 3.7). It is important to distinguish this from AV dissociation due to a subsidiary pacemaker, usually junctional, that discharges more rapidly than the underlying sinus rate. In contrast, in third-degree AV block, the atrial rate is faster than the ventricular escape and there is no AV nodal conduction. Third-degree AV block may be congenital or acquired. In the congenital form, there is anatomical discontinuity in the conduction pathway. Pacing for this disorder was controversial for many years, because many patients with congenital complete heart block consider themselves asymptomatic, and there was conflicting information on the risk of sudden death associated with the condition. However, subjective improvement is usually noted once chronotropic competency is restored with permanent pacing. In addition, data support improved survival with permanent pacing. Indications for pacing in patients with congenital complete heart block are the presence of a wide QRS escape rhythm, complex ventricular ectopy, or ventricular dysfunction; or, in an infant, a ventricular rate of < 50–55 bpm, or a ventricular rate < 70 bpm if congenital heart disease is also present. In addition,

![Fig. 3.6 Subsequent electrocardiogram from the patient whose recording is shown in Fig. 3.5. Here, there is intermittent third-degree atrioventricular block with junctional escape beats (arrows indicate P waves).](image)

![Fig. 3.7 Third-degree atrioventricular block (complete heart block) is characterized by complete lack of conduction from atrium to ventricle and dissociation of atrial and ventricular activity. Here, the atrial rate is 27 bpm, whereas the ventricular rate is 75 bpm.](image)
a class IIa indication is congenital complete heart block after the first year of life with an average ventricular rate < 50 bpm or abrupt pauses in ventricular rate that are two or three times the basic cycle length. Finally, congenital complete heart block in an asymptomatic infant, child, adolescent, or young adult with an acceptable rate, narrow QRS, and normal ventricular function is considered a class IIb indication. In summary, pacing can be justified in any patient with congenital complete heart block.

Acquired third-degree AV block is due most commonly to aging, with or without calcification of the conduction system, or ischemic disease (e.g., myocardial infarction with damage involving the conduction system). Complete heart block has been associated with a number of systemic illnesses, many of which have been described in case reports (Table 3.2). Iatrogenic complete heart block can occur with open heart surgery or with inadvertent AV node ablation during treatment of supraventricular tachyarrrhythmias. Acquired complete heart block can be either intermittent or fixed. Patients may be asymptomatic or experience severe symptoms related to profound bradycardia, AV dissociation, or ventricular arrhythmias. It has been well documented that patients with complete heart block have improved survival with permanent pacing.

The most important factor in the decision to implant a pacemaker in a patient with AV block is whether or not the patient has symptoms that may be directly attributed to the arrhythmia. These symptoms may include overt or near syncope, lightheadedness, fatigue, activity intolerance, dyspnea, confusion or other cognitive changes, or symptoms of heart failure. Another important factor in the decision to implant a pacemaker for AV block is the expected irreversibility of the conduction disturbance. Potentially reversible causes of AV block include electrolyte abnormalities, Lyme disease, periprocedural hypothermia or inflammation, sleep apnea, and vagally mediated bradyarrhythmias related to medical illness or physiological changes in autonomic tone (e.g., bradycardia during emesis).

Atrial fibrillation with a slow ventricular response reflects the presence of AV block, although it is often categorized as sinus node dysfunction. Patients with this condition should receive a pacemaker if the bradycardia is causing symptoms (Fig. 3.8).

Areas of controversy exist in the indications for permanent pacing for AV block, and there are situations in which deviations from the ACC/AHA/HRS guidelines may be appropriate. Although the guidelines designate asymptomatic complete heart block with ventricular escape rates >40 bpm as a class IIa indication for pacing, we believe it should be a class I

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Table 3.2 Causes of acquired atrioventricular (AV) block

<table>
<thead>
<tr>
<th>Category</th>
<th>Causes</th>
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</thead>
<tbody>
<tr>
<td>Idiopathic (senescent) AV block</td>
<td>Idiopathic (senescent) AV block</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>Coronary artery disease</td>
</tr>
<tr>
<td>Calcific valvular disease</td>
<td>Calcific valvular disease</td>
</tr>
<tr>
<td>Postoperative or traumatic</td>
<td>Postoperative or traumatic</td>
</tr>
<tr>
<td>AV node ablation</td>
<td>AV node ablation</td>
</tr>
<tr>
<td>Therapeutic radiation to the chest</td>
<td>Therapeutic radiation to the chest</td>
</tr>
<tr>
<td>Infections</td>
<td>Infections</td>
</tr>
<tr>
<td>Syphilis</td>
<td>Syphilis</td>
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<tr>
<td>Diphtheria</td>
<td>Diphtheria</td>
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<tr>
<td>Chagas’ disease</td>
<td>Chagas’ disease</td>
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<tr>
<td>Tuberculosis</td>
<td>Tuberculosis</td>
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<tr>
<td>Toxoplasmosis</td>
<td>Toxoplasmosis</td>
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<tr>
<td>Lyme disease</td>
<td>Lyme disease</td>
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<tr>
<td>Viral myocarditis</td>
<td>Viral myocarditis</td>
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<tr>
<td>Infective endocarditis</td>
<td>Infective endocarditis</td>
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<tr>
<td>Collagen-vascular diseases</td>
<td>Collagen-vascular diseases</td>
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<tr>
<td>Rheumatoid arthritis</td>
<td>Rheumatoid arthritis</td>
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<tr>
<td>Scleroderma</td>
<td>Scleroderma</td>
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<tr>
<td>Dermatomyositis</td>
<td>Dermatomyositis</td>
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<tr>
<td>Ankylosing spondylitis</td>
<td>Ankylosing spondylitis</td>
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<tr>
<td>Polyarteritis nodosa</td>
<td>Polyarteritis nodosa</td>
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<tr>
<td>Systemic lupus erythematosus</td>
<td>Systemic lupus erythematosus</td>
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<tr>
<td>Marfan’s syndrome</td>
<td>Marfan’s syndrome</td>
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<tr>
<td>Infiltrative diseases</td>
<td>Infiltrative diseases</td>
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<tr>
<td>Sarcoïdosis</td>
<td>Sarcoïdosis</td>
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<tr>
<td>Amyloidosis</td>
<td>Amyloidosis</td>
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<tr>
<td>Hemochromatosis</td>
<td>Hemochromatosis</td>
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<tr>
<td>Malignant disease (lymphomatous or solid tumor)</td>
<td>Malignant disease (lymphomatous or solid tumor)</td>
</tr>
<tr>
<td>Neuromuscular diseases</td>
<td>Neuromuscular diseases</td>
</tr>
<tr>
<td>Progressive external ophthalmoplegia, Kearns-Sayre syndrome</td>
<td>Progressive external ophthalmoplegia, Kearns-Sayre syndrome</td>
</tr>
<tr>
<td>Myotonic muscular dystrophy</td>
<td>Myotonic muscular dystrophy</td>
</tr>
<tr>
<td>Peroneal muscular atrophy, Charcot-Marie-Tooth disease</td>
<td>Peroneal muscular atrophy, Charcot-Marie-Tooth disease</td>
</tr>
<tr>
<td>Scapuloperoneal syndrome</td>
<td>Scapuloperoneal syndrome</td>
</tr>
<tr>
<td>Erb’s (limb-girdle) dystrophy</td>
<td>Erb’s (limb-girdle) dystrophy</td>
</tr>
<tr>
<td>Drug effects</td>
<td>Drug effects</td>
</tr>
<tr>
<td>Digoxin</td>
<td>Digoxin</td>
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<tr>
<td>β-Blockers</td>
<td>β-Blockers</td>
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<tr>
<td>Calcium-blocking agents</td>
<td>Calcium-blocking agents</td>
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<tr>
<td>Amiodarone</td>
<td>Amiodarone</td>
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<tr>
<td>Procainamide</td>
<td>Procainamide</td>
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Class I agents: propafenone, encainide, flecaïnide
indication. The rate cut-off of 40 bpm is arbitrary, and it is not the escape rate per se that is critical to stability, but rather the site of origin of the escape rhythm (i.e., the AV node, His bundle, His-Purkinje system, or the ventricle). No definitive evidence exists regarding the conduction system localization or long-term stability of an escape rate of 40 bpm. Unfortunately, rate stability is not obvious or predictable. Clinically, one must grapple with whether the patient is truly asymptomatic and whether any diagnostic procedures, such as ambulatory monitoring or exercise testing, should be performed. No clinical trials or observational studies provide answers to these questions. From a practical and safety standpoint, irreversible acquired complete heart block should be a class I indication for permanent pacing.

Some asymptomatic patients with type I second-degree AV block have as poor a prognosis as patients with type II second-degree AV block, and permanent pacing improves survival in those patients >45 years old. Asymptomatic type I second-degree AV block not known to be at intra- or infra-Hisian levels is a class III indication (a contraindication) for pacing. Generally, an electrophysiology study to obtain His bundle recordings is not recommended. However, if an electrophysiology study is performed for other reasons and asymptomatic type I second-degree AV block with a narrow QRS is found to be infranodal, then pacemaker implantation is a class IIa indication according to the current guidelines. Because in this setting a patient is likely to have diffuse conduction system disease, we believe it is reasonable to consider this a class I indication.

In patients with specific neuromuscular diseases, pacing is advocated as a class I indication in third-degree AV block and as a class IIb indication in first-degree or second-degree AV block. The potential for sudden death in this group of patients is well documented. Because of the unpredictable progression to symptomatic bradycardia in patients with first-degree or second-degree AV block, the safest and most rational approach may be to offer pacing once any conduction abnormality is noted and subsequent follow-up reveals any progression. As discussed later, ICD therapy may also need to be considered for these patients.

**Acute myocardial infarction**

Conduction disturbances associated with acute myocardial infarction are largely related to the site of the infarction and the extent of myocardial injury. Given a greater awareness of the symptoms of acute myocardial infarction, the seeking of healthcare earlier, and more aggressive acute intervention, there are fewer extensive infarcts and permanent pacing is becoming less frequently required in this situation. Inferior myocardial infarctions are accompanied by a variety of conduction disturbances, including sinus bradycardia, sinus arrest, atrial fibrillation, atrial flutter and all grades of AV block. Anterior myocardial infarctions are more likely to be accompanied by AV block or intraventricular conduction defects (or both).

The indications for permanent pacemaker implantation after an acute myocardial infarction are based on the persistence of AV block and the presence of concomitant intraventricular conduction disturbances (Table 3.3). A general rule is that a permanent pacemaker is not indicated in a patient with an acute myocardial infarction if AV block is expected to resolve and if the conduction disturbance is not associated with a poor long-term prognosis (such as high-degree AV block in the setting of inferior myocardial infarction). The indications are less dependent on the presence or absence of symptoms, unlike pacing indications in other clinical settings. Clear class I indications are persistent severe conduction disturbances (second-degree AV block in the His-Purkinje system with bilateral bundle branch block, or third-degree AV block at or below the His-Purkinje system). Also considered a class I indication is transient advanced (second- or third-degree) infranodal AV block and
associated bundle branch block. A reasonable rule of thumb is useful in the patient with transient second- or third-degree A V block and associated bundle branch block: after an anterior wall myocardial infarction, a pacemaker is indicated if vagal block is excluded, because the block is almost certainly infranodal; after an inferior wall myocardial infarction, no pacemaker is needed, because the prognosis is good and is not adversely affected by the A V block. Also, it is important to note that a requirement for temporary pacing in a patient with acute myocardial infarction does not constitute an indication for permanent pacing for that patient.

**Chronic bifascicular and trifascicular block**

Bifascicular block is defined as a conduction disturbance of two fascicles of the ventricular conduction system (e.g., isolated left bundle branch block, right bundle branch block [RBBB] and left anterior fascicular block, or RBBB and left posterior fascicular block). Trifascicular block is defined as a conduction disturbance in all three fascicles (either simultaneously or in successive electrocardiograms) or the presence of first-degree A V block and bifascicular block. A special example of trifascicular block is alternating bundle branch block (also referred to as bilateral bundle branch block).

Indications for permanent pacing in patients with bifascicular or trifascicular block depend on the risk of development of transient or permanent advanced A V block (Table 3.4). This is because patients with bifascicular or trifascicular block and advanced A V block have higher rates of sudden death and all-cause death. Syncope is common in patients with bifascicular block, but it is not associated with an increased risk of sudden death. Thus, defining the cause of syncope in patients with bifascicular and trifascicular block is important, and a permanent pacemaker should be implanted if transient or persistent advanced A V block is documented. If, after investigation, the cause of syncope is undetermined, then implantation of a permanent pacemaker is reasonable (class IIa indication), since the syncope may be due to intermittent advanced A V block. It is notable, however, that the incidence of progression of bifascicular block to complete heart block is low, and that there are no reliable predictors of death from progression of the conduction disease. Patients with RBBB and left anterior hemiblock are at higher risk of cardiovascular death, but this is not specifically due to bradycardia or conduction disease, and permanent pacing is not recommended in these patients in the absence of symptoms or advanced A V block.

Although the incidence of progression to advanced A V block is relatively low in patients with bifascicular or trifascicular block, measurement of the HV interval (a measure of conduction of the His-Purkinje system) may rarely help in identifying patients at higher risk of developing symptomatic advanced A V block. The degree of HV interval lengthening necessary to justify pro-
Phylactic pacemaker placement is controversial. Some have advocated pacing for an HV interval of > 100 ms, and others have considered pacing for an HV interval of > 70 ms, especially if the patient is to receive cardioactive drugs that have the potential for further impairment of the conduction system. The development of nonphysiological infra-His block with increasingly rapid atrial pacing during an electrophysiology study may reflect increased risk of progression to advanced AV block or symptomatic bradycardias (and thus has a class IIa indication for permanent pacing); however, failure to develop infra-His block is not a reliable indicator that advanced AV block will not develop in the future.

Sinus node dysfunction

Sinus node dysfunction (sick sinus syndrome) includes a variety of cardiac arrhythmias, including sinus bradycardia (Fig. 3.9), sinus arrest (Fig. 3.10) and sinoatrial block. It also includes the tachycardia-bradycardia syndrome, in which paroxysmal supraventricular tachycardias alternate with periods of bradycardia or asystole (Fig. 3.11). Indications for permanent pacing in patients with sinus node dysfunction are summarized in Table 3.5.

The definition of clinically significant bradycardia varies, but it is generally agreed to denote rates of < 40 bpm during waking hours. Sinus pauses of ≥ 3 s or symptomatic sinus rates < 40 bpm in the awake patient are indications for permanent pacing. It should be recognized, however, that there is disagreement about the absolute cycle length of an asystolic period that requires pacing. The patient’s entire clinical condition should be considered, including age, associated diseases, medications and symptoms. For example, sleeping endurance athletes have sinus rates as low as 30 bpm, but are asymptomatic and do not require pacing. Sinus bradycardia during sleep in an asymptomatic patient is not an indication for pacing. Sleep apnea is a potential cause of

![Fig. 3.9 Sinus bradycardia with a rate of 39 bpm. The patient had symptoms that resolved after DDD pacing.](image-url)
Indications for Pacemakers, ICDs and CRT

nocturnal bradycardias that should be treated if considered clinically significant. In sinus node dysfunction, correlation of symptoms with the specific arrhythmia is essential. The use of ambulatory electrocardiographic monitors and event loop recorders is helpful to document patients’ rhythms during specific symptoms. Patients who develop symptomatic bradycardias due to required pharmacological therapy for other conditions require permanent pacing, but pacing is unnecessary if the bradycardias are asymptomatic. Chronotropic incompetence may be demonstrated by the absence of an appropriate physiological increase in sinus rate with exercise, and it is a class I indication for implantation of a rate-responsive permanent pacemaker.

When permanent pacing is indicated for patients with sinus node dysfunction, special thought should be given to selection of the appropriate device and programming. Single-chamber ventricular pacemakers are not appropriate for patients with sinus node dysfunction. Single-chamber atrial pacemakers with rate-responsive capability are attractive due to their relative simplicity.
and lower cost compared with dual-chamber pacemakers. In patients without coexisting AV block, AAIR pacing may be appropriate. The major concern is that AV block develops in patients with sinus node dysfunction after atrial pacemaker implantation at an annual rate of 0.6–5%, with a higher risk in patients with preexisting bundle branch block.20,21 Although this risk is relatively low, there is some benefit to provide ventricular pacing in the event it is needed. Conversely, pacing the ventricle unnecessarily can result in a higher incidence of atrial fibrillation and congestive heart failure.22–25 Avoidance of ventricular pacing can be accomplished by programming a very long AV interval, or by use of an algorithm that effectively avoids ventricular pacing except when absolutely required by predefined criteria (discussed further in Chapter 8 on Programming). Thus, the best options for sinus node dysfunction are either a single-chamber atrial pacemaker or a dual-chamber pacemaker that is programmed either manually or via an algorithm to avoid ventricular pacing. The decision must be made on an individual basis, with consideration of the extent of conduction disease, severity of its clinical manifestations, comorbidities, risk of future revision to a dual-chamber system, and the patient's own preferences. In the USA, dual-chamber pacemakers are most commonly used.

**Neurally mediated reflex syncope**

Permanent pacing is indicated in the proper clinical setting for some types of neurally mediated reflex syncope, which includes carotid sinus syndrome and neurocardiogenic (vasovagal) syncope (Table 3.6). Understanding the physiology involved in these different types of syncope is crucial to understanding their clinical manifestations and the appropriateness of permanent pacing.26

<table>
<thead>
<tr>
<th>Table 3.5 Indications for pacing in sinus node dysfunction</th>
</tr>
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<tbody>
<tr>
<td><strong>Class I</strong></td>
</tr>
<tr>
<td>1 Sinus node dysfunction with symptomatic bradycardia or frequent symptomatic sinus pauses. In some patients, bradycardia is iatrogenic and occurs as a consequence of essential long-term drug therapy for which there is no acceptable alternative. The definition of bradycardia varies with the patient’s age and expected heart rate.</td>
</tr>
<tr>
<td>2 Symptomatic chronotropic incompetence.</td>
</tr>
<tr>
<td><strong>Class IIa</strong></td>
</tr>
<tr>
<td>1 Sinus node dysfunction occurring spontaneously or as a result of necessary drug therapy, with heart rate &lt; 40 bpm, when a clear association between significant symptoms consistent with bradycardia and the actual presence of bradycardia has not been documented.</td>
</tr>
<tr>
<td>2 Syncope of unknown etiology when sinus node dysfunction is provoked or discovered during electrophysiological testing that is felt to be clinically significant.</td>
</tr>
<tr>
<td><strong>Class IIb</strong></td>
</tr>
<tr>
<td>In minimally symptomatic patients, chronic heart rate &lt; 40 bpm while awake.</td>
</tr>
<tr>
<td><strong>Class III</strong></td>
</tr>
<tr>
<td>1 Sinus node dysfunction in asymptomatic patients.</td>
</tr>
<tr>
<td>2 Sinus node dysfunction in patients with symptoms that are clearly documented in the absence of bradycardia.</td>
</tr>
<tr>
<td>3 Sinus node dysfunction with symptomatic bradycardia due to non-essential drug therapy.</td>
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<tr>
<th>Table 3.6 Indications for pacing in neurally mediated reflex syncope</th>
</tr>
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<tbody>
<tr>
<td><strong>Class I</strong></td>
</tr>
<tr>
<td>1 Recurrent syncope caused by carotid sinus hypersensitivity, defined as minimal carotid sinus pressure inducing ventricular asystole of ≥ 3 s in patients not receiving medication that depress the sinus node or AV conduction.</td>
</tr>
<tr>
<td><strong>Class IIa</strong></td>
</tr>
<tr>
<td>Syncope in the absence of definite provocative event with a pause of ≥ 3 s with carotid massage.</td>
</tr>
<tr>
<td><strong>Class IIb</strong></td>
</tr>
<tr>
<td>Recurrent symptomatic neurocardiogenic syncope with a cardioinhibitory response during tilt-table testing.</td>
</tr>
<tr>
<td><strong>Class III</strong></td>
</tr>
<tr>
<td>1 A cardioinhibitory response during carotid sinus stimulation without symptoms or vague symptoms.</td>
</tr>
<tr>
<td>2 Situational vasovagal syncope in which avoidance behavior is effective.</td>
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</table>
The carotid sinus reflex is the physiological bradycardic response to stimulation of the carotid sinus baroreceptors. Although this reflex is normal, some individuals have an exaggerated response that may be pathological. This reflex has two components. The first, a cardioinhibitory response, results from increased parasympathetic tone and may be manifested by any or all of the following: sinus bradycardia, sinus arrest (Fig. 3.12), PR prolongation and advanced AV block. The second component, a vasodepressor response, is due to decreased sympathetic activity and results in peripheral vasodilation and hypotension (independently of changes in heart rate). Pure cardioinhibitory or pure vasodepressor responses may occur, but a mixed response is most common.

The definitions of normal and abnormal responses to carotid sinus stimulation are somewhat arbitrary. Generally, an abnormal response is defined as development of ventricular asystole of ≥3 s and/or a decrease in blood pressure of 30–50 mm Hg. As many as 40% of patients ≥65 years old have carotid sinus hypersensitivity, thus the causal relationship between carotid sinus hypersensitivity and symptoms is critical to decisions regarding therapy. Patients with syncope may have a typical history related to a tight collar or neck extension, but more commonly definite provocative maneuvers cannot be identified. If carotid sinus massage reproduces the patient’s symptoms and is associated with significant cardioinhibition or vasodepression, a diagnosis of carotid sinus syndrome can be made and treatment should be initiated. If carotid sinus massage yields a positive result but does not reproduce the patient’s symptoms, a hypersensitive carotid reflex has been demonstrated, but it may not be of clinical significance, and other causes for syncope should be investigated. Certain situations exist where it is difficult to correlate symptoms. If the result of carotid sinus massage is negative, tilt testing may be indicated to identify other mechanisms of neurally mediated reflex syncope.

Neurocardiogenic (vasovagal) syncope may be benign or malignant. The exact pathophysiology of these syndromes is unclear, but important mechanisms include prolonged orthostatic stress, venous pooling, activation of cardiac mechanoreceptors, and an abnormal decrease in sympathetic activity. These processes result in bradycardia, peripheral vasodilation, and syncope. The episodes can be triggered by various stimuli, such as pain, warm environments, visceral sensations, and acute psychological stress. Manifestations include a prodrome of nausea, diaphoresis and light-headedness followed by a brief episode of unconsciousness, after which there is quick and complete cognitive recovery. In the elderly, prodromal symptoms are often absent and loss of consciousness may occur suddenly, mimicking other causes of syncope.

Head-up tilt-table testing can reproduce neurocardiogenic syncope in susceptible patients. Passive head-up tilt creates an exaggerated orthostatic state by stressing the autonomic system in the absence of the usual compensatory skeletal muscle tone. This triggers the cascade of mechanisms described above. Tilt-table testing can help identify whether the predominant cause of symptoms is vasodepressor or cardioinhibitory in origin (Fig. 3.13), which is important because therapy may differ for each situation.

Benign vasovagal syncope, or a “simple faint,” usually does not require therapy. Associated rhythms are usually prolonged sinus arrest without a junctional or ventricular escape rhythm (Fig. 3.14). Situational syncope, which occurs stereotypically with swallowing, cough, micturition or defecation, does not significantly increase mortality and usually does not require permanent pacing. However, recurrent events may be disabling to some patients, and the situations during which the events occur, such as operation of a motor vehicle or heavy machinery, may predispose the patient or bystanders to danger. In these cases, permanent pacemaker implantation may be an effective means of preventing syncope; however, this is unproven and controversial.
In patients with significant cardioinhibition, permanent pacing is a seemingly intuitive intervention. Despite early promise, two randomized trials have shown that permanent pacing does not improve outcomes in patients with neurocardiogenic syncope. The data suggested a benefit with permanent pacing only in patients with severe cardioinhibition (i.e., asystole) compared with those with less severe bradycardias during tilt-table testing. The indications for permanent pacing in patients with neurocardiogenic syncope include (i) a significant cardioinhibitory component with severe bradycardia or asystole during syncope, (ii) lack of a prodrome, and (iii) recurrent syncope despite maximally tolerated medical therapy. Pacemakers with the capability of triggering a faster pacing rate for a defined period of time in response to a specified sudden drop in heart rate have been developed specifically for this syndrome. For all patients with neurocardiogenic syncope, principal therapy includes diet and lifestyle modification, compression stockings, and pharmacological agents. Most patients have a mixed cardioinhibitory and vasodepressor response, and it is most common for hypotension to precede bradycardia during an episode, so the medical therapies must continue even after pacemaker implantation.

**Tachyarrhythmias**

Previously, national guidelines recognized a number of class I indications for implantation of permanent pacemakers that detect and pace to terminate tachyarrhythmias. These devices initiate programmed stimulation or bursts of rapid pacing to terminate reentrant arrhythmias.
mias after their automatic detection or after user activation (by magnet application). However, due to the wide availability and success of treatment by catheter ablation, there are currently no class I indications for permanent pacemakers to treat supraventricular tachyarrhythmias (Tables 3.7 and 3.8). Indications for device implantation in the treatment of ventricular tachyarrhythmias is discussed in the subsequent section, Indications for the implantable cardioverter-defibrillator.

The implantation of a permanent pacemaker to prevent or treat tachyarrhythmias is still indicated in rare situations. A class I indication is for patients with or without a long QT interval who experience recurrent pause-dependent ventricular tachycardia (VT). The device of choice in these individuals is an implanted cardioverter-defibrillator with a bradycardia pacing function. The use of pacemakers to terminate supraventricular tachycardias or to prevent AV node reentrant or AV reentrant tachycardias when drugs or catheter ablation fail are class IIa indications. Atrial fibrillation, atrial flutter and atrial tachycardia are common in patients with sinus node dysfunction and an

Table 3.7  Indications for pacemakers to terminate tachycardia

<table>
<thead>
<tr>
<th>Class</th>
<th>Indication</th>
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<tbody>
<tr>
<td>Class I</td>
<td>None.</td>
</tr>
<tr>
<td>Class IIa</td>
<td>Symptomatic recurrent supraventricular tachycardia that is reproducibly terminated by pacing in the unlikely event that catheter ablation and/or drugs fail to control the arrhythmia or produce intolerable side effects.</td>
</tr>
<tr>
<td>Class III</td>
<td>The presence of accessory pathways with the capacity for rapid anterograde conduction.</td>
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Atrial antitachycardia pacing therapy is about 50% effective in terminating organized atrial arrhythmias, and in subsets of patients this success translates into a significant reduction in the overall burden of atrial arrhythmias. However, important improvements in clinical outcomes have not been shown, limiting the role of atrial therapy practice to patients who also have another device indication. Implantation of a pacemaker for prevention, reduction or treatment of atrial arrhythmias is a class IIb indication. In patients with an implanted pacemaker and recurrent symptomatic atrial arrhythmias, consideration could be given to the use of a device with atrial antitachycardia pacing capabilities. Also, some devices have specific programmable algorithms to reduce the burden of atrial arrhythmias. The algorithms attempt to limit the variability in rhythm that promotes atrial fibrillation and to overdrive suppress triggers, and they include rate-adaptive atrial overdrive pacing, pacing to suppress premature atrial complexes, and rate response to limit the rate of decrease in heart rate after exercise. The ADOPT trial and preliminary results of the Atrial Fibrillation Therapy (AFT) study (unpublished) showed that the addition of a rate-adaptive atrial pacing algorithm to dual-chambered rate-responsive pacemakers reduced the frequency of symptomatic atrial fibrillation. Currently, there are insufficient data to support the use of permanent pacemakers solely to prevent or reduce the burden of atrial fibrillation.

### Congestive heart failure

One-quarter to one-third of patients with congestive heart failure have left bundle branch block, which has been associated with increased mortality. With left bundle branch block, the left ventricle is initially activated at the anteroseptum, with delayed activation and contraction of the left ventricular lateral wall (Fig. 3.15). This dyssynchrony of ventricular activation leads to impaired pumping efficiency. In selected patients with refractory heart failure, pacing both ventricles (referred to as biventricular pacing or cardiac resynchronization therapy [CRT]) restores synchrony and improves exercise tolerance, clinical and biochemical markers of heart failure, ejection fraction, quality of life, and survival. The left ventricle is most commonly paced by placing an electrode in a coronary sinus tributary (Fig. 3.16). The implant technique is discussed in detail in Chapter 5. Most patients with depressed ventricular function and heart failure are also at risk for sudden cardiac death due to a potentially lethal arrhythmia (e.g., torsades de pointes). The implantable cardioverter-defibrillator (ICD) is an effective therapy for primary and secondary prevention of sudden death due to ventricular fibrillation or ventricular tachycardia. It is a Class I indication. The indications for ICD therapy in patients with HCM are discussed in the subsequent section, Indications for the implantable cardioverter-defibrillator.

### Hypertrophic cardiomyopathy

Standard indications for permanent pacing related to sinus node dysfunction or AV block, discussed above, apply equally to patients with hypertrophic cardiomyopathy (HCM). Patients who undergo transcatheter septal ablation to alleviate an outflow gradient have an estimated 11% chance of developing subacute complete heart block, and they are treated with dual-chamber permanent pacing. Although an additional class IIb indication exists for the use of dual-chamber pacing in patients with HCM who have a demonstrable significant left ventricular outflow gradient and continue to have symptoms despite medical and/or surgical treatment, randomized trials have not convincingly demonstrated that this intervention significantly improves symptoms or outcomes. Indications for ICD therapy in patients with HCM are discussed in the subsequent section, Indications for the implantable cardioverter-defibrillator.
to ventricular tachyarrhythmias, and they commonly receive a device capable of both resynchronization and defibrillation (CRT-D) (as opposed to a device solely for resynchronization pacing [CRT-P]). The indications for CRT-D are discussed in the subsequent section, Indications for the implantable cardioverter-defibrillator.

It is reasonable to consider the indications for cardiac resynchronization in two broad categories:

1. **Patients with conventional indications for anti-brady-cardia pacing who also have ventricular dysfunction.**

2. **Patients with end-stage heart failure who do not have ventricular dysfunction.**

Standard indications for permanent pacing related to sinus node dysfunction or AV block, discussed earlier, apply equally to patients with heart failure. However, there are special considerations when pacing patients with depressed ventricular function. Pacing the right ventricle activates the right ventricle first, resulting in a left bundle branch block morphology on the surface electrocardiogram. As with naturally occurring left bundle branch block, dyssynchrony is present during right ventricular apex (RVA) pacing, since the left

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**Fig. 3.15** Sequence of left ventricular activation in patients with left bundle branch block. The left ventricular lateral wall is depolarized last and leads to dysynchronous global ventricular function that impairs hemodynamics. (Reproduced from: Jarcho J. Biventricular pacing. N Engl J Med 2006; 355:288-94, Massachusetts Medical Society.)
The ventricular septum is depolarized before the left ventricular lateral wall. Ventricular dyssynchrony caused by RVA pacing results in asymmetric hypertrophy and redistribution of cardiac mass, mitral regurgitation, increased left atrial diameter, and reduced ejection fraction. In patients with ejection fraction under 40%, a prospective, randomized trial has found that the DDD mode incurs a greater risk for the development of atrial fibrillation, progression of heart failure, heart failure hospitalizations and death than backup VVI pacing. Post hoc analysis of several trials has shown this risk results from RVA pacing. This concept is further supported by the observation that upgrading patients with refractory congestive heart failure and chronic RVA pacing leads improves New York Heart Association (NYHA) functional class, ejection fraction and other clinical parameters. The risk of adverse clinical outcomes as a consequence of right ventricular pacing is a function of a patient’s substrate (“dyssynchrony reserve”) and the “dose” of dyssynchrony delivered. Patient risk factors for RVA pacing-induced heart failure include a low ejection fraction, history of heart failure, and wide baseline QRS. The “dose” of dyssynchrony is a function of the paced QRS duration and the RVA pacing frequency; AV dysynchrony also plays a role. These findings explain

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**Fig. 3.16** Typical biventricular pacemaker lead placement. Three pacemaker leads are implanted via the extrathoracic veins and superior vena cava. One lead is placed in the right atrium, a second lead is placed across the tricuspid valve into the right ventricle, and a third lead is placed via the coronary sinus into a venous tributary on the lateral wall of the left ventricle. Ideally, the left and right ventricular pacing leads are spatially separated as shown on the posteroanterior and lateral chest radiographs. This provides nearly simultaneous activation of the ventricles and produces a narrow QRS complex on the electrocardiogram.
the clinical observation that most patients with preserved ventricular function and no history of heart failure tolerate pacing systems incorporating an RVA lead well, whereas patients with a history of heart failure and low ejection fraction are prone to RVA pacing-associated heart failure exacerbation. In practice, in patients with existing pacemakers who have a low ejection fraction (< 40%) and persistent heart failure, strong consideration is given to revising (upgrading) the system to a biventricular pacemaker. For de novo implants for bradycardia in patients with clinical heart failure and ejection fraction ≤ 40%, we offer CRT when the ventricular pacing frequency is anticipated to exceed 40%. This necessarily includes patients undergoing AV nodal ablation. The exception to the rule are patients in whom tachycardia-induced cardiomyopathy is implicated. A third of such patients will experience improved ventricular function following AV node ablation with RVA pacing alone due to improved rate control and ventricular regularity. At this time CRT is not indicated in patients with only mildly depressed ventricular function (EF 40–55%). In such patients cardiac function is periodically assessed and the pacing system upgraded to CRT if function declines.

2. Patients with refractory heart failure and no pacing indication. Prospective, randomized, clinical trials have demonstrated that CRT improves left ventricular systolic function, heart failure symptoms, exercise tolerance, quality of life, and survival. Patient selection for CRT is based on the inclusion criteria used in these trials and extrapolating based on these data (Tables 3.9 and 3.10). Generally accepted criteria include non-ischemic or ischemic cardiomyopathy, systolic dysfunction with left ventricular ejection fraction ≤ 35%, stable NYHA class III or IV heart failure symptoms, sinus rhythm, a QRS duration > 120 ms, and maximally tolerated medical therapy (including dietary management, β-blockers, angiotensin-converting enzyme inhibitors or angiotensin receptor blockers, aldosterone antagonists, and diuretics). The patients’ symptoms and therapy should be stable for 3 months prior to consideration of biventricular pacing.

QRS duration

The presumed mechanism by which patients improve with CRT is restoration of synchrony. The QRS duration has been used as a surrogate for electrical and mechanical dyssynchrony in nearly all large randomized clinical trials. The CARE HF trial required echocardiographic measures of dyssynchrony in patients with QRS 120–149 ms, but this constituted a small minority of patients; patients with a QRS > 150 ms required no additional measurements. Limitations to use of the QRS duration as a marker for dyssynchrony exist, and indeed may partially explain the nonresponse rate to CRT of approximately 30% in most trials. Approximately one-third of patients with heart failure and QRS > 150 ms do not have dyssynchrony, and over one-quarter of patients with advanced heart failure and a normal QRS have dyssynchrony. However, most patients with native QRS > 150 ms and paced QRS > 200 ms have mechanical dyssynchrony. If ancillary studies (e.g., tissue Doppler, myocardial strain imaging, or cardiac magnetic resonance imaging) demonstrate normal or adequate ventricular synchrony, then intuitively “resynchronization” with biventricular pacing would not be necessary. However, because the large, prospective randomized trials enrolled patients predominantly based on the pres-
ence of a wide QRS, and the studies utilizing imaging modalities are small and generally from single specialized centers, patients with a QRS > 120 ms are offered CRT if they are otherwise appropriate candidates. It is reasonable to counsel patients with a wide QRS, but with imaging studies that do not demonstrate dysynchrony, that they may be less likely to respond to CRT. Conversely, emerging data suggest that patients with a narrow QRS who have echocardiographic evidence of left ventricular dysynchrony benefit from CRT. The role of imaging to establish the degree of dysynchrony to guide patient selection is not fully established, and ongoing studies will better define the strategies to select patients for CRT based on measures of dysynchrony.

Severity of CHF

Patients without heart failure symptoms (i.e., NYHA class I, stage B or C) have not been included in published CRT trials with the exception of the PAVE trial, in which biventricular pacing was delivered after AV nodal ablation (see below). While this population has little to gain in terms of symptoms improvement, it is unknown whether they would experience a reduction in heart failure hospitalizations or an improvement in survival with CRT, and, if so, what the magnitude of such benefits would be relative to the risks and costs. Currently, the role of CRT in patients with mild heart failure symptoms (i.e., NYHA class II, early stage C) is controversial. Only a few trials to date have enrolled these patients. Federal government reimbursement for implantation of biventricular pacemakers is currently approved only for patients with NYHA class III or IV symptoms. Most implants are performed in patients with NYHA class III–IV, stage C heart failure. Ongoing randomized controlled trials aim to clarify the utility of CRT in patients with class I or II symptoms.

Right bundle branch block

While inclusion in the randomized trials of CRT was based on a prolonged QRS duration irrespective of morphology, only 5–13% of patients had RBBB. Thus, the effects of biventricular pacing for patients with RBBB are less well studied than left bundle branch block. Nevertheless, current indications are predicated on the original trial inclusion criteria, i.e., the QRS duration regardless of its morphology, and thus patients with RBBB who meet the other criteria should be offered CRT. An echo dysynchrony study prior to implantation may be helpful in making the determination to proceed in difficult cases.

Atrial fibrillation

Patients with atrial fibrillation thus far have been excluded from all but a few observational studies and three small controlled trials assessing CRT. MUSTIC-AF was a crossover trial of 59 patients (of whom only 58% completed the trial) with NYHA class III heart failure, chronic atrial fibrillation, right ventricular pacemakers, and wide-paced QRS complexes (> 200 ms). Those with effective CRT therapy had improved exercise tolerance and decreased hospitalizations compared with right ventricular pacing alone. The PAVE trial was a randomized trial of 184 patients with atrial fibrillation undergoing AV node ablation for control of rapid ventricular rates. Those who received CRT, particularly those with decreased left ventricular systolic function and NYHA class II or III heart failure, had improved exercise tolerance and increased left ventricular ejection fraction compared with those who received a conventional right ventricular lead. A meta-analysis has demonstrated a trend toward reduced all-cause mortality in patients with CHF and AF treated with CRT. While awaiting larger trials in broader groups of patients, we recommend that patients with atrial fibrillation receive CRT if they meet all other criteria (class IIa indication). In this population, care is taken to insure a high frequency of ventricular pacing (> 90%) following device implantation.

Nonresponders

Up to 25% of patients in clinical trials did not respond to CRT. Reasons for nonresponse to CRT include poor patient selection (absence of electrical or mechanical dysynchrony), poor lead location, or insufficient pacing frequency. Management of non-responders is covered in Chapter 10, Troubleshooting.

Pacing after cardiac transplantation

The incidence of bradyarrhythmias after cardiac transplantation is 8–23%. Sinus node dysfunction is the usual cause of bradyarrhythmias in these patients, but risk factors for its development are unknown. Most bradyarrhythmias are transient, and half will resolve
6–12 months after transplantation.71 Pacing is indicated (class I) for symptomatic persistent inappropriate bradyarrhythmias or chronotropic incompetence are not expected to resolve, and also indicated for sinus node dysfunction or AV block, as discussed above for the general population.1 Pacing may be considered (class IIb) when relative bradycardia is recurrent or prolonged, thus preventing rehabilitation on discharge after transplantation, and when syncope occurs after cardiac transplantation even if a bradyarrhythmia has not been documented.

Indications for the implantable cardioverter-defibrillator

Development of the ICD was pioneered by Dr Michel Mirowski in the late 1960s after the death of a close friend and mentor, who had been hospitalized with recurrent ventricular tachyarrhythmias. His frustration with the limitations of available therapies for high-risk patients led to the concept of an implantable device that continuously monitors cardiac rhythm and delivers defibrillating shocks for ventricular tachyarhythmias when they occur. During the 1970s, experimental models were built and refined, leading to the first implantation of an ICD in 1980 in a patient with two previous cardiac arrests.72 By the next decade, the indications had broadened to include patients with drug-refractory ventricular fibrillation (VF) or VT, patients with VT or VF in whom arrhythmias could not be induced (so that electrophysiological study could not assess drug effectiveness), and patients who did not tolerate antiarrhythmic drugs.73 With additional significant refinements in ICD technology, disillusionment with effectiveness of drug therapy, and the publication of prospective clinical data demonstrating effectiveness, the ICD has become the gold standard therapy for patients at high risk for lethal arrhythmias.1,2

It is useful to consider ICD indications in terms of secondary or primary prevention. Secondary prevention refers to ICD use in patients who have previously experienced an out-of-hospital cardiac arrest or life-threatening arrhythmia. Primary prevention refers to ICD use in patients who may have significant cardiovascular disease, but who have never experienced a life-threatening arrhythmia. Current indications for the ICD for primary and secondary prevention of sudden cardiac death are detailed below.

**Secondary prevention**

Indications for an ICD for secondary prevention of sudden cardiac death are summarized in Table 3.11. The ICD has a well-established survival benefit in patients with prior out-of-hospital cardiac arrest, documented VF or VT, or syncope in association with structural heart disease and inducible ventricular arrhythmias.2 Patients who have a cardiac arrest due to VF or VT without acute myocardial infarction or a clearly reversible cause are at high risk for recurrent cardiac arrest (30–50% recurrence within 2 years). Randomized controlled trials have shown more than a 50% relative risk reduction of death in patients with prior events who receive an ICD compared with patients treated empirically with amiodarone or treated with sotalol guided by electrophysiological testing.74 Furthermore, they showed that the ICD was effective regardless of the presence or absence of structural heart disease, revascularization, or type of dysrhythmia (VT or VF).74 Electrophysiological study is not required, as it does not alter the decision to implant an ICD in a patient with a fatal or near-fatal arrhythmic event. However, the study may guide ICD programming, inform device selection (by assessing

<table>
<thead>
<tr>
<th>Table 3.11 Indications for the implantable cardioverter-defibrillator for secondary prevention of sudden cardiac death</th>
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<tbody>
<tr>
<td>1. Cardiac arrest due to ventricular fibrillation (VF) or ventricular tachycardia (VT) not within 24–48 h of acute myocardial infarction and not due to a transient or reversible cause. <strong>Class I</strong></td>
</tr>
<tr>
<td>2. Cardiac arrest when clear evidence of acute myocardial ischemia is documented to immediately precede the onset of VF, but when coronary revascularization cannot be carried out. <strong>Class I</strong></td>
</tr>
<tr>
<td>3. Spontaneous sustained VT or VF in patients with structural heart disease. <strong>Class I</strong></td>
</tr>
<tr>
<td>4. Unexplained syncope with clinically-relevant, significant VT or VF induced at electrophysiology study. <strong>Class I</strong></td>
</tr>
<tr>
<td>5. Sustained ventricular arrhythmias in patients with ischemic or non-ischemic cardiomyopathy but normal or near-normal left ventricular function. <strong>Class IIa</strong></td>
</tr>
<tr>
<td>6. Unexplained syncope in patients with significant left ventricular dysfunction who are receiving chronic optimal medical therapy. <strong>Class IIa</strong></td>
</tr>
</tbody>
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concomitant need for pacing modalities), and detect VT that is amenable to ablation.

In patients with ventricular dysfunction and hemodynamically stable VT, catheter-based ablation is palliative, and an ICD remains indicated due to the risk of subsequent unstable VT. In patients with stable VT and preserved left ventricular function (i.e., ejection fraction > 40%), antiarrhythmic drugs or catheter-based treatments are often helpful; however, the decision not to implant an ICD after the occurrence of sustained VT is controversial. Exceptions to this rule include the well-characterized idiopathic VTs in the setting of a structurally normal heart, for which catheter ablation is accepted primary therapy (discussed further, below).

Syncope of undetermined etiology in patients with either ventricular dysfunction or normal ventricular function but hemodynamically significant sustained VT or VF induced at electrophysiology study is an indication for an ICD, regardless of adjunctive drug therapy.

Lastly, for patients at high risk of recurrent VT or VF, but in whom comorbidities preclude immediate ICD implantation or for whom the risk is transient (i.e., awaiting cardiac transplantation or in the immediate period after acute myocardial infarction in certain situations), a possible option is a wearable cardioverter defibrillator, such as one in the form of a vest currently approved by the US Food and Drug Administration. Experience with this therapy is limited, and its role in general practice is yet to be defined.

**Primary prevention**

Given that the majority of sudden cardiac death occurs in people without a prior event, identification of individuals at high risk of a first potentially fatal arrhythmia who would benefit from prophylactic implantation of an ICD is paramount. A large proportion of these events occur in asymptomatic community-dwelling individuals without recognized cardiac disease. A smaller proportion of these events occur in asymptomatic or symptomatic patients with established cardiac disease, and it is in this population that major strides have been made in primary prevention of sudden cardiac death. Importantly, if a strategy of implanting ICDs only in patients with an antecedent life-threatening event were adopted, 60–90% of individuals who might benefit from the device would not receive therapy, as they would have succumbed to the initial event. As shown in Fig. 3.17, a strategy that focuses only on the highest risk patients will provide benefit to those individuals, with little impact on sudden death rates for society as a whole; conversely, implanting ICDs in excessively broad populations results in exposing individuals at low risk to device implantation. Strategies focusing on patients with depressed ventricular function and heart failure lead to device placement in patients who benefit and whose conditions are sufficiently common for a societal benefit to be seen. Thus, it has become increasingly clear that the severity of underlying organic heart disease is an important prognostic factor and influences the decision to implant an ICD. The greatest information regarding future sudden death risk is available for patients with coronary artery disease, dilated cardiomyopathy, or congestive heart failure. A disease-specific approach to selecting candidates for prophylactic ICD follows, and indications are summarized in Table 3.12.

**Coronary artery disease**

Patients with a history of myocardial infarction and reduced left ventricular function are at increased risk of sudden cardiac death. This occurs most commonly as a result of VT due to reentry around infarct scars or electrically heterogeneous areas in infarct border zones. Other arrhythmia mechanisms are contributory. In the past, patients with prior myocardial infarction, ventricular dysfunction and nonsustained VT were further risk stratified by assessing the inducibility of VT or VF during an electrophysiology study. Randomized trial data from the MADIT study and observational data from a subanalysis of the MUSTT study showed that patients with inducible VT or VF survived longer when treated with an ICD compared with medical therapy. However, this approach had important limitations. First, those patients with ventricular dysfunction who had negative electrophysiological studies and were not treated with an ICD still had a high risk of death. Additionally, a strategy to determine ICD utility that required an invasive electrophysiology study in all potential candidates had inherent practical limitations.

Consequently, the pivotal MADIT II trial assessed the benefit of the ICD in patients with prior myocardial infarction and significant left ventricular dysfunction (ejection fraction ≤ 30%) without requiring an electrophysiology study. It showed that ICD implantation led to a 31% relative risk reduction (a 6% absolute risk
reduction) in death. These findings were extended by the SCD-HeFT trial, which assessed the benefits of the ICD in patients with ischemic or nonischemic heart disease, significant left ventricular dysfunction (ejection fraction ≤ 35%), and NYHA functional class II or III.44 SCD-HeFT found that therapy with an ICD compared with amiodarone or standard medical treatment yielded a 23% relative risk reduction (7% absolute risk reduction) in death. Subsequent to these trials, routine invasive electrophysiology has been abandoned.

**Table 3.12** Indications for the implantable cardioverter-defibrillator for primary prevention of sudden cardiac death in patients with ischemic or non-ischemic cardiomyopathy

1. Left ventricular ejection fraction < 35% and NYHA functional class II–III symptoms in patients with a myocardial infarction ≥ 40 days prior and no coronary revascularization in the last 3 months who are receiving optimal medical therapy. **Class I**
2. Left ventricular ejection fraction < 35% and NYHA functional class II–III symptoms in patients with nonischemic heart disease who are receiving optimal medical therapy. **Class I**
3. Left ventricular ejection fraction < 30% and NYHA functional class I symptoms in patients with a myocardial infarction ≥ 40 days prior and no coronary revascularization in the last 3 months who are receiving optimal medical therapy. **Class I**
4. LVEF < 40%, prior MI, nonsustained VT, and inducible sustained VT or VF at electrophysiological study. **Class I**
5. Left ventricular ejection fraction ≤ 35% and NYHA functional class I symptoms in patients with nonischemic heart disease who are receiving optimal medical therapy. **Class IIb**
6. In combination with CRT in patients with left ventricular ejection fraction ≤ 35% and NYHA functional class III–IV symptoms who meet all other criteria for CRT. **Class IIb**

NYHA, New York Heart Association; CRT, cardiac resynchronization therapy.
Currently, the main determinant of candidacy for an ICD for the primary prevention of sudden cardiac death is the severity of left ventricular dysfunction.\(^1\) In general, patients with an ejection fraction \(\leq 35\%\) are likely to benefit from a prophylactic ICD. Although incompletely supported by randomized trials, recent guidelines promote the use of NYHA functional class to determine which patients with less severely depressed ventricular function (i.e., ejection fraction 35–40\%) may benefit from an ICD. Implantation has been recommended if NYHA functional class II or III is present in this setting.\(^2\) Current ICD indications for primary prevention are summarized in Table 3.12.\(^3\) In addition to the left ventricular ejection fraction, other important factors that should be considered when determining whether to place a prophylactic ICD are listed in Table 3.13. Of these, the timing of implantation relative to myocardial infarction and revascularization is discussed in greater detail below.

For patients with ischemic cardiomyopathy, trial data provide guidance regarding the timing of ICD implantation. Most ICD trials excluded patients with recent myocardial infarction (usually within 1 month). Despite the fact that the risk of sudden cardiac death is high immediately after acute myocardial infarction, the DINAMIT trial, which prospectively randomized patients to ICD or medical therapy 6–40 days after acute infarction, did not show a survival benefit with the ICD.\(^8\) As a result, routine, prophylactic ICD implantation should be delayed at least 40 days after an acute myocardial infarction. Additionally, as over one-half of patients with left ventricular dysfunction during acute myocardial infarction will experience an increase in systolic function within 3 months of the acute infarct, left ventricular ejection fraction should be reassessed after that time period to determine long-term risk and ICD candidacy.\(^9\)

Also excluded from most prophylactic ICD trials were patients with recent (usually within 3 months) coronary artery bypass surgery (CABG). The CABG-Patch trial randomized patients with left ventricular dysfunction undergoing coronary artery bypass graft surgery to ICD or medical therapy, and found that an ICD placed at the time of surgery did not improve survival.\(^8\) As a result of this study and the exclusion from the primary prevention ICD trials of patients who had CABG in the prior 3 months, implantation of a prophylactic ICD is deferred for 3 months after coronary artery bypass graft surgery or percutaneous coronary intervention. A subanalysis of the MADIT II trial showed no ICD survival benefit in patients who had coronary revascularization 3–6 months before implantation of the ICD, suggesting that the ICD can be further deferred up to 6 months after coronary revascularization.\(^8\) Taken together, these trials highlight the importance of revascularization and biological healing following a cardiac insult. Since left ventricular function may improve after revascularization,\(^9\) left ventricular ejection fraction and ICD candidacy should be reassessed after a 3–6-month time period. Importantly, these findings apply only to prophylactic ICD placement, and not to patients with significant clinical dysrhythmias.

The other common clinical scenario is management of the patient with reduced left ventricular function due to a remote infarction who is clinically stable and has no arrhythmic symptoms. Available evidence suggests these patients do benefit from a prophylactic ICD, and that the benefit of ICD implantation increases as the time from infarction increases (Fig. 3.18).\(^9\)

In patients with coronary artery disease, ejection fraction \(< 35\%\), QRS \(\geq 120\) ms and NYHA class III or IV heart failure, an ICD with cardiac resynchronization is indicated for the treatment of heart failure. This is discussed in detail above in the section on pacing in congestive heart failure.

### Dilated cardiomyopathy

Patients with left ventricular dysfunction that is not attributable to coronary artery disease (i.e., nonischemic cardiomyopathy) are also at increased risk of arrhythmic death and all-cause death.\(^31\)–\(^35\) The cause of sudden cardiac death in nonischemic cardiomyopathy is usually VT due to reentry around areas of myocardial fibrosis that result from the underlying pathophysiology of the cardiomyopathy. In contrast to ischemic heart disease, in which there are usually discrete areas of scar, fibrosis

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**Table 3.13** Patient characteristics to consider when assessing indications for a prophylactic implantable cardioverter-defibrillator

| 1 | Left ventricular ejection fraction |
| 2 | New York Heart Association functional class |
| 3 | Use of optimal medical therapy for heart failure |
| 4 | Timing relative to myocardial infarction or coronary revascularization |
| 5 | Comorbidities |
| 6 | Patient life expectancy |

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\(^1\) Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach

\(^2\) Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach

\(^3\) Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach

\(^4\) Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach

\(^5\) Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach

\(^6\) Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach

\(^7\) Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach

\(^8\) Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach

\(^9\) Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach

\(^10\) Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach

\(^11\) Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach

\(^12\) Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach

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\(^32\) Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach

\(^33\) Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach

\(^34\) Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach

\(^35\) Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach
in nonischemic cardiomyopathy may be diffusely scattered throughout the myocardium. The extent of this fibrosis, which is the substrate for ventricular arrhythmias, is demonstrable with recent advances in cardiac imaging and correlates with the risk of ventricular arrhythmias and sudden cardiac death.94,95

Invasive electrophysiology study is insensitive in predicting significant ventricular arrhythmias and sudden cardiac death in nonischemic cardiomyopathy.96–101 Other risk stratification tools have been studied in this population, including signal-averaged ECG, baroreflex sensitivity, heart rate variability and T wave alternans; however, none has been as robust a risk marker for sudden cardiac death as left ventricular ejection fraction.102

Several randomized controlled trials have assessed the role of the ICD for primary prevention of sudden cardiac death in patients with nonischemic cardiomyopathy.98–101 The first two trials, CAT and AMIOVIRT, did not show a survival benefit with the ICD, but their study samples were small.98,100 CAT randomized 104 patients with an ejection fraction ≤ 30% and within 9 months of their diagnosis of cardiomyopathy to receive either an ICD or no device, and survival was not different between the two groups after 4 years of follow-up.101 AMIOVIRT randomized 103 patients with an ejection fraction ≤ 35% and nonsustained ventricular arrhythmias to receive either an ICD or amiodarone, and survival was not different between the two groups after 3 years of follow-up.101 The first larger trial, DEFINITE, randomized 458 patients with nonischemic cardiomyopathy, an ejection fraction < 36%, a history of heart failure symptoms, and nonsustained ventricular arrhythmias to receive either an ICD or no device in the setting of optimal medical therapy.105 After an average of 29 months' follow-up, the risk of death was 35% less and the risk of sudden arrhythmic death was 80% less in the patients who had received an ICD. The most recent and definitive data come from SCD-HeFT, which randomized 2521 patients with an ejection fraction ≤ 35% and NYHA class II or III heart failure to either an ICD, amiodarone, or placebo (all in addition to conventional medical therapy).84 Compared with placebo, patients who received an ICD had a 23% relative reduction and a 7% absolute reduction in the risk of death after an average follow-up of 46 months. The overall results were similar regardless of the etiology of heart failure (48% of the study population had nonischemic cardiomyopathy).

Based on these data, current indications for a prophylactic ICD in patients with nonischemic cardiomyopathy are NYHA class II or III symptoms, an ejection fraction < 30–35%, and chronic medical therapy (Tables 3.12 and 3.13). Also, despite the relative lack of data for patients with NYHA class I symptoms, current guidelines provide a IIb indication for the ICD in these patients.

The ICD is also indicated for primary prevention of sudden cardiac death as part of a CRT-D system in selected subgroups of patients who meet all other criteria for conventional CRT (as discussed in the above section on Indications for permanent pacing: heart failure). Patients with NYHA class III heart failure with an ejection fraction ≤ 35%, sinus rhythm, and QRS interval > 120 ms, as well as some patients with NYHA class II heart failure meeting these criteria, benefit from CRT-D as opposed to CRT-P.2 In the COMPANION trial, 1520 patients with ischemic or nonischemic cardiomyopathy, NYHA functional class III or IV, and a QRS ≥ 120 ms were randomized to CRT-P, CRT-D, or no device therapy (all combined with optimal medical therapy).47 CRT-D reduced the risk of all-cause mortality by 36% (which was similar to the risk reduction with CRT-P and significantly greater than that with no device therapy). Essentially, the indications for each therapy (CRT and ICD) can be considered independently of one another for a given patient and then prescribed accordingly as either CRT-P, ICD or CRT-D. Lastly, while the ICD is generally contraindicated in

**Fig. 3.18** Relationship between the time after myocardial infarction and benefit from the implantable cardioverter-defibrillator for primary prevention of sudden cardiac death. (Modified from Wilber DJ, Zareba W, Hall WJ et al. Time dependence of mortality risk and defibrillator benefit after myocardial infarction. Circulation 2004; 109:1082-4 with permission from the American Heart Association.)
NYHA class IV heart failure (due to the limited prognosis in these patients), it has a proven survival benefit and is indicated as part of a CRT-D system when CRT is otherwise indicated in patients with NYHA class IV symptoms.98

For patients diagnosed with nonischemic cardiomyopathy, the correct time to implant an ICD is debatable. The COMPANION trial excluded patients within 9 months of the diagnosis of cardiomyopathy, and the SCD-HeFT and DEFINITE trials did not specify a specific time interval, but excluded patients if they had a reversible cause of cardiomyopathy.62,64,103 The uncertainty regarding the appropriate timing of ICD implantation centers on our limited ability to identify the causes of nonischemic cardiomyopathy and to predict the occurrence and magnitude of improvements in cardiac function after diagnosis. Currently, Medicare and Medicaid reimburse ICD implantations performed 9 months after the diagnosis of nonischemic cardiomyopathy. They also reimburse ICD implants performed 3–9 months after the diagnosis of nonischemic cardiomyopathy. While current practice is to implant the ICD 3 months after diagnosis of non-ischemic cardiomyopathy, the correct time to implant an ICD is debatable. The COMPANION trial excluded patients within 9 months of the diagnosis of cardiomyopathy, and the SCD-HeFT and DEFINITE trials did not specify a specific time interval, but excluded patients if they had a reversible cause of cardiomyopathy.62,64,103

The uncertainty regarding the appropriate timing of ICD implantation centers on our limited ability to identify the causes of nonischemic cardiomyopathy and to predict the occurrence and magnitude of improvements in cardiac function after diagnosis. Currently, Medicare and Medicaid reimburse ICD implantations performed 9 months after the diagnosis of nonischemic cardiomyopathy. They also reimburse ICD implants performed 3–9 months after the diagnosis of nonischemic cardiomyopathy for patients enrolled in a national registry, which was created to clarify the benefits of the ICD during this early time period.106 A recent analysis from this registry at one institution107 and a post-hoc subgroup analysis of the DEFINITE trial105 have shown that the ICD was beneficial immediately after the diagnosis of nonischemic cardiomyopathy when a reversible cause was excluded. Since unrecognized persistent tachycardia may result in reduced ventricular systolic function, it is important to recognize the potential impact of both atrial fibrillation and frequent premature ventricular contractions on the etiology of cardiomyopathy. Recovery from tachycardia-induced cardiomyopathy may be evident months after adequate control of ventricular rates is achieved.55,108 While current practice is to implant the ICD 3 months after diagnosis of non-ischemic cardiomyopathy, the optimal patient selection for earlier ICD implantation will be honed by future research regarding clinical and laboratory-based predictors of irreversible ventricular dysfunction.109

**Long QT syndrome**

Long QT syndrome (LQTS) comprises a group of uncommon inherited disorders of cardiac ion channels, which result in abnormal repolarization and usually manifest on the surface electrocardiogram with a long corrected QT interval and abnormal T-wave morphology (Fig. 3.19 and Table 3.14).110 Patients with LQTS have a propensity for syncope, seizures, and/or sudden cardiac death secondary to polymorphic VT (torsades de pointes).111 For the highest risk subset, the untreated mortality is approximately 5–10%/year.111 Since this is primarily an electrical disorder typically seen in patients without significant cardiac structural or functional abnormalities, arrhythmia control results in an excellent prognosis. β-Blockers are first-line therapy, and they improve symptoms and reduce significantly (but do not eliminate) the risk of death, especially for patients with type 1 LQTS (LQT1). Unfortunately, about 30% of patients remain symptomatic despite high-dose β-blocker therapy, and 9% of sudden deaths among patients with LQTS occur in those taking β-blockers.110 Both antiarrhythmic pacing (to prevent proarrhythmic pauses and to shorten depolarization) and left cardiac sympathetic denervation also reduce the risk of sudden death in these patients.111

ICD therapy is recommended for patients with LQTS and previous aborted sudden death (class I indication) and those with sustained ventricular arrhythmias or recurrent syncope despite β-blocker therapy (class IIa indications).2 Observational data from a series of patients with LQTS and aborted sudden death or recurrent syncope demonstrated 1.3% mortality during an average 3 years’ follow-up in the 73 patients with an ICD compared with 16% mortality during an average 8 years’ follow-up in the 161 patients without an ICD.112 At present, it is unclear whether a positive family history provides relevant information regarding an individual’s risk for sudden death and the potential benefit from ICD implantation. Patients with LQTS and a strong family history of sudden death are often offered an ICD (class IIb indication), particularly if other risk factors are present (deafness, syncope, female sex, or corrected QT interval > 500 ms). However, it is possible that family history provides an emotional impetus for more aggressive therapy rather than an evidence-based one. In persons with a strong family history and a normal corrected QT interval, familial genetic evaluation can be helpful. Family members found to have a putative mutation but who do not have symptoms or a prolonged corrected QT interval are generally observed or treated with β-blockers, whereas those with significant risk factors listed above are usually offered an ICD.

Some unique considerations exist for patients based on the specific genetic defects identified. The SCN5A cardiac ion channel mutation (in LQT3) is associated
Fig. 3.19 Long QT syndrome. (A) Electrocardiogram shows a QTc of 575 ms, suggesting increased risk of an arrhythmic event. This patient had a confirmed mutation affecting the cardiac potassium channel KVLQT1. (B) In a rhythm strip from a different patient, torsades de pointes is initiated during a dobutamine challenge. A short interval is terminated by a premature ventricular contraction (PVC), which in turn is followed by a long interval. The short-long sequence results in prolongation of repolarization, and a second PVC initiates the dysrhythmia.
with poor responsiveness to β-blockers and a higher risk of sudden death, so implantation of an ICD in patients who have this mutation may be of greater benefit. Also, ICD therapy should be strongly considered in patients who are noncompliant or cannot tolerate β-blocker therapy, since β-blocker noncompliance can be fatal. Indications for the ICD in patients with LQTS are not standardized, and must be individualized since the understanding of its natural history, genetic basis and response to treatments is still emerging. Table 3.15 lists significant risk factors for sudden cardiac death in patients with LQTS. Genetic analysis may be useful for risk stratification, and genetic testing is now commercially available. The unique issues to programming ICDs in patients with LQTS are discussed in Chapter 8 (programming).

Brugada syndrome and sudden unexplained death syndrome
Individuals with the Brugada syndrome, now recog-
nized also to include the sudden unexplained death syndrome (SUDS) that predominantly affects young Southeast Asian men, have an increased risk of sudden cardiac death despite a structurally normal heart. It is an inherited condition with autosomal dominant transmission and increased manifestation in men. Various mutations in the gene encoding the α subunit of the cardiac sodium channel SCN5A have been identified, but < 20% of familial cases of Brugada syndrome are associated with recognized mutations of this gene. Diagnosis depends on the demonstration of an electrocardiogram with incomplete RBBB and coved ST-segment elevation in leads V1–V3 (which sometimes is evident only after administration of a sodium-channel blocker, such as procainamide or ajmaline) (Fig. 3.20). Other precipitants of the typical electrocardiographic pattern are vagotonic agents, α-adrenergic receptor agonists, β-adrenergic receptor blockers, tricyclic or tetracyclic antidepressants, alcohol, cocaine, hyperthermia, or hypokalemia.

The risk of sudden cardiac death in the Brugada syndrome has been found to be between 0.35%/year to 4%/year, depending on the population being studied. A meta-analysis of over 30 prospective studies estimated that sudden death, syncope, or ICD therapy occurred in 10% of patients during an average 32 months’ follow-up. Pharmacological therapy to prevent sudden death is relatively ineffective. Patients at highest risk of sudden death for whom the ICD is recommended have the typical electrocardiogram findings and either prior cardiac arrest (class I indication) or prior syncope or VT (class IIa indications). A randomized trial of ICD therapy has not been performed in the broader Brugada syndrome population, but one randomized controlled trial in 86 Southeast Asian patients with SUDS with prior confirmed or suspected cardiac arrest showed a clear benefit of the ICD for preventing recurrent sudden cardiac death in this specific subgroup. Patients at an intermediate risk of sudden death have the typical electrocardiogram findings, but no history of syncope. Previously, the decision to implant an ICD was aided by electrophysiology study; however, this strategy is not well supported by the available data and the ongoing PRELUDE study may clarify this issue in the future. Finally, those at lowest risk of sudden death are asymptomatic individuals who either have the typical electrocardiogram findings only with pharmacological challenge or are known from screening to be silent carriers of the gene mutation.

Other channelopathies

The short QT syndrome is a condition characterized by a structurally normal heart with a short QT interval.

### Table 3.16 Risk factors for sudden cardiac death in the Brugada and sudden unexplained death syndromes

<table>
<thead>
<tr>
<th>Risk Factor</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Prior cardiac arrest</td>
</tr>
<tr>
<td>2. Male</td>
</tr>
<tr>
<td>3. Spontaneous electrocardiographic Brugada pattern</td>
</tr>
<tr>
<td>4. Prior syncope and a spontaneous electrocardiographic Brugada pattern</td>
</tr>
<tr>
<td>5. Inducible ventricular tachycardia or fibrillation (controversial)</td>
</tr>
<tr>
<td>6. Fever</td>
</tr>
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</table>
(<300–320 ms) and narrow, peaked T waves. Published case reports in very few individuals with these findings have suggested that it is associated with a markedly increased risk of sudden cardiac death. To date, mutations in three genes encoding different cardiac potassium channels that result in abnormally short ventricular repolarization have been identified. However, selection bias and the lack of a controlled study limit the inferences regarding risk that can be made from these series of cases. Recent controlled studies in large populations have shown that a short QT interval is exceedingly rare (no QT interval ≤300 ms was identified among 118,444 people in these studies) and that individuals with QT intervals in the shortest half centile of the normal distribution did not experience increased mortality after an average of 8 years’ follow-up. The duration of the QT interval that might be associated with an increased risk of sudden death, other clinical characteristics associated with an increased risk of sudden death, the role of electrophysiology study, pharmacological management, and the role of the ICD in these patients are currently unknown.

Catecholaminergic polymorphic ventricular tachycardia (CPVT) is a condition characterized by sustained ventricular arrhythmias that occur during acute emotional or physical stress. These arrhythmias can include sustained monomorphic VT, bidirectional VT (i.e., alternating QRS axis), polymorphic VT, and VF. It is an inherited condition with autosomal dominant and autosomal recessive modes of transmission, and it usually manifests during childhood. Associated mutations have been identified in the genes encoding the cardiac ryanodine receptor and calsequestrin, both responsible for intracellular handling of calcium. Implantation of an ICD is generally indicated for all patients with CPVT who have had a prior cardiac arrest (class I) or syncope or sustained VT while receiving β-blocker therapy (class IIa). Currently, other clinical characteristics and the results of genetic testing do not add to risk stratification and the decision to implant an ICD.

Arrhythmogenic right ventricular dysplasia

Arrhythmogenic right ventricular dysplasia (ARVD) is characterized by fibrous and fatty replacement of right ventricular myocardium, although both ventricles can be involved. Over half of recognized patients have a family history, and mutations in genes encoding specific cell adhesion proteins have been identified. Patients present with asymptomatic premature ventricular contractions, palpitations, syncope due to fast monomorphic VT, or sudden death. Up to 10% of unexplained sudden deaths in young patients are thought to be due to ARVD in some geographic regions. The electrocardiographic manifestations of ARVD include inverted T waves in leads V1–V3, RBBB, and epsilon waves (Fig. 3.21). The premature ventricular contractions and VT usually have a left bundle branch block pattern due to their origin in the right ventricle (Fig. 3.22). The diagnosis is sometimes challenging and requires cardiac magnetic resonance imaging or computed tomography to identify the presence and extent of fibrous or fatty tissue within the ventricular myocardium. Endomyocardial biopsy is insensitive due to the apical location and patchy nature of the involved tissue, but it may be performed to distinguish arrhythmogenic right ventricular dysplasia from other cardiomyopathies. The diagnostic criteria for ARVD are listed in Table 3.17. (Reproduced with permission from Oxford University Press.)

Indications for the ICD in patients with ARVD are based on emerging data regarding the risk factors for sudden death. Aborted sudden death defines an individual as high risk with a clear secondary prevention indication. In patients without prior cardiac arrest, a history of syncope or sustained VT or VF are class I indications for the ICD. Other factors associated with

Fig. 3.21 Arrhythmogenic right ventricular dysplasia. Electrocardiogram shows right precordial T wave inversions and epsilon waves (arrows).
death or ICD firings include severe right ventricular dilation, inducible VT during electrophysiology study, early onset of symptoms, male gender, increased QT dispersion, and left ventricular involvement.\textsuperscript{127,128,130} Class IIa indications for the ICD endorsed by the 2006 ACC/AHA/ESC guidelines include “extensive disease,”

Table 3.17 Diagnostic criteria for arrhythmogenic right ventricular dysplasia (ARVD)

<table>
<thead>
<tr>
<th>Major</th>
<th>Minor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Global or regional dysfunction and structural alterations (imaging techniques)</td>
<td>Severe right ventricular dilation</td>
</tr>
<tr>
<td></td>
<td>Decrease in RVEF with normal/nearly normal LV</td>
</tr>
<tr>
<td></td>
<td>RV localized aneurysms (akinetic or dyskinetic areas with systolic bulging)</td>
</tr>
<tr>
<td></td>
<td>Severe RV segmental dilation</td>
</tr>
</tbody>
</table>

Tissue characterization of the RV myocardium

<table>
<thead>
<tr>
<th>Repolarization abnormalities</th>
<th>Fatty infiltration of the RV myocardium</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Inverted T waves in V\textsubscript{2}–V\textsubscript{3} (if age &gt; 12 years, in the absence of RBBB)</td>
</tr>
</tbody>
</table>

Depolarization or conduction abnormalities

<table>
<thead>
<tr>
<th>Arrhythmias</th>
<th>Late potentials</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epsilon waves, widening of the QRS complex (&gt; 110 ms) in V\textsubscript{1}–V\textsubscript{3}</td>
<td></td>
</tr>
</tbody>
</table>

Diagnosis of ARVD requires two major criteria, one major and two minor criteria, or four minor criteria.\textsuperscript{128} Reproduced with permission from Oxford University Press.

ARVD, arrhythmogenic right ventricular dysplasia; EF, ejection fraction; LBBB, left bundle branch block; LV, left ventricle; RBBB, right bundle branch block; RV, right ventricle.
involvement of the left ventricle, family members with ARVD and sudden death, and undiagnosed syncope when VT and VF have not been excluded as its cause.1 The frequency of ventricular arrhythmias in patients with arrhythmogenic right ventricular dysplasia may be high, necessitating other therapies to prevent excessive shocks while maintaining ICD therapy as a lifesaving back-up measure.13 In these cases, alternative or adjuvant treatment approaches include medications, catheter ablation, and surgical ventriculotomy or disarticulation of the involved myocardium.

**Hypertrophic cardiomyopathy**

HCM is a heterogeneous disease, associated with various mutations of the genes encoding myocardial contractile proteins, as well as variable cardiac structural abnormalities and clinical presentations.132 The natural history of HCM is characterized by slow progression of symptoms such as dyspnea and angina, but also by sudden cardiac death. The characteristic myocardial disarray and interstitial fibrosis provide the substrate for re-entrant ventricular arrhythmias, which are probably the cause of sudden death in most patients and may be triggered or modulated by autonomic dysfunction, subendocardial ischemia, or conduction abnormalities.132 The annual risk of sudden death is 5% in patients without prior cardiac arrest and 11% for patients with a history of resuscitated arrest.133 About 50% of sudden deaths in young athletes are due to HCM, and about 50% of deaths in patients with HCM occur suddenly.134–136

The 2006 ACC/AHA/ESC guidelines assign a class IIa indication for an ICD for the primary prevention of sudden cardiac death in patients with HCM who have one or more major risk factors (Table 3.18).2,135 The ICD is undoubtedly a necessary and proven therapy in these patients,133 but identifying the patients most likely to benefit is challenging. About 50% of patients with HCM who have sudden death do not have any of the recognized major risk factors.136 Despite these challenges, there are several areas of broad consensus. Patients who survived cardiac arrest or who have spontaneous sustained ventricular tachyarrhythmias clearly should receive an ICD (class I indication). Strong consideration should be given to ICD implantation in patients with multiple risk factors, particularly the following: young age (<35 years old); sudden death related to HCM in multiple family members; unexplained syncope, especially if recurrent or occurring during exertion; a hypotensive response to exercise, especially in patients <50 years old; a left ventricular wall thickness ≥30 mm, especially in younger patients; significant left ventricular outflow tract obstruction; and a high-risk mutation.132 The presence of even one risk factor, when considered significant for that individual (such as a malignant family history), is sufficient to warrant ICD implantation.2,132–135 Because of low implant morbidity and the uncertain effectiveness of antiarrhythmic drugs for this disease, we often offer ICD therapy to these patients with a highly individualized approach. Each patient’s preferences must be understood regarding the commitment to lifelong device implantation, the long-term need for system revisions, and the possibility of inappropriate shocks and associated anxiety.

Therapies such as β-blockers, alcohol septal ablation, and septal myectomy are successful in treating the symptoms of HCM related to left ventricular outflow tract obstruction and heart failure, but of these, only septal myectomy may provide survival benefit and reduce the rate of sudden death.137 However, septal myectomy is performed in only about 5% of patients with HCM, and it does not eliminate the risk of sudden cardiac death.137 Further risk stratification is required in patients who have undergone septal myectomy, and ICD therapy should continue to be offered to these patients based on their risk factor profile.

**Congenital heart disease**

Success in palliating many congenital heart defects during infancy and childhood has resulted in a growing population of adult patients with postsurgical congenital heart disease. These patients often have altered hemodynamics, abnormal cardiac chamber sizes, and reduced ventricular function. Specific conditions that have been associated with an increased risk of sudden cardiac death in adulthood are the tetralogy of Fallot,
transposition of the great arteries, and the univentricular heart. Most available data regarding risk stratification have been derived in patients with corrected tetralogy of Fallot due to its higher prevalence. Characteristics associated with sudden cardiac death are older age at surgery, ventricular dysfunction, wide QRS (> 180 ms) and hemodynamic abnormality due to pulmonary regurgitation.140 The role of invasive electrophysiology study has been controversial.139,140 A recent large multicenter study that included more than 250 tetralogy of Fallot patients found that inducible monomorphic or polymorphic VT predicted ventricular arrhythmias and sudden cardiac death.141 In tetralogy of Fallot patients with clinical symptoms or documented arrhythmias, the negative predictive value of electrophysiology study was 86% and the positive predictive value was 67%. These data suggest that invasive electrophysiology study, while imperfect, has a role in conjunction with other clinical characteristics for risk stratification and decision-making regarding ICD therapy. In general, these strategies, which have been demonstrated in patients with tetralogy of Fallot, are often extrapolated to patients with other congenital heart defects due to the paucity of data in these other conditions.

**Class I indications for ICD therapy in patients with congenital heart disease** include resuscitated cardiac arrest in patients for whom a reversible etiology of the cardiac arrest is excluded and who are receiving optimal medical therapy, or the occurrence of sustained VT that cannot be managed by catheter ablation or cardiac surgery.2 Class IIa indications for ICD therapy include unexplained syncope in patients with reduced ventricular systolic function after electrophysiology and hemodynamic studies. As discussed above, ICD therapy should be considered in patients at high risk of sudden cardiac death based on clinical characteristics and the results of electrophysiology study.

**Contraindications to implantable cardioverter-defibrillator therapy**

Contraindications to ICD therapy are summarized in Table 3.19. Patients who have incessant arrhythmias that cannot be controlled by adjuvant medical, catheter-based or surgical therapy are not candidates for the ICD. Patients whose arrhythmias are clearly due to a transient or reversible disorder, such as acute myocardial infarction, significant electrolyte imbalance, drug ingestion, or trauma, do not have a greatly increased risk of recurrent arrhythmia and should not receive an ICD. Patients with significant psychiatric illnesses that may be aggravated by device shocks and patients with terminal illness and unacceptable functional status should not receive an ICD. This includes patients with NYHA class IV drug-refractory congestive heart failure who are not candidates for transplantation (except when ICD therapy is offered as part of an appropriately indicated CRT device). Also, in general, patients should not undergo ICD therapy for arrhythmias amenable to surgical or catheter ablation, such as right ventricular outflow tract VT, idiopathic left ventricular VT, fascicular VT, or Wolff–Parkinson–White syndrome with ventricular tachyarrhythmias secondary to rapid anterograde conduction of atrial fibrillation (Figs 3.23 and 3.24).

<table>
<thead>
<tr>
<th>Contraindications to the implantable cardioverter-defibrillator</th>
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<tbody>
<tr>
<td>1 Syncope of undetermined cause in a patient without inducible ventricular tachyarrhythmias and without structural heart disease</td>
</tr>
<tr>
<td>2 Incessant ventricular tachycardia (VT) or ventricular fibrillation</td>
</tr>
<tr>
<td>3 Ventricular fibrillation or VT resulting from arrhythmias amenable to surgical or catheter ablation; for example, atrial arrhythmias associated with the Wolff–Parkinson–White syndrome, right ventricular outflow tract VT, idiopathic left VT, or fascicular VT</td>
</tr>
<tr>
<td>4 Ventricular tachyarrhythmias due to a transient or reversible disorder (e.g., acute myocardial infarction, electrolyte imbalance, drugs, trauma)</td>
</tr>
<tr>
<td>5 Significant psychiatric illnesses that may be aggravated by device implantation or may preclude systematic follow-up</td>
</tr>
<tr>
<td>6 Expected survival with acceptable functional status &lt; 1 year</td>
</tr>
<tr>
<td>7 Patients with coronary artery disease who have left ventricular dysfunction and prolonged QRS duration without spontaneous or inducible sustained or nonsustained VT and who are undergoing coronary bypass surgery</td>
</tr>
<tr>
<td>8 New York Heart Association class IV drug-refractory congestive heart failure in patients who are not candidates for biventricular pacing or cardiac transplantation</td>
</tr>
</tbody>
</table>
Acknowledgement

The authors thank Dr Michael Ackerman for his review of the LQTS section of this chapter.

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CHAPTER 3 Indications for Pacemakers, ICDs and CRT


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CHAPTER 3  Indications for Pacemakers, ICDs and CRT


CHAPTER 4

Generator and Lead Selection

Samuel J. Asirvatham, David L. Hayes, Paul A. Friedman

The purpose of this chapter is to provide direction in choosing the most appropriate pulse generator—pacemaker, internal cardioverter-defibrillator (ICD) or cardiac resynchronization therapy (CRT)—and leads for a given patient. It is not possible to provide guidelines that meet the needs of every patient; indeed, pulse generator selection, whether pacemaker, ICD or CRT, must be individualized. Our goal is to provide practical considerations and a generic approach to determine the type of hardware most appropriate for the patient that is receiving an implantable cardiac device.

Pacemaker selection

Pacemaker selection today involves a decision as to whether cardiac resynchronization is required. A significant proportion of patients receiving devices have combined abnormalities that may include chronotropic incompetence, atrioventricular (AV) nodal dysfunction, intraventricular conduction delay (electrical ventricular dyssynchrony) and the propensity for ventricular arrhythmia. Thus, decisions for patients requiring pacemakers may involve consideration and exclusion of the need for a defibrillator lead or a left ventricular (LV) pacing lead. The data supporting the hemodynamic benefits of cardiac resynchronization and the present indications are discussed in Chapters 2 and 3. In this chapter, after a brief summary of the generally used algorithm for deciding which pulse generator, mode selection and leads are to be used, we point out specific issues relevant to cardiac resynchronization devices.

Many algorithms have been used for pacemaker and mode selection (Figs 4.1 and 4.2). Although all rules have exceptions, a very simple approach is appropriate for most patients. For patients with chronic atrial fibrillation and a slow ventricular response in whom pacing is required, VVIR is the mode of choice. This is the only clear-cut indication for single-chamber ventricular pacing. Obviously, other patients may have associated comorbidities or other issues that would favor use of a single-chamber ventricular pacemaker, either VVI or VVIR. For example, in a patient with other irreversible medical problems in whom longevity or activity level (or both) is markedly limited, a simple device may be the most appropriate. These decisions must be made individually. However, even in the most ill patient in whom a pacemaker is being implanted, one must be certain that a simpler device, i.e., VVI, does not make the patient worse by causing adverse hemodynamic symptoms, e.g., pacemaker syndrome.

Even for a patient with another irreversible and possibly progressive medical illness and marked limitations in activity in whom a decision is made to implant a pacemaker, if VVI pacing results in pacemaker syndrome, the patient may actually feel worse. Although such an outcome may be impossible to predict, it should at least be considered and, at a minimum, blood pressures should be compared in the native underlying rhythm vs. ventricular pacing.

At one time there were significant cost differences between simpler devices (VVI) vs. those that had rate-adaptive pacing, etc. Today, these differences are largely negligible in most parts of the world, and the main decision that needs to be made is whether a single-chamber device will suffice (chronic atrial fibrillation) or not. Internationally, however, the simplest device (single-chamber nonrate adaptive) may be necessary to save lives amidst more significant economic constraints.
In most practices, however, devices retaining options for more complex pacing requirements are placed and programmed “on” when required (Table 4.1).

**Symptomatic bradycardia**

For most patients with bradyarrhythmias requiring pacing who do not have chronic atrial fibrillation, a dual-chamber pacemaker is indicated. Using a dual-chamber pacemaker with rate-adaptive capabilities (DDDR) in all patients provides the greatest long-term flexibility, and most patients with symptomatic bradycardia often have associated chronotropic incompetence or at least the potential for this to occur. In some situations, however, rate-adaptive pacing is essential, including dual-chamber devices placed specifically for patients with sick sinus syndrome and fatigue from documented chronotropic incompetence and patients with chronic atrial fibrillation with slow ventricular rates. Because rate responsiveness in patients with chronic atrial fibrillation depends on autonomic regulation of the AV node (poorly regulated), these patients necessarily require sensor-driven pacing. For example, if chronic atrial fibrillation with a slow ventricular response develops at a later date, the DDDR pacemaker could be reprogrammed to VVIR. A DDD pacemaker, which has no rate-adaptive capability, could be programmed only to VVI, potentially a suboptimal option.

**Pure sinus node dysfunction**

For the patient with pure sinus node disease, i.e., no documented or provicable abnormalities in AV nodal conduction, AAIR pacing may be appropriate. (Preimplantation criteria for AAI/AAIR pacing are described in Chapter 3.) If preimplantation testing has established normal AV nodal conduction, the annual risk of AV block developing is 1–2%. If AV block develops, another procedure is required to implant a ventricular lead and upgrade the pacemaker. This additional procedure is obviated if a DDDR pacemaker is used initially. Although DDDR pacing gives the most flexibility of options, some disadvantages of placing a ventricular lead and pacing the right ventricle require consideration. The adverse effects of right ventricular pacing are discussed in Chapter 2, Hemodynamics, and ventricular avoidance pacing algorithms are discussed in Chapter 7, Timing Cycles and Chapter 8, Programming.
CHAPTER 4  Generator and Lead Selection  123

Table 4.1  Indications for various pacing modes

<table>
<thead>
<tr>
<th>Mode</th>
<th>Generally agreed-upon indications</th>
<th>Controversial indications</th>
<th>Contraindications</th>
</tr>
</thead>
<tbody>
<tr>
<td>VVI</td>
<td>Symptomatic bradycardia in the patient with associated terminal illness or other medical conditions from which recovery is not anticipated and pacing is life-sustaining only</td>
<td>Patients with known PM syndrome or hemodynamic deterioration with ventricular pacing at the time of implantation</td>
<td>CI patient who will benefit from rate response</td>
</tr>
<tr>
<td>VVIR</td>
<td>Fixed atrial arrhythmias (AF or atrial flutter) with symptomatic bradycardia</td>
<td>Same as for VVI</td>
<td>Patients with known PM syndrome or hemodynamic deterioration with ventricular pacing at the time of implantation</td>
</tr>
<tr>
<td>AAI</td>
<td>Symptomatic bradycardia as a result of SND in the otherwise CC patient when AV conduction can be proven normal</td>
<td>SND with associated AV block demonstrated either spontaneously or during preimplantation testing</td>
<td>In the unlikely event that atrial sensing is inadequate</td>
</tr>
<tr>
<td>AAIR</td>
<td>Symptomatic bradycardia as a result of SND in the CI patient when AV conduction can be proven normal</td>
<td>Same as for AAI</td>
<td></td>
</tr>
<tr>
<td>VDD</td>
<td>Congenital AV block AV block when sinus node function can be proven normal</td>
<td>AV block when sinus node function can be proven normal</td>
<td>SND AV block accompanied by SND When adequate atrial sensing cannot be attained</td>
</tr>
<tr>
<td>VDDR</td>
<td>Same as for VDD, but when a potential need for ventricular rate-adaptive pacing also exists</td>
<td>For any rhythm disturbance when atrial sensing and capture are possible, with the exception of AF or atrial flutter, potentially to minimize future AF, reduce morbidity, and improve survival For the suppression of tachyarhythmias by overdrive suppression</td>
<td>Chronic AF, atrial flutter, giant inexcitable atrium, or other frequent paroxysmal supraventricular tachyarrhythmias When adequate atrial sensing cannot be attained</td>
</tr>
<tr>
<td>DDD*</td>
<td>AV block and SND in the CC patient Need for AV synchrony, e.g., to maximize cardiac output, inactive patients Previous PM syndrome with VVI(R) pacemaker</td>
<td>For any rhythm disturbance when atrial sensing and capture are possible, with the exception of AF or atrial flutter, potentially to minimize future AF, reduce morbidity, and improve survival For the suppression of tachyarhythmias by overdrive suppression</td>
<td>Chronic AF, atrial flutter, giant inexcitable atrium, or other frequent paroxysmal supraventricular tachyarrhythmias When adequate atrial sensing cannot be attained</td>
</tr>
<tr>
<td>DDDR</td>
<td>AV block and SND in the CI patient</td>
<td>Same as for DDD</td>
<td>Same as for DDD</td>
</tr>
</tbody>
</table>

AF, atrial fibrillation; AV, atrioventricular; CC, chronotropically competent; CI, chronotropically incompetent; PM, pacemaker; SND, sinus node dysfunction.

*DDI and DDIR are not included as separate modes in this table because they are not commonly used as the preimplantation modes of choice.

**Pure atrioventricular block**

For the patient with pure AV node disease, i.e., no documented abnormalities in sinus node behavior, VDD or VDDR pacing may be appropriate. Specifically, the patient with congenital complete heart block may do well with VDD(R) pacing. As before, a problem arises if sinus node dysfunction develops in the future, resulting in a suboptimal pacing mode without
reasonable programming options. However, from a “hardware” perspective, use of a single-lead VDD or VDDR pacing system helps to minimize the amount of hardware needed. This can be especially important for pediatric patients.

Single-pass VDD(R) systems are occasionally favored in pediatric patients, but otherwise this pacing mode is not commonly used. Single-pass VDD leads, i.e., atrial sensing occurs via “floating” electrodes in the atrialized portion of the lead, have generally been passive fixation leads, and for pediatric patients many implanters, including our institution, would prefer active-fixation leads.

It should be mentioned that single-pass leads have also been used for DDD pacing, i.e., the floating atrial electrodes are capable of pacing and sensing.7 This approach to DDD pacing has never been widely embraced. Concerns have been the requirement for high atrial outputs in order to achieve capture and phrenic nerve stimulation. Placing a single-pass DDD lead in the coronary sinus has also been attempted for left atrial and left ventricular stimulation.8

Neurocardiogenic syncope and carotid sinus hypersensitivity
If a patient requires pacing for neurocardiogenic syncope, whether the vasovagal variety or carotid sinus hypersensitivity, dual-chamber pacing is necessary for several reasons.9–11 In a patient with vasovagal syncope that most likely has some component of vasodepression together with the cardioinhibition that requires pacing, ventricular pacing alone could result in pacemaker syndrome and further aggravate symptoms caused by hypotension. The bradycardia that occurs in patients with carotid sinus hypersensitivity may be due to either AV block or sinus arrest. Therefore, ventricular pacing support is required, and dual-chamber pacing is superior for the reasons already noted. For specific programmable options that are desirable when pacing patients with these disorders see Chapter 8, Programming and Chapter 7, Timing Cycles.

Choosing specific programmable options
Programming and the wide variety of programmable options available in current devices are covered in Chapter 8, Programming.

Choosing the rate-adaptive sensor
When choosing hardware for a specific patient, there may be advantages of one rate-adaptive sensor over another. This is discussed in Chapter 9, Rate-Adaptive Pacing.

Choosing the lead or leads
A detailed discussion of the merits of various lead types is beyond the scope of this chapter, as is a thorough discussion of the evolution of pacing leads. Rather, the purpose of the chapter is to provide the reader with an understanding of the types of leads that are available and future trends that are likely to be seen.

With the exception of a few specific circumstances, choice of the pacing lead or leads becomes one of personal preference and personal bias. Choice of pacing leads should also be based on performance data of the specific model. Options that must be considered for all leads are:

- Type of insulation: silicone or polyurethane or a “hybrid” combination (such as in a bipolar coaxial lead where the inner and outer insulations are different materials)
- Mechanism of fixation: active or passive
- Polarity: unipolar or bipolar
- Compatibility of lead and pulse generator connection system.

Choice of an atrial pacing lead must take into account insulation, fixation, polarity, and whether the lead is straight or preformed J type (Fig. 4.3). The interelectrode distance of a bipolar lead also deserves consideration. This distance may not only affect the duration of the intracardiac electrogram signals, but it also usually has an effect on the amplitude and slew rate of the electrogram’s signals, depending on the low- and high-frequency band pass filters in the input amplifiers of the device. A lead with a greater distance between electrodes will generally have larger amplitude far-field signals (i.e., R-waves sensed on an atrial lead). This characteristic may affect the reliability of mode switching. In pacing systems with more than one ventricular sensing lead, i.e., pacemaker and ICD in the same patient or ICD with biventricular pacing, minimizing far-field signals may also be of increased importance.

The same decisions must be made for choice of the ventricular lead, except that right ventricular leads are straight (Fig. 4.4). Specific issues related to LV pacing are discussed below.
Although multiple mechanisms have been used to achieve lower thresholds, steroid elution has been the most successful and most widely used method for threshold reduction.\textsuperscript{12,13} Steroid elution has been accomplished in several ways, such as from a steroid-saturated silicone plug within the lead’s tip electrode or from a steroid-eluting collar adjacent to the tip electrode (Fig. 4.5). Steroid elution significantly minimizes the post-implant pacing threshold increases and peaking that typically occurs with nonsteroid-eluting electrodes. The steroid-eluting plug or collar contains a very small amount of steroid, e.g., < 1 mg of dexamethasone sodium phosphate or dexamethasone acetate, which do not have any systemic effect. Steroid elution is available on atrial and ventricular leads with both active and passive fixation means, and is also available in coronary venous and epicardial leads. In nearly all institutions, steroid-eluting leads are used routinely.

Fractal coating (Fig. 4.6) and other techniques have also been used to lower pacing thresholds.

\textbf{Insulation}

With few exceptions, the materials that have been used for most pacing leads for almost five decades are silicone rubber and polyurethane. Historically, both have generally had excellent performance records. Table 4.2 compares the basic characteristics of silicone rubber and polyurethane. When first introduced, polyurethane insulated leads were widely used by many implanters because they could be designed to have a smaller lead body diameter than many of the contemporary silicone rubber insulated leads and were said to handle better when two leads were implanted due to their greater lubricity in the body. Improvements in silicone insulation and use of lubricious coatings have made these differences less significant.

Manufacturers often make the same lead available with either silicone or polyurethane outer insulation. Implanters base their choice on past experience, ideally taking into account product surveillance reports.
Steroid-lead

that detail the survival of specific leads. Not all agree on what is advantageous or disadvantageous about the characteristics of the insulating material.

For silicone rubber, being "very flexible" may or may not be an advantage. This quality means that something else in the design, such as the conductor coil, must increase the stiffness of the lead to the optimal value. A lead that is too flexible can damage the heart with excessive movement and may cause lead dislodgment or deterioration of the lead’s electrical parameters. A lead that is too stiff may cause perforation or dislodgment. A stiff conductor coil can be more prone to fracture.

Some presently available bipolar coaxial leads utilize both silicone (inner insulation) and polyurethane (outer insulation) to benefit maximally from both their properties. A new insulation material, created specifically for cardiac leads, is available and used in various types of leads, which is a silicone rubber–polyurethane copolymer (Optim®; St Jude Medical, Inc.).

"Smaller diameter possible" is not included in Table 4.2 because it is neither an advantage nor a disadvantage. It is the result of the polymer’s mechanical characteristics.

Fig. 4.5 Diagrammatic representation of a steroid-eluting lead. Steroid (dexamethasone) is slowly eluted through the porous, platinized tip of a silicone rubber plug.

Fig. 4.6 Magnified images of a fractally coated electrode, (A) at 2 µm; (B) at 20 µm. The fractal surface of the lead electrodes creates a larger effective surface area, and as a result maximizes the myocardial interface, which is a major factor in determining a lead’s sensing characteristics.
properties such as high tear strength and higher stiffness. That is, even if silicone rubber had the same tear strength as polyurethane, smaller diameter insulation could make a lead like a whipsaw. Higher stiffness allows a thinner tube to maintain high torque strength for implantability, but the thinner tube makes the structure more flexible in bending. Therefore, an advantage of polyurethane in certain designs is “higher stiffness” combined with “higher tear strength.”

Polyurethane has vastly superior compressive properties, specifically low creep or “cold-flow.” It is also much less prone to abrasion from physical rubbing contact with other leads or the device. In addition, some believe that polyurethane is inherently less thrombogenic than silicone rubber. Although silicone rubber has been available longer, polyurethane has been used in humans as lead insulation for > 30 years.

Polyurethane has been available in a softer, more flexible version known as “80A” and a harder, more flexible version known as “55D.” Early leads made with the 80A polyurethane exhibited higher levels of degradation of the polyurethane insulation. Most current polyurethane leads utilize the 55D version insulation, which is much less prone to the degradation.

Silicone rubber is sometimes criticized for “absorbing lipids (calcifications).” Although lipid absorption is reported in the literature for ball and cage heart valves, it has not been proven to be clinically significant in pacing leads and does not appear to result in failures. Lipid absorption and calcification are two different phenomena. Even though mineralization of encapsulating sheaths (extrinsic mineralization) is common, there is no evidence that it causes lead insulation failure (although it greatly hinders removal of old leads). Mineralization of the silicone rubber per se (intrinsic mineralization) has also been rarely observed, but lead failures from this mechanism have been very rare.

Polyurethane, being “relatively stiffer,” is often used advantageously, especially in a portion or certain segment of a lead, and allows manufacturers to make smaller, tough leads that can have greater torquability, resulting in easier implantability.

At one time, not being “repairable” was a disadvantage for polyurethane insulated leads vs. silicone leads. In the earlier years of cardiac pacing, experienced individuals would at times attempt outer insulation repair of silicone rubber insulated leads with medical adhesive and silicone film. This worked relatively well if done correctly. Also, terminal pin replacement on unipolar leads could be performed, if necessary, with specific repair “kits” from the manufacturers. At this time, repair of any portion of the lead should not be attempted. If a lead is malfunctioning or grossly damaged, it should be abandoned and capped, or extracted and replaced.

Although no longer a significant issue, “sensitivity to manufacturing process” can also be a disadvantage for both materials. Knowledge is required to work with either one. The potential for environmental sur-
face cracking remains true for the 80A polyurethane, but is not a significant mechanism of clinical failure in contemporary 55D polyurethane leads. The potential for metal ion oxidation remains an issue for contemporary 55D polyurethane insulated leads. Despite a blemish on polyurethane leads due to a high failure rate in the 1980s of the 80A version of polyurethane, the overall survival rate for other, newer polyurethane leads utilizing the 55D version has been excellent.14,15

What is an acceptable rate of lead failure? Ideally, leads would never fail, but this level of reliability will never be reached. In the last edition of this text we stated that “Most manufacturers strive for a 5-year lead survival rate of 98% or higher.” The acceptable failure rate for permanent pacing and defibrillation leads (and pulse generators for that matter) is a matter of continued debate.16 During the preparation of this text there were a number of leads and pulse generators that were placed on advisory status, which has significantly fuelled this debate. Some experts have suggested that no worse than 5% cumulative failure rate at 10 or 20 years should be goals for ICD and pacing leads, respectively. However, at this time, no specific acceptable failure rate can be quoted for any component of a pacing, defibrillation or CRT system.

Lead polarity
Bipolar leads are used more frequently in the USA. This preference exists largely because of a lower susceptibility to electromagnetic interference and other far-field signals when pacemakers are in a bipolar sensing configuration.17–19 If examined over decades of use, bipolar leads have had an overall higher incidence of failure than unipolar leads, largely because of the specific bipolar leads which utilized the 80A version of polyurethane that failed in high numbers in the 1980s. If these leads were excluded from the analysis, the survival difference between unipolar and bipolar leads would be minimal.

Lead diameter
Over the years the diameter of pacing leads has decreased. There is still significant variation in the lead body size. Contemporary stylet-driven leads are commonly in the range of 4.5–7 Fr in size. Lead selection may be driven by “size” in some patients. It may be advantageous to use preferentially a small-diameter lead in children and in patients with existing transvenous leads in whom the vessel lumen is compromised.

More recently, a 4.1-Fr lumenless lead has been used. This lead is delivered through a deflectable sheath and has the potential advantage of more site-specific placement of the lead. Long-term experience with the lead is limited at this time (Fig. 4.7).20

Compatibility of lead and pulse generator
The pacemaker lead or leads and pulse generator selected do not need to be from the same manufacturer. It is generally acceptable to “mix and match” leads from company X with a pulse generator from company Y, assuming similar functionality. What is mandatory is that the lead connector be compatible with the connector cavity of the pulse generator.

Historically, unipolar leads were 5 or 6 mm in diameter and bipolar leads were of the bifurcated design with similar 5- or 6-mm sizes. Most leads currently used are of “in-line” bipolar design with a 3.2-mm diameter (Fig. 4.8). In 1986, a voluntary standard for lead connectors and connector cavities was established. This voluntary standard for leads and connectors incorporated sealing rings on a 3.2-mm lead connector and is referred to as VS-1.21 Subsequently, an industry-wide, international standard configuration known as IS-1 (“International Standard 1”) was developed and accepted. Figure 4.9 shows the types of lead connectors available, and Fig. 4.10 demonstrates varieties of in-line bipolar pulse generator headers. “Unipolar” leads comply with the same design features, but the ring electrode on a dedicated unipolar lead is electrically inactive. Currently, virtually all pacing leads comply with the IS-1 connector standard configuration.

Epicardial leads
Epicardial pacing may still be necessary in patients

![Fig. 4.7 Passive fixation bipolar single-pass VDD lead. Note that in the portion of the lead that will be positioned within the atrium, there are two ring electrodes (arrows). Sensing occurs via these “floating” atrial electrodes and allows P-synchronous pacing with a single lead.](image-url)
with congenital cardiac anomalies that prevent the access needed for transvenous leads, with a prosthetic tricuspid valve, or with other tricuspid valve abnormalities that preclude lead placement across the valve. Figure 4.11 demonstrates available types of epicardial fixation mechanisms. Epicardial leads have historically had higher pacing thresholds and less mechanical reliability than transvenous leads. Platinized and steroid-eluting epicardial leads are used in an attempt to keep epicardial pacing thresholds lower.22,23

**Resources for lead performance and survival data**

Lead choice is often determined by the implanter’s personal experience. As new leads become available, it is important to be aware of resources of lead survival and performance. Resources include:

- Manufacturers’ data (e.g., active lead registries, returned lead analysis, etc.)
- Published information from individual centers or consortium of larger medical centers
- Public databases.

Manufacturers are required by law to collect post-market surveillance data on hardware performance. Various manufacturers use different approaches for collection and analysis of this information, but performance data should be available from any manufacturer on request. Figure 4.12 is an example of lead performance and survival data from one manufacturer in a report that is regularly updated.

Follow-up data may be obtained from active registry information24,25 or from centers that publish survival and performance data on individual leads (see Chapter 10, Troubleshooting). A literature search is likely to yield implanters with information on many of the most widely used leads.26
Fig. 4.11 Commonly used epicardial fixation mechanisms. From left to right: Medtronic 5071 screw-in lead; Medtronic 4968 steroid-eluting suture-on bipolar epicardial lead; Metronic 4965 steroid-eluting suture-on unipolar epicardial lead.

Fig. 4.12 Examples of product performance data from three different manufacturers that is taken from the product performance reports available for use by caregivers. (Information is in the public domain so anyone can access the product performance information.)

A Postmarket surveillance data from product performance report by (A) Medtronic—data regarding a dual-chamber ICD; (B) St. Jude Medical—data regarding a dual-chamber pacemaker; (C) Boston Scientific—data regarding specific “family” of pacing leads.
**Integrity® AFx DR (Models 5342 & 5346)**

<table>
<thead>
<tr>
<th></th>
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<th>Normal Battery Depletion</th>
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<tr>
<td>US Market Release</td>
<td>(5342)</td>
<td>April 2000</td>
<td>4083</td>
</tr>
<tr>
<td>(5346) July 2001</td>
<td></td>
<td></td>
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<td>Registered US Implants</td>
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<tr>
<td>Estimated Longevity</td>
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**Including Normal Battery Depletion**

- **Y-axis:** Percentage
- **X-axis:** Years After Implant

<table>
<thead>
<tr>
<th>Year</th>
<th>Survival Probability</th>
<th>1 standard error</th>
<th>Sample Size</th>
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<tr>
<td>2</td>
<td>99.93%</td>
<td>0.01%</td>
<td>42,100</td>
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<tr>
<td>4</td>
<td>99.80%</td>
<td>0.02%</td>
<td>33,700</td>
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<tr>
<td>6</td>
<td>97.02%</td>
<td>0.07%</td>
<td>17,400</td>
</tr>
<tr>
<td>at 84 months</td>
<td>85.02%</td>
<td>0.15%</td>
<td>500</td>
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**Excluding Normal Battery Depletion**

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<td>6</td>
<td>99.77%</td>
<td>0.03%</td>
<td>17,400</td>
</tr>
<tr>
<td>at 84 months</td>
<td>99.76%</td>
<td>0.02%</td>
<td>500</td>
</tr>
</tbody>
</table>

Fig. 4.12 (Continued.)
There is no independent comprehensive database of leads or pulse generators in the USA. Although there have been attempts at developing such a database, it has yet to be accomplished.

In 2005 in the USA, the Center for Medicare and Medicaid Services initiated regulations that require implanting centers/hospitals to supply certain follow-up information about ICD patients in order to get reimbursement. However, the registry, the National Cardiovascular Device Registry (http://www.accncdr.com/webncdr/ICD/Default.aspx, etc.) is run privately with the American College of Cardiology and Heart Rhythm Society jointly involved with ICD data management.27

Another database is operational for documenting hardware failures for leads, pacemakers and ICDs. Because only failures are reported, the database does not provide the incidence with which specific failures might occur, but it may alert one to a potential problem or allow a search to see if others have reported a similar problem (http://www.pacerandicdregistry.com).24,25

**Generator and lead selection in defibrillators**

Many of the considerations in selecting an appropriate pacing system for a patient, e.g., the use of tined or active fixation leads, the need for specific pacing features, etc., are identical in defibrillator selection and are not repeated here. Other issues, however, either are unique to defibrillators or take on added dimensions, including:

- Use of integrated or bipolar sensing
- Pulse generator size in relation to longevity
- Maximum shock output
- Upper pacing rate
- Pacing and defibrillation with a dual- or a single-chamber device and arrhythmia discrimination
- Features of a specific device (or lead) to allow solving commonly encountered ICD problems (inappropriate shocks, far-field oversensing, etc.).

**Integrated vs. bipolar sensing**

The transvenous defibrillator lead, typically placed in the right ventricle, serves to sense signals (local electrogram), pace the heart (antibradycardia and antitachycardia pacing (ATP)), and deliver shocks. ICD leads are tripolar (tip and ring electrodes with one coil, or tip electrode with two coils) or quadripolar (tip and ring with two coils) with variation in the distribution of function across these electrodes. True bipolar sensing occurs between the tip electrode and a closely spaced dedicated ring (Fig. 4.13). Since the spacing between this bipolar pair is small, far-field electrograms
for noise related to lead interaction or electromagnetic interference is less likely to be sensed.

Integrated sensing lead systems are constructed with a distal tip electrode for pacing and sensing, a distal coil for both pacing and sensing and for defibrillation, and a proximal coil for defibrillation (Fig. 4.13). This design has the advantage of incorporating two defibrillation coils, which in conjunction with an active can device lowers defibrillation thresholds (DFTs), although this effect has not been uniformly observed.28,29 In early lead designs, the distance from the tip to the distal coil was small (6 mm) to minimize detection of far-field signals; however, it was found that with a distally placed coil, the amplitude of the recorded electrogram diminished significantly after a shock, an effect that could on rare occasions lead to failure to redetect continuing ventricular fibrillation if an initial shock was unsuccessful (Fig. 4.14).30 Subsequent to this early experience, integrated leads have been redesigned with a greater distance between the distal coil and the lead tip (12 mm), ameliorating postshock electrogram diminution, so that it is no longer a clinical consideration.31,32 Although the greater distance between the tip and the coil has resolved postshock sensing problems, with the trade-off is an increased risk of oversensing of far-field signals. The cause is the effectively larger antenna created by the greater intra-bipole distance. Moreover, to detect the small-amplitude fibrillation signals that may follow a relatively large R wave, defibrillators utilize dynamic sensing or gain. In most defibrillators, the effective sensitivity after a sensed R wave increases with each passing millisecond until the maximum sensitivity is reached33 (Fig. 4.15). Thus, patients with slow heart rates—which allow more time after a QRS complex for the effective sensitivity to increase—are at increased risk for this type of oversensing. Since sensitivity (or gain) is rapidly maximized after a paced event, patients with slow rates of pacing are the most likely to experience far-field myopotential oversensing, often because phrenic potentials are oversensed (Fig. 4.16). When it occurs, it can result in suppression of bradycardia pacing or inappropriate detection of ventricular tachyarrhythmias.34 Increasing the lower rate and decreasing sensitivity usually eliminate the problem. In our practice, the predominant situation in which true bipolar sensing is preferred is in the setting of abandoned intravascular leads. In theory, the distal coil in integrated lead systems may make contact with an abandoned lead and generate lead noise. In practice, this is an uncommon occurrence (unpublished observation), and it may be further mitigated with the use of expanded polytetrafluoroethylene (ePTFE)-insulated coils.35

Although having two coils in the ICD leads offers modest advantages in terms of lowering DFT, disadvantages exist. When lead extraction becomes necessary, the proximal [superior vena cava (SVC)] coil is often densely adherent to the SVC and may be difficult to remove. In a small minority of patients (< 5%), the location of the proximal coil may be ill suited for defibrillation, necessitating excluding it from the shocking circuit or the addition of other defibrillation leads. Because of this, some implanters prefer to use a single coil–true bipolar sensing lead and then add an additional lead if DFTs are
high. The additional lead may be placed in the SVC or in the coronary sinus and its ventricular tributaries or the IVC. The additional coil may also be placed in a branch of the SVC such as the azygous vein.

In summary, for most patients dual- or single-coil and true or integrated bipolar systems are effective. Single-coil systems are favored in younger patients who have a potential need for future extraction, although ePTFE-coated leads may mitigate the disadvantage of dual coils. True bipolar sensing is preferred in pacemaker-dependent patients, in the setting of abandoned leads, and for patients who may be at increased risk for exposure to electromagnetic interference (e.g., a factory worker). Dual-coil systems may be preferable when higher DFTs are anticipated (hypertrophic cardiomyopathy, antiarrhythmic therapy, some sodium channel defects), although clinical predictors of high DFTs have been limited, and <5% of patients require system revision.36–38

**Size and longevity**

In general, larger devices have greater battery capacity and thus greater longevity. Patient size, particularly in smaller patients or children, may rarely constrain the site of device placement, because of either limited pectoral tissue to support the device or cosmetic concerns. In these situations, the options available are smaller
CHAPTER 4 Generator and Lead Selection

135

devices with shorter longevity, subpectoral placement, and less commonly used sites. When device size becomes a significant factor, the technically simplest solution may be to use the smallest available device and insert it prepectorally, despite potential limits to longevity. Even with selection of the smallest device, alternative insertion sites may be preferable at times. Submuscular placement is discussed in greater detail in Chapter 5, Implantation Techniques. Further technological advancements will result in progressively smaller devices in the near future.

Programmable waveforms

Elevated DFTs are an uncommon but challenging problem affecting approximately 5–10% of the ICD population, and 5% of current implants. Patients with elevated DFTs and reduced safety margins may have reduced survival compared with other ICD recipients. Most ICDs utilize fixed tilt biphasic waveform. At present, a single manufacturer (St. Jude) manufactures ICDs in which the waveforms are programmable. Small studies suggest that tuning waveforms improves defibrillation and permits a majority of high DFT patients to achieve implant criteria, although experts disagree about the benefit of pulse-width-based waveform tuning.

Dual-chamber or single-chamber ICD?

The single most important decision in selecting a defibrillator is determining whether to implant a single- or a dual-chamber system. This decision may be influenced by several considerations:

- The need for dual-chamber bradycardia pacing because of a standard indication, such as sinus node dysfunction or AV block
- Specific conditions (e.g., long QT syndrome or hypertrophic cardiomyopathy) that may respond to dual-chamber pacing
- Congestive heart failure, which occasionally may require traditional dual-chamber or, more commonly, biventricular pacing
- Paroxysmal atrial arrhythmias, for improved specificity, prevention, and therapy.

These are discussed in greater detail below. Table 4.3 summarizes conditions favoring the use of dual-chamber systems. For any given patient, the potential advantages of dual-chamber systems must be weighed against the strengths of single-chamber defibrillator-system simplicity, reduced risk of lead dislodgment, reduced cost, and greater longevity per unit size (see Table 4.4).
Factors favoring single-chamber defibrillators
Patients with chronic atrial fibrillation or patients who lack the factors favoring dual-chamber devices should receive single-chamber defibrillators. This is particularly true for younger or smaller patients, in whom the greater longevity per unit size and minimal intravascular hardware requirements of single-chamber systems may be more compelling. In addition to simplicity and longevity, single-chamber benefits include fewer complications. Although previous studies suggested no significant difference in overall complication rates between single- and dual-chamber pacemakers, the preponderance of evidence suggests that atrial leads dislodge more frequently than do ventricular leads.43,44 Unless dislodged atrial leads are electrically abandoned (thus functionally reducing the implanted system to a single-chamber ICD), this increased rate of dislodgment may result in higher reoperation rates for dual-chamber ICDs. Thus, for patients who have infrequent pacing, who lack episodic atrial arrhythmias, and who do not have specific conditions warranting dual-chamber devices, single-chamber devices are preferred.

Factors favoring dual-chamber defibrillators
Dual-chamber defibrillators are preferred in patients with an accepted indication for dual-chamber pacing. In sinus node dysfunction, atrial pacing (combined with avoidance of ventricular pacing) modestly lowers the risk of atrial fibrillation.45–47 The addition of β-adrenergic blockers or antiarrhythmic medications may further exacerbate bradycardia and the need for atrial pacing support. In patients with high-grade AV block, a dual-chamber biventricular device is preferred if heart failure is present, given the association of heart failure and chronic right ventricular apical pacing.47,48 These are discussed further below.

In addition to providing dual-chamber pacing functionality, dual-chamber defibrillators use the information acquired simultaneously from atrial and ventricular leads to enhance arrhythmia diagnosis. Early dual-chamber ICDs were similar or slightly superior to single ICDs in correctly discriminating supraventricular tachycardia (SVT) from ventricular tachycardia (VT). More recently, prospective, randomized trials have found that the odds of inappropriate detection of SVT as VT were decreased by half with the use of dual-chamber detection.49 As expected, the improved rhythm classification led to a reduction in inappropriate therapy. In this study, more ATP was programmed in the single-chamber arm, and shock rates did not differ, but another study has demonstrated a reduction by one-third in clinically significant adverse events with use of dual-chamber detection enhancements.50 Although expert opinion is divided, selection of a dual-chamber ICD to improve SVT-VT rhythm classification is reasonable in patients in whom a VT zone with rates < 200 ppm will be programmed who are not in chronic atrial fibrillation and who do not have complete or high-grade AV block (Table 4.4).

Clinical factors that should be considered when making an ICD selection are summarized in Table 4.5.

Specific device and lead features influencing selection
When selecting a particular device or lead for an ICD
system, the implanter should briefly consider whether any specific patient characteristic warrants a distinct programmable feature or lead characteristic. In general, today’s generators and leads are highly flexible in their programming options and lead characteristics (true bipolar sensing along with dual coil, etc.), so that any choice will work in most situations. However, in certain situations, specific features may be more appropriate on a certain device to offset the likelihood of the most common ICD-related problem, namely, inappropriate shock for SVTs and sensing related issues.

**Avoiding adverse effects from the lead or from frequent and unnecessary pacing**

As discussed under Pacemaker selection, frequent right ventricular pacing may promote cardiomyopathy. Some ICDs incorporate similar methods to decrease ventricular pacing in addition to enhanced programmability of the AV intervals (AV search hysteresis, MVP). When dual-chamber devices are selected for a patient primarily because of possible improved discrimination between SVT and VT, care must be taken to minimize ventricular pacing. This may be done algorithmically, or by utilizing a non-tracking pacing mode at sufficiently low rates to minimize pacing.

As noted previously, tricuspid valve regurgitation as a result of the lead crossing this valve appear more likely to occur with ICD leads than with standard pacing leads. The exact incidence of tricuspid regurgitation and relative benefits of choosing a particular lead or lead location are not presently known. In general, smaller profile ICD leads placed with care to avoid perforation and avoiding placement close to the insertion of the papillary muscle on the moderator band are likely to be beneficial.

**Cardiac resynchronization therapy**

There are some specific considerations when selecting a generator, lead, and device function when using car-

<table>
<thead>
<tr>
<th>Clinical factor</th>
<th>Lead and device considerations</th>
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<tr>
<td>Pacemaker-dependent patient</td>
<td>• True bipolar sensing preferable</td>
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<tr>
<td>Need for bradycardia pacing</td>
<td>• Confirm that appropriate pacing (sensor, dual-chamber) is made available</td>
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<tr>
<td>Paroxysmal atrial arrhythmias</td>
<td>• Dual-chamber devices enhance specificity and lower risk of inappropriate therapy (particularly if VTs with HR &lt; 200 are present)</td>
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<tr>
<td>Chronic atrial fibrillation</td>
<td>• Atrial pacing modestly reduces paroxysmal atrial fibrillation (in sinus node dysfunction) as long as ventricular pacing minimized</td>
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<tr>
<td>Anticipated elevated defibrillation</td>
<td>• In patients with atrial flutter, previous heart surgery and incisional atrial flutters, atrial ATP may be useful</td>
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<tr>
<td>threshold (previously high defibrillation threshold, hypertrophic cardiomyopathy, marked enlargement)</td>
<td>• Atrial shocks are infrequently used</td>
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<tr>
<td>Small body habitus; younger patient; need for limited intravascular hardware</td>
<td>• Single-chamber device (VVIR or rarely VVI)</td>
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<tr>
<td>Very slow ventricular tachycardia</td>
<td>• Higher output device</td>
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<td></td>
<td>• Dual coil lead</td>
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<td></td>
<td>• Consider programmable waveform if fixed tilt waveform has failed</td>
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<tr>
<td></td>
<td>• Smaller size, single chamber preferable; consider single coil lead or ePTFE-coated lead</td>
</tr>
<tr>
<td></td>
<td>• Consider relationship of upper rate limit to ventricular tachycardia detection zone</td>
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VT, ventricular tachycardia; HR, heart rate; ATP, antitachycardia pacing; ePTFE, expanded polytetrafluoroethylene.
Cardiac resynchronization devices. In order for the patient to benefit, as close to 100% of QRS complexes should be resynchronized (i.e., paced with capture). CRT pulse generators require particular timing algorithms to maximize biventricular pacing, and LV pacing leads employ special design to maximize stability and minimize extracardiac stimulation.

**Promoting continuous biventricular pacing**

As discussed previously in this chapter, unnecessary right ventricular pacing may promote ventricular dysfunction and is possibly disadvantageous, thus, pacing algorithms have evolved to minimize unnecessary right ventricular pacing.

The exact opposite goal is the cornerstone of CRT. The goal when implanting these devices is to have near-continuous biventricular stimulation (perhaps with a programmed RV-LV offset). The main interruptors of resynchronization are atrial fibrillation with rapid intrinsic conduction, an inappropriately long programmed AV interval, and frequent premature ventricular contractions. A variety of programmable variables exist to help manage these specific challenges. These are discussed in Chapter 10, Troubleshooting and Chapter 8, Programming.

Because continuous biventricular pacing is the goal, in general, generator replacement is required earlier with CRT devices than with standard pacemakers. In addition, LV pacing thresholds are often higher than those obtained from endocardial right ventricular pacing. Specific device features have been developed to try to optimize battery life without compromising biventricular pacing. Algorithms have been developed to determine the minimum amplitude that consistently results in ventricular capture and calculate the new amplitude based on a programmable safety margin and the programmed maximum LV adapted amplitude. In essence, the LV output is kept at a safe margin as determined by these programmed guidelines, yet maintaining capture and optimizing device longevity. Such features should be considered when device selection is made.

**Leads for resynchronization devices**

As discussed in Chapter 5, Implantation, distinct challenges are present when negotiating the coronary venous tree and create unique requirements for LV pacing leads. LV pacing leads should be sufficiently flexible in negotiating sharp angulation and tortuosity of the venous system without traumatizing or dissecting these veins. The lead must be large enough to wedge into a venous tributary, yet small enough to negotiate collateral veins, often requiring subcannulation to reach the LV free wall.

Coronary venous leads should be maneuverable to an intramyocardial location to decrease the likelihood of extracardiac stimulation and should be designed to allow multisite or multivector stimulation (see below).

Historically, when LV pacing was first being performed, standard tined right ventricular pacing leads were maneuvered into the coronary sinus and into the ventricular branches with appropriate shaping of the inserted stylet. Shapes were made mimicking standard preformed coronary sinus mapping catheters used in electrophysiology. Later, dedicated left ventricular pacing leads that had a relatively more flexible curved distal segment (Medtronic model 2187) were developed. This lead and another similar lead (Medtronic 2188) were those used in the first large multicenter trial evaluating left ventricular pacing. Despite some designed facilitation of lead delivery, these leads were stylet driven and could not be passed through the then available coronary sinus guiding sheaths. Dislodgment rates were high (8%), and extracardiac stimulation continued to be a significant problem, with reprogramming to turn off the LV lead occurring in 8.8% of patients. Further acute hemodynamic studies suggested that the midlateral LV free wall should be the target for pacing, and maximal separation from the right ventricular lead was thought desirable.

As implanters attempted to place leads at specific sites thought to optimize synchrony, stability of these
leads at these specific sites became more important. Sometimes the coronary venous system is large (varices) making lead contact with myocardium suboptimal, and at other times excess tortuosity makes judging “slack” on these leads difficult and results in extrusion of the lead from the vein with patient movement or deep respiration. The St. Jude model 1056k LV lead (5.6 Fr polyurethane body and 5 Fr silicone distal tip) is a lead that promotes stabilization in the coronary vein with an S-shaped tip designed to stabilize itself on opposing walls of a larger vein of interest. Another attempt to optimize stability in relatively larger veins is Boston Scientific’s Easy Track 3 lead (6 Fr polyurethane body and 5.7 Fr silicone distal tip). When the stylet or guidewire is removed from this lead, a terminal helix curls and shortens in the coronary vein, promoting stability. Other leads do not have a preformed helix or curve mechanism and are essentially straight (Boston Scientific’s Easy Track 2 lead) to allow ease of maneuverability into the distal coronary venous system. In the Medtronic 4194 lead, the construction of the proximal electrode is essentially a coil rather than an annular electrode with a large (38 mm²) surface area.

A variety of coronary venous leads are available. Examples of contemporary coronary venous leads are shown in Fig. 4.17. Developing approaches include active fixation mechanism, extrusion of adhesion molecules, and multiple electrodes, all aiming to promote stability or continued capture even if the lead is unstable.

**RV-LV pacing offset and vector of pacing**

The benefits and technique for sequential biventricular pacing are discussed elsewhere in this book. In observational studies, acute hemodynamic improvement and more optimal Doppler indices of resynchronization are observed when the LV output occurs earlier than that of the right ventricle by approximately 40 ms. Several presently available CRT generators allow programmability of the V-V interval. In addition, bipolar or multipolar pacing leads allow noninvasive manipulation of the pacing vector. For example, if a bipolar LV pacing lead is placed, bipolar LV stimulation may be chosen or stimulation between the LV lead tip (or vein) at the cathode and the right ventricular defibrillator coils. This may not only allow adequate pacing thresholds when bipolar capture thresholds are high, but a different pacing vector that may promote better synchrony. Manipulation of the pacing vector may also help in troubleshooting extracardiac stimulation and anodal stimulation (see Chapter 10).

In addition to the general requirements for optimal pacemaker/ICD generator programmability and lead characteristics, unique requirements thus exist for CRT devices because of the requirements for continu-
ous pacing and the unique lead location in the coronary venous system.

**Conclusion**

The broad array of devices and lead systems now available has enhanced the opportunity to tailor device therapy to the individual patient.

Pacemaker selection should be made after consideration is given not only to the underlying rhythm disturbance but also to the patient’s activity level and need for specific programmable options. For ICD selection, factors such as pacemaker dependency, anticipated defibrillation threshold, need for bradycardia pacing, and paroxysmal or chronic atrial arrhythmias may all affect the choice of system (summarized in Table 4.5). Although most defibrillator systems are suitable for most patients, careful selection of system components can provide the best match between patient and device.

Depending on whether the CRT patient requires CRT-P or CRT-D, multiple factors need to be weighed in choosing a system for this subset of patients. The patient’s underlying rhythm also needs to be carefully considered in order to maintain biventricular advantage and take complete advantage of resynchronization.

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**Fig. 4.17 (Continued.)**
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CHAPTER 5

Implantation and Extraction Techniques

David L. Hayes, Paul A. Friedman, Samuel J. Asirvatham

The physician implanting a cardiac rhythm device must have a thorough working knowledge of cardiac pacing, defibrillation and resynchronization as well as a complete understanding of sterile technique and the specific surgical skills necessary for pulse generator implantation. For cardiac resynchronization therapy this also means thorough knowledge of coronary venous anatomy and techniques for cannulating and navigating the coronary venous system.

Also included in this chapter is a discussion of lead extraction as an implant-related procedure. Complications that may result from device implantation are discussed in Chapter 6.

Implantation facility

Pulse generators should be implanted in a surgical environment, whether a specially equipped operating room or a catheterization suite (Fig. 5.1). Requirements include excellent fluoroscopy, electrocardiographic monitoring, oxygen saturation monitoring (by finger plethysmography), and standby defibrillator and life-support equipment. Additional desirable features are facilities for simultaneous lateral and anteroposterior fluoroscopy projection, a fluoroscopy table capable of tilting, cineangiographic capability with freeze-frame, and intra-arterial pressure monitoring equipment.

Anesthesia

Local anesthesia is used for most adult patients unless contraindicated. Pediatric patients, uncooperative or confused patients undergoing device implantation, and patients with special circumstances may require general anesthesia. Lead extraction, discussed later in this chapter, is usually performed under general anesthesia.

Supplemental parenteral sedatives are used as needed for patient comfort. We commonly use intravenously administered midazolam and fentanyl, the dose depending on individual patient requirements, associated cardiopulmonary disease, other comorbidities, and age.

Conscious sedation may be administered by certified registered nurse anesthetists or registered nurses (RNs) trained specifically to perform this function. An expert consensus document is available that details the use of intravenous (conscious) sedation/analgesia by non-anesthesia personnel in patients undergoing device implantation and other electrophysiological procedures.

The pulse generator pocket

Forming the pulse generator pocket is an integral part of pulse generator implantation. The pocket is commonly developed in the prepectoralis fascia (Fig. 5.2). It should be large enough to allow for easy placement of the pulse generator and leads. It is important to avoid a pocket which is too tight, because it can result in erosion or an oversized pocket, which can permit excessive movement and device migration (see Chapter 6, Implantation-related Complications).

The site of placement of the pulse generator is extremely important in providing long-term comfort and complete mobility for the adjacent shoulder. The pulse generator should be implanted in the subcutaneous tissue, deep to the fatty layer of the pectoral region. An inexperienced operator may not find the
plane between the subcutaneous tissue and the pectoral fascia. Occasionally, the space developed may be subcuticular, with the subcutaneous fatty layer deep to the pulse generator. In that situation, the pulse generator presses on the undersurface of the skin, which may be continually painful. Characteristically, light touch of the overlying skin produces exquisite pain. An equally inexperienced evaluator of this circumstance may not understand the problem, which can be solved by repositioning the pulse generator into a deeper and subcutaneous site.

The pulse generator should be sufficiently inferior to the clavicle so that a full range of shoulder motion is not restricted by its impingement against the clavicle. The device should be sufficiently medial to keep it from approaching the anterior axillary fold. Otherwise, every anterior movement of the arm past the pulse generator will be uncomfortable.

Infiltration of local anesthetic in the deltopectoral groove (lateral incision) may also result in injury to the thoracoabdominal nerves and result in chronic pain. After dissection and development of the pocket it should be inspected for any bleeding and bleeding areas cauterized. A sponge soaked with saline or antibiotic solution may be kept within the pocket during placement of the leads in an effort to minimize oozing
and blood in the field. However, removal of the sponge is mandatory before wound closure. We have discontinued this practice, given the potential for leaving a foreign object in the pocket (Fig. 5.3).

As pulse generator size has decreased, this has become less of a selection parameter for most patients. The smallest available pulse generators may be of particular importance in pediatric patients, thin patients and patients with cosmetic concerns. However, the downside of the smallest pulse generators is often a compromise in device longevity. If the prepectoral position is not suitable because the optimal pulse generator is too large or because cosmetic or other considerations predominate, other choices are possible. Although rarely necessary, the pulse generator can be placed deep to the pectoral muscle. When the implanting physician is experienced with this technique it can be accomplished with relative ease. It should not be performed by someone inexperienced, because vascular and neural structures could be damaged if there is no familiarity with the anatomy encountered with this procedure.

Retromammary pulse generator placement, a rarely used option, may be employed for cosmetic purposes or, in a thin person, for protection of the implant site by the fatty layer of the breast (Fig. 5.4). However, most contemporary pulse generators are small enough to be implanted prepectorally without causing any significant cosmetic concern. A potential disadvantage of a retromammary technique is that of potentially compromising breast imaging.

Implantable cardioverter-defibrillators (ICDs) and cardiac resynchronization therapy (CRT) devices are larger than pacemakers and may cause more cosmetic concerns; placing either of these pulse generators in a retromammary position could potentially impair optimal mammography and therefore be of clinical concern. In fact, all devices should be implanted in such a way that the device obscures the minimum of breast tissue. It is also reasonable to be certain that a female patient has had recent mammography before ICD or CRT-D implantation.
Venous approaches

Before 1979, transvenous pacing leads were almost always placed through a cephalic vein cutdown in the deltopectoral groove. If the cephalic vein was too small or friable, or had previously been used for implantation or if a second lead was required, the ipsilateral external or internal jugular vein was used. Rarely, subclavian or axillary vein cutdowns were performed. Deep dissection demanded precise surgical techniques—techniques in which cardiologists are not often skilled.

Each venous approach has its own particular advantages and disadvantages. The axillary, also referred to as the extrathoracic subclavian approach, or subclavian puncture and cephalic cutdown approaches, are most commonly used today. Placement through the external or internal jugular veins, in addition to more local dissection, requires the operator to tunnel the lead over or under the clavicle to the pulse generator and is largely of historical interest. Techniques for permanent lead placement via the iliac vein have also been described, and should be considered when there is limited venous access.

Axillary (extrathoracic subclavian) approach

The introducer approach for axillary and subclavian venepuncture is frequently used, because this technique is fast, usually causes minimal trauma, and facilitates placement of multiple leads. The basic procedure, a modification of the Seldinger technique, requires detailed knowledge about the route of the axillary and subclavian vein and the relationship of the vein to the clavicle, first rib, arterial blood supply, and apex of the lung (Fig. 5.5). The ease, efficacy and safety of the technique are directly related to adherence to specific guidelines and to the expertise of the physician performing the venepuncture.

Prepackaged kits containing a needle, guidewire, dilator and peel-away sheath are available for venous puncture. The vein is entered through the incision with an 18-G needle (Fig. 5.6). (Placing the patient in the Trendelenburg position may facilitate entry, because the vein may be less distended in the recumbent patient with normal venous pressure. However, this is not an option with some tables used in the operating suite. Entry is even more of a problem if the patient

has been fasting for several hours before the procedure and is volume-depleted.)

The axillary vein is a continuation of the brachial vein. The basilic and cephalic veins are tributaries of the axillary vein and the axillary artery courses alongside the axillary vein. The axillary vein terminates at the lateral margin of the first rib, at which point it becomes the subclavian vein, i.e., the axillary vein is extrathoracic. If venous access is accomplished in an extrathoracic position there is no risk of pneumothorax (Fig. 5.7). (Multiple techniques have been described for axillary puncture.4–6)

The technique may be facilitated by inserting the needle in the lateral aspect of the incision. Under fluoroscopic guidance the needle is directed at the first rib, taking care not to cross the lateral margin of the first rib and thus avoiding entry into the intrathoracic space. The needle can be “walked” along the first rib. Venepuncture should be performed with a syringe with saline or 1% lidocaine attached to the needle. As the puncture is performed, a slight vacuum should be maintained on the syringe. The syringe should be constantly observed for the aspiration of air, blood or other fluids. Once the vein is successfully punctured, the guidewire is advanced through the needle and into the right side of the heart under fluoroscopic control (Fig. 5.8). On occasion, this guidewire enters the jugular system and ascends. This error can usually be corrected by partial withdrawal of the wire, rotation, and re-advancement under fluoroscopy. After removal of the needle, the introducer, dilator and peel-away sheath are advanced over the guidewire into the central circulation (Fig. 5.9). Selection of the appropriately sized introducer is based on the size of the lead or leads to be used. After removal of the dilator and guide, the pacing lead or leads are advanced through the sheath into the right side of the heart. One should
take care to avoid air embolism during this procedure. At a minimum, the patient should not talk during this portion of the procedure, and some implanters prefer to have the patient hold respiration until passage of the lead into the right side of the heart. The sheath is then peeled away (Fig. 5.10).

If the vein is not successfully located by probing with walking the needle along the first rib, a peripheral contrast injection will allow visualization of the axillary vein and redirection of the needle to the point on the first rib where the vein is identified. Some implanters prefer to perform contrast venography before the initial puncture attempt.

Contrast venography requires no additional preparation with the exception of determining that the patient does not have a contrast allergy and placing an intravenous line (preferably 20G or larger) in the arm on the side of the pulse generator implantation. The intravenous line should be checked for patency before injection of contrast material. The contrast injection can be done by anyone in the room with access to the peripheral, ipsilateral intravenous line. Approximately 10–20ml
of preferably nonionic iodinated contrast medium is injected through the intravenous line in the forearm. A saline bolus may be used to flush the contrast agent into the venous system if necessary. It may also be helpful to massage the arm to assist in venous return and venous visualization. This can be done either under the sterile drapes by an assistant or over the sterile drapes by the implanter. The subclavicular area is monitored with fluoroscopy for the appearance of contrast, which usually takes from 5 to 20 s (Fig. 5.11). Repeat injections are given as needed, although the total contrast burden should be monitored and kept to the minimum possible, and the implanter should be aware of the patient’s renal function. If the contrast venogram demonstrates a nonpatent vein the implanting physician may need to choose a different venous route. This may be especially useful when a new lead needs to be placed due to a failed lead or when upgrading a system (Fig. 5.12).

As noted, some implanters prefer to use contrast venography routinely. Alternatively, in patients with normal costoclavicular relationships, no prior permanent pacing leads, and no venous thrombosis, a “blind” venepuncture can be first attempted as previously described. In patients who may have unusual venous anatomy, it may be desirable to use contrast venography before the initial attempt to ensure venous patency and location. Such patients might include those with kyphoscoliosis, prior clavicular fracture, prior surgery in the vicinity of the puncture site, or previous lead implantation. For difficult cases, ultrasound can also be used to identify the position of the vein by placing the probe in a sterile sleeve.

**Subclavian approach**

Although subclavian puncture was the preferred ve-
CHAPTER 5  Implantation and Extraction Techniques

nous route at one time, many implanters have converted to an axillary approach to minimize the risk of pneumothorax. However, implanting physicians should be familiar and comfortable with the subclavian approach. Historically, it was taught that the subclavian vein was entered at the junction of the middle and inner thirds of the clavicle or even with a very medial approach, which was referred to as the “safe introducer technique.” A very medial approach predisposes the lead or leads to crush injury between the clavicle and the first rib.4 As with the axillary technique, some implanters prefer a contrast-guided technique. The anteroposterior location of the subclavian vein is not appreciated with anteroposterior fluoroscopy. Needle passes may occasionally be too anterior or posterior, despite appearing to be within the column of contrast. Varying the fluoroscopic view may aid exact visualization of the vein.

Potential complications of subclavian vein puncture include pneumothorax, hemopneumothorax, lung laceration, inadvertent arterial puncture, air embolism, arteriovenous fistula, thoracic duct injury and brachial plexus injury. Pneumothorax is the most common complication, and, as noted above, use of contrast venography minimizes this risk. Contrast venography also lessens the risk of inadvertent subclavian artery puncture. Meticulous attention to technique is required to minimize any of these risks.

Cephalic approach

Some implanters prefer the cephalic cutdown approach, which avoids the risks of a pneumothorax.10 The cephalic vein lies within the deltopectoral groove. The deltopectoral groove is a constant anatomical site between the deltoid and pectoralis major muscles. The cephalic vein always accommodates a single lead and often accommodates two leads. The operative skills required for the cephalic approach are modest and can be readily taught to anyone sufficiently skilled to perform subclavian puncture or other invasive cardiovascular procedures. Cannulation of the cephalic vein is free of significant complications. If damaged, the vein can be ligated, with prompt cessation of bleeding. In addition, the normal venous pressure and the venous valves prevent aspiration of air into the central circulation.

If the cephalic vein is too small to accommodate even a single lead, a guidewire technique may be useful. For this technique, the cephalic vein is opened in the usual manner, but instead of attempting to pass the lead, the implanter places a guidewire through the opening into the superior vena cava (SVC) or right atrium. The introducer is then placed over the guidewire, as in a conventional subclavian approach, and a lead or leads are introduced (Fig. 5.13).

Jugular approach

This approach is largely of historical interest and should be attempted only by someone with a thorough knowledge of the anatomy and required techniques. In the unusual situation where the external or internal jugular vein is selected, two incisions are required. An incision is made immediately above the clavicle, over the area between the posterior border of the sternocleidomastoid muscle and the anterior border of the trapezius muscle.1 External jugular access to the heart is usually easier by the right external jugular vein than by the left, because the vessel is often less tortuous. If no satisfactory external jugular
vessel is found, the incision is extended to a point anterior to the clavicular head of the sternocleidomastoid muscle. The carotid sheath is exposed after the superficial fascia is opened behind the posterior border of the sternocleidomastoid muscle. The muscle is then elevated to visualize the carotid sheath optimally. On occasion, the clavicular head of the sternocleidomastoid muscle must be divided to expose the carotid sheath (Fig. 5.14). The carotid sheath is then opened; the internal jugular vein is identified and isolated with nonabsorbable ligatures. After venotomy, the lead or leads can be introduced. Use of either the external or the internal jugular vein requires that the lead be tunneled down to the pulse generator site, either superficial or deep to the clavicle. In addition, the internal jugular procedure requires more extensive dissection, with the possibility of damage to the subclavian artery and vein and the recurrent laryngeal nerve.

An alternative method of placing a pacemaker lead via the internal jugular vein is with percutaneous access to the jugular vein and subsequent tunneling of the lead to an infraclavicular pocket. In this approach, cutdown of the jugular vein is not performed. Rather, a standard Seldinger technique with percutaneous access to the supraclavicular portion of the internal jugular vein is performed. Once access has been obtained, a peel-away sheath and, through this, the pacing lead is placed as described below. A small incision at the site of jugular venepuncture is created and the leads secured in the region of this incision. A second infraclavicular incision is made and the pacemaker pocket fashioned.
CHAPTER 5  Implantation and Extraction Techniques

as usual. The pacing lead is now tunneled (usually over the clavicle) and secured with a sleeve onto the pectoralis muscle as well. Some operators may only secure the lead to the pectoralis muscle with no suturing of the lead above the clavicle. The lead is then interfaced with a generator in the usual fashion.

Iliac vein approach
Although rarely used at our institution, the iliac vein approach is worth knowing in the event that the more commonly used venous routes are not accessible (Fig. 5.15).

The recommended procedure is to puncture the iliac vein with a standard puncture technique after making a small incision just above the inguinal ligament and to carry the dissection down to the fascia above the vein. After the puncture is made and lead or leads placed, a purse-string suture is placed to provide hemostasis. A pocket, superficial to the rectus sheath, is created lateral to the umbilicus.11,12

Ventricular lead placement
Successful placement of a reliable right ventricular (RV) pacing system requires knowledge of, and experience with, the specific lead used, as well as knowledge of right heart anatomy and catheterization techniques. Great care must be taken during implantation to avoid damage to the lead. The lead stylet, in particular, should not be forced, because forcing may result in perforation of the stylet through the conductor coil and into the insulation. Keeping the stylet clean, free of blood, and moistened with saline helps avoid trauma to the lead during multiple stylet changes. Several placement techniques are used; all must result in a stable RV catheter position with adequate pacing and sensing thresholds. RV pacing leads have most commonly been positioned in the RV apex. However, a great deal of controversy exists over whether other positions, such as the septum or outflow tract, may be hemodynamically superior. A
nonapical site may also need to be considered because of local myocardial problems, such as previous infero-apical infarction, or possibly to decrease the risk of perforation. In thin patients, a distal apical position may predispose towards costal muscle stimulation.

When any lead is initially advanced into the central circulation, it should first be seen to pass to the right of the vertebral column through the SVC. If the lead is introduced from the left side and passes to the left of the vertebral column, it is probably within a persistent left SVC, assuming venous (as opposed to arterial) access is confirmed (Fig. 5.16). If this occurs, there are two options. One is to continue to advance the lead or leads through the coronary sinus (CS) towards the right atrium. Although this approach has been used many times, lead placement can be difficult. This results because the lead will enter the right atrium from the CS at an angle that makes positioning in traditional atrial and ventricular sites challenging. The alternative is to abandon the procedure and then perform contrast venography from a peripheral intravenous line in the right arm to determine whether a right-sided SVC also exists. If it does, the implant site can be moved to the right and leads placed in a traditional manner. One should be more alert to the possibility of a persistent left SVC in a patient with associated congenital anomalies.
The lead can be initially passed through the introducer with a straight or a curved stylet in place. A curved stylet is helpful in introducing the lead across the tricuspid valve and into the pulmonary outflow tract. (A curved stylet is created by wetting the stylet and the gloved index finger and thumb and pulling the stylet through the apposed fingers while rotating the fingers to impart a curve to the wire. A curve can also be formed by pulling the distal end of the stylet between the index finger and needle-driver or other instrument.) Initially placing the lead in the outflow tract ensures that the lead is indeed in the RV and not in the CS or in an extracardiac vessel, such as the hepatic vein. The lead may cause premature ventricular contractions as it passes through the RV, but these are usually inconsequential. Once the lead tip is in the outflow tract, the curved guidewire should be replaced with a straight stylet. The straight stylet is not passed completely into the lead initially. The lead should be slowly withdrawn from the outflow tract, allowing the lead tip to fall toward the RV apex. The straight stylet should be simultaneously advanced as the lead is slowly withdrawn, allowing the straightened lead to fall toward the apex. Once the lead falls from the outflow tract and is directed toward the apex, the lead, with stylet in place, should be advanced toward the apex. This maneuver may be assisted by asking the patient to breathe deeply, causing the RV apex to descend with the diaphragm, at which point the lead can be advanced.

Another technique involves using the curved stylet to advance the lead into the RV. Instead of continuing to advance the lead all the way to the outflow tract, the stylet is withdrawn in the inlet portion of the RV itself. By withdrawing the curved stylet, the lead tends to “straighten” and can be advanced towards the apex. If the lead’s advance becomes hindered by an intracavitary structure such as the moderator band, then the curved stylet can be readvanced until the tip of the lead points upward and now can be advanced over the moderator band. By repeating the maneuver (withdrawing the stylet to straighten the leads and advancing when an obstruction is encountered), the lead tip can be “marched” towards the septal portion of the RV apex.

It is sometimes possible to position the lead without a curved stylet. Once the lead is in the right atrium, the straight stylet should be withdrawn about 5 cm and the lead moved inferiorly. It will often catch in the right atrium and be deflected toward the tricuspid valve (Fig. 5.17). If the lead passes the tricuspid valve, it will be in the inflow tract of the RV.
Intracardiac manipulation is basic to successful pacemaker implantation. Implantation of a dual-chamber pacemaker involves placement of an atrial and a ventricular lead. (A) Both leads may be introduced via the subclavian, cephalic, or external jugular vein. Initially, both are in the superior vena cava or the right atrial appendage. (B) Because ventricular stimulation is usually more important than atrial stimulation, the ventricular lead is positioned first. The atrial lead can be introduced immediately after or simultaneously with the ventricular lead or introduced after the ventricular lead is positioned. If the atrial lead is introduced before ventricular lead positioning, the atrial lead can be left in the upper inferior vena cava or low right atrium until time to position the lead. (C) Once the ventricular lead is past the tricuspid valve and in the midright ventricle, it should be advanced into the pulmonary artery so that it is clear that it has not entered the coronary sinus (panel F). (D) If the lead tip will not pass the tricuspid valve, entry by deflection from the lateral atrial wall may be successful. (E) Advancing the bowed lead in the right ventricle and then the guidewire within the bow can flip the lead tip into the right ventricular outflow tract. Note that during this entire maneuver, the atrial lead is ‘parked’ in the inferior vena cava. (F) As above, the lead is passed into the pulmonary artery. (G) Once there, the lead is slowly withdrawn so that it falls toward the right ventricular apex. (H) Once at the diaphragmatic surface of the ventricle, the lead is advanced into the apex. A deep breath angulates the apex downward and allows easier access. (I) When the ventricular lead has been positioned properly, the guidewire is allowed to remain withdrawn about 1 in from the tip to hold it in position while the atrial lead is being manipulated. (J) Because the two leads often adhere lightly to each other, when one lead is manipulated, the other should be held so that it is not inadvertently displaced. The atrial lead should be pulled into the low right atrium and the guidewire withdrawn about 7 cm; the J will form. (K) The atrial lead should then be pulled upward slowly. Entry into the base of the atrial appendage is recognized by straightening of the J with gentle traction on the lead. (L) Additional traction is stopped, and the lead is advanced into the tip of the atrial appendage. (By permission of Mayo Foundation.)
Another situation where a straight stylet alone is useful in getting a lead to the RV is with right-sided implantations and a torturous subclavian/SVC system. Here, because of the near right-angle turn from the right subclavian vein into the SVC, a curved stylet will tend to point the lead in the atrium and make it difficult to cross the tricuspid valve. In this situation, a straight stylet can be used to advance into the right atrium. Now, by pulling back the straight stylet, the lead is advanced until it curves in the right atrium itself. Now, the lead is rotated so that the heel of the curved lead begins to prolapse across the tricuspid valve. In a simultaneous maneuver, the straight stylet is then advanced while pulling back the lead. As the heel of the prolapsed lead now begins to be extended, the straight stylet reaches near the tip of the lead and the lead falls into the inflow portion of the RV, often close to the RV apex.

Alternatively, with the straight stylet withdrawn approximately 5 cm, the lead tip can be projected against the lateral atrial wall and the curved portion of the lead backed into the tricuspid valve.

These maneuvers are readily visible fluoroscopically and, if seen, ensure that the lead has traversed the RV. The lead occasionally enters the CS from the right atrium. A lead within the CS may appear to be within the RV, but the passage is not superiorly but rather more laterally toward the left cardiac border. Should the lead begin to curl about the left cardiac border, it is likely to be in the CS. In addition, no ventricular ectopy occurs if the lead is in the CS. Attempts at entering the pulmonary artery will be unsuccessful, but the most important clue is in the lateral fluoroscopic view. The outflow tract of the RV is an anterior structure, and a lead in it is seen in the retrosternal position. The CS is on the posterior wall of the heart, and a lead within it is visualized on the posterior cardiac border.

When the ventricular lead is positioned in the RV apex, radiographically, the end of the ventricular lead appears on the posteroanterior projection to be between the left border of the vertebral column and the cardiac apex. The position of the heart, vertical or more horizontal, largely determines the position of the lead in relation to the cardiac apex and varies among patients. The lateral view is necessary to distinguish between apical positions in which the lead tip is anterior and caudally directed, is posteriorly directed in the RV, and is on the posterior surface of the heart, i.e., within the CS. Fluoroscopically, in the right anterior oblique (RAO) view, the fat pad highlights the atrioventricular (AV) valve plane, and if the lead is in the CS it tracks along this plane, unless it falls into a ventricular cardiac vein. In contrast, a lead in the RV will have a course orthogonal to this radiographic view and will be seen going towards the apex. In the left anterior oblique (LAO) view, a lead in the RV is coming towards the image intensifier, whereas one in the CS has a distinguishing leftward trajectory. On the posteroanterior radiographic view, the ventricular lead should have a gentle curve along the lateral wall of the right atrium and cross the tricuspid valve to the ventricular apex (Fig. 5.18). Additionally, the LAO projection will help distinguish a septal from free wall (risk of perforation) location of the lead tip.

If an atrial lead is not to be implanted, the guidewire can be removed from the ventricular lead and the lead can be checked for stability by deep breathing or coughing and by assessment of pacing and sensing thresholds. In addition, diaphragmatic stimulation should be assessed. Our technique is to pace at 10 V, the maximum voltage available on the pacing system analyzer used. During 10-V pacing, the left hemidiaphragm is assessed fluoroscopically for detection of pacing-induced excursions of the diaphragm. If an atrial lead is being tested, the right hemidiaphragm is assessed fluoroscopically. Alternatively, a hand can be placed over the appropriate hemidiaphragm to feel for diaphragmatic stimulation. If diaphragmatic stimulation occurs, the lead should be repositioned. Intercostal stimulation should also be looked for during high-output pacing.

As previously noted, it may be desirable to position the lead somewhere other than in the RV apex. The hemodynamic superiority of one position over the other has not yet been firmly established. However, so long as pacing and sensing thresholds are acceptable and the lead is secure, it can be positioned anywhere within the right ventricle.

Because of the difficulty in comparing different ventricular pacing sites, nomenclature is necessary to allow uniform description of proposed alternative RV pacing sites (other than apical pacing). One approach would be to define the sites as high septal, low septal, high free wall and low free wall (Fig. 5.19A). Radiographic representations of high and low septal (Fig. 19B), and high and low free wall (Fig. 19C) are also shown.

Special circumstances may lead to innovative lead positioning. One example would be the patient with a congenital anomaly or prosthetic tricuspid valve.
that prevents transvenous access to the nonsystemic ventricle. Although CS lead positioning may permit transvenous pacing in a patient in whom the right ventricle is not accessible, in some patients, especially those with a congenital anomaly, the CS may not be accessible. An approach that we have occasionally used for such a patient, in collaboration with our cardiovascular surgeons, is transmyocardial lead placement (Fig. 5.20).

The ventricular lead of an ICD system, whether single- or dual-chamber, is placed in exactly the same manner as described above. However, alterations in technique may be required if defibrillation thresholds are unacceptable. Historically we would routinely place the lead as apically as possible in most patients. If the patient requires bradycardia support, as previously noted, there may be adverse hemodynamic consequences to RV apical pacing. The RV lead can be placed in any RV position where the lead is stable and pacing, sensing and defibrillation thresholds meet expectations. Often, use of a low apical septal location results in acceptable function. If the defibrillation thresholds are unacceptable, the lead should be repositioned to an alternative RV site where thresholds are again acceptable. Acceptable thresholds can be obtained with a single RV lead in > 95% of patients.\(^{15}\)

If adequate defibrillation thresholds cannot be achieved despite repositioning of the RV lead, retesting

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**Fig. 5.18** Posteroanterior (A) and lateral (B) chest radiographs demonstrating gentle curve of the ventricular lead as it passes across the tricuspid lead.

**Fig. 5.19** (A) Schematic drawing of low and high septal and low and high free wall positions for right ventricular outflow tract lead positioning. (B) High and low septal lead positions in anteroposterior (AP) and left anterior oblique (LAO) projections. (C) High and low free wall positions in AP and LAO projections.\(^{14}\) (Reproduced with permission from Blackwell Publishing.) (Continued.)
with reversal of polarity is often assessed, although its benefit has been questioned, particularly if the RV coil is the anode (positive electrode). If a single-coil lead is used, a SVC coil can be added either as a separate lead or more commonly by exchanging the single-coil lead for a dual-coil lead (Fig. 5.21). This results in acceptable de-
fibrillation thresholds for most patients. In our practice, we preferentially use dual-coil leads, and infrequently use single-coil leads. If defibrillation safety margins remain unacceptable, remaining options include using a pulse generator with programmable waveforms (St Jude Medical), or the addition of another venous defibrillation electrode (such as the CS), or use of a subcutaneous array.17

A subcutaneous lead is infrequently needed to achieve an adequate defibrillation threshold. Two types of subcutaneous leads are currently available. A subcutaneous array consists of three electrically common multifilar coils that form one electrode (Fig. 5.22). The coils come together in an insulated cable that is connected to a terminal pin that can be connected to the connector block. The “array” provides a greater surface area over the lateral chest to facilitate defibrillation (Fig. 5.23). Our approach for the placement of the “array” is to make an incision near the midaxillary line at the level of the nipple. Dissection is carried to the level of the muscle. With a

Fig. 5.20 Posteroanterior (A) and lateral (B) chest radiograph from a patient with transposition of the great vessels and prosthetic valves in both the tricuspid and mitral positions and complete atrioventricular block with pacemaker dependency. An active fixation steroid-eluting transvenous lead was placed via a transmyocardial approach. (C) Photograph obtained in the operating room at the time the lead was placed.
trochar that is supplied with the array, three tunnels are formed, one at a time, with a peel-away introducer over the trochar. The trochar is removed, and one of the three coils is placed within the sheath, which is carefully peeled away. Once all three are placed, each coil is secured to the underlying tissue with the sleeve provided, and the “yoke,” or point where the three coils join, is secured to the underlying tissue as well. The insulated cable is then passed into the inferior portion of the previously formed prepectoral pocket and connected to the ICD.

A subcutaneous lead is also available as a single element or finger. This variety, also placed with a trochar as previously described, can sometimes be placed via the inferior portion of the pocket, obviating the need for a separate incision (Fig. 5.24).

**Coronary sinus lead placement**

CS lead placement for left ventricular stimulation is commonly used in patients that meet criteria for CRT. The hemodynamic implications of biventricular stimulation or cardiac resynchronization are discussed in detail in Chapter 2.

Familiarity with placing left ventricular leads as part of biventricular device implantation is important, as up to 50% of new ICD implants are CRT systems at some US centers. It is imperative that the implanting physician has a thorough knowledge of coronary venous anatomy (Figs. 5.25, 5.26 and 5.27).

Left ventricular leads may be deployed by either a right- or left-sided implant. We generally place CS leads from the left side. If placing a CS lead from the right, there are specific differences in sheath and lead manipulation, discussed below.

We avoid placing multiple leads through a single venepuncture site because of the propensity for lead dislodgment while manipulating the non-left ventricular leads, for an increase in backbleeding, and...
for difficulty with sheath or left ventricular lead manipulation.

The left ventricular lead may be placed before or after the right ventricular and right atrial leads have been placed. The advantages of placing the left ventricular lead first include greater ease in maneuvering the guiding sheath and lead, elimination of the risk of partial occlusion of the orifice of the CS with the RV lead, and prevention of inadvertent RV lead manipulation in the CS, resulting in spasm or dissection. The major advantages of placing the RV lead first include a fixed reference to the apex (thus delineating the interventricular septum) and tricuspid annulus and ventricular pacing support if the patient has extensive conduction system disease. We prefer to place the left ventricular lead first. In difficult cases, particularly with asymmetric dilation of the left ventricle or clockwise rotation of the heart (or both), the RV lead is placed at the apex first to provide optimal orientation.

The RAO projection is used to define the tricuspid valve annulus, and the LAO projection is used to de-
Fig. 5.25 Coronary sinus (CS) and ventricular vein anatomy ideally are visualized in two orthogonal fluoroscopic views. In the right anterior oblique (RAO) projection, the CS is viewed end-on. The ostium of the CS is just superior to the usually visualized translucency of the posterior fat pad. The body of the CS and great cardiac vein proceed end-on, separating the ventricles and atria. In this projection, the ventricular veins then proceed towards the sternum (towards the right, as depicted in the left image). In this view, right-sided and left-sided positions are not readily distinguished. In the left anterior oblique (LAO) projection, the ostium of CS is in the plane of the interatrial septum. The vein proceeds posteriorly from right to left, wrapping around the lateral wall of the heart. In this view, atrial and ventricular veins cannot be distinguished. The LAO projection is ideal for distinguishing lateral and septal locations of veins and contained leads. Thus, the LAO view defines left vs. right position, and the RAO view defines atrial vs. ventricular position. (From Chapter 4, Biventricular Device Implantation. In: Hayes DL, Wang PJ, Sackner-Bernstein J, Asirvatham SJ [eds] Resynchronisation and Defibrillation for Heart Failure: A Practical Approach. Oxford, UK: Blackwell Futura, 2004.)

Fig. 5.26 The lateral left ventricle (LV) is drained by branches of the posterolateral vein (PLV) in most patients. It should be noted that lateral branches of the anterior intraventricular vein and lateral branches of the middle cardiac vein also drain this region. CS, coronary sinus; LA, left atrium. (From Chapter 4, Biventricular Device Implantation. In: Hayes DL, Wang PJ, Sackner-Bernstein J, Asirvatham SJ [eds] Resynchronisation and Defibrillation for Heart Failure: A Practical Approach. Oxford, UK: Blackwell Futura, 2004.)
fine the interventricular septum and apex. In patients with dilated cardiac chambers, the “usual” amount of RAO and LAO to achieve this orientation may be insufficient. The characteristic change in the contour of the RV lead at the tricuspid annulus and its placement at the apex can help to line up these views in a given patient.

**Coronary sinus cannulation**

Once venous access has been established, the CS must be cannulated with a guiding sheath. The ventricular lead is deployed through the sheath, after which the sheath is removed. Attention to detail and experience in troubleshooting problems that arise with cannulating the CS will decrease procedure time and minimize CS-related complications.19

**The guiding sheath**

Several varieties of guiding sheaths are available, but there are major differences in shape and method of sheath removal. Straight sheaths with no preformed curves typically require a deflectable catheter for initial cannulation of the CS. Preformed curves are available in several varieties; no one curve is ideally suited for all hearts. Generally, gentle curves are preferred to give flexibility if subselection of a coronary vein is required or multiple catheter exchanges are needed to cannulate the CS. The ideal curve allows the body of the catheter to rest against the free wall of the right atrium and yet engage the CS ostium. This type of stability allows torque application to probe the ostial septum anterior to the Eustachian ridge in both the anteroposterior and superoinferior directions.

**Use of angiographic wires vs. deflectable electrophysiological catheters to engage the coronary sinus**

Angiographic wires that are soft-tipped and torqueable are well suited for use with preshaped guiding sheaths.20 To engage the CS, the wire is advanced preferably as far as the natural curve of the CS, great cardiac vein, anterior vein and interventricular vein will allow. The guiding sheath is then advanced along the wire while the wire is pulled back. The sheath should not be advanced when the wire has not freely advanced into the axis of the coronary veins. This is to avoid CS
dissection. If the wire has not advanced sufficiently to allow it to be pulled back a substantial amount while the sheath is being advanced, then repeatedly pulling back the wire and readvancing the sheath in small increments will allow the CS to be safely engaged with the guiding sheath (Fig. 5.28).

Deflectable electrophysiological catheters can be used with either straight or preformed curved guiding sheaths. We often use deflectable catheters capable of recording electrograms and whose deflection mechanism allows bidirectional changes. After the CS has been cannulated, pulling back on the catheter slightly while advancing the sheath will allow the sheath to be deployed into the CS (Figs 5.29, 5.30 and 5.31).

A third option entails using preformed curved sheaths designed to facilitate CS access, in conjunction with gentle puffs of contrast to confirm cannulation. The sheath may have an obturator within it to provide mechanical support, or a smaller sheath with a 50–90° bend at the distal end. Most commonly, such a sheath or sheath-within-a-sheath system is positioned in the RV at the septum. Gentle counterclockwise torque and slight retraction of the guiding sheath results in the distal tip approaching the ostium of the CS. Gentle puffs of contrast outline characteristic endocardial contours until the CS itself is seen. The advantage of this technique is that with current preformed guide catheters, CS access is rapid and reproducible. Disadvantages include the need for extra contrast, the risk of injury/dissection if contrast is injected too forcefully, and the fact that the large curves used to access the CS often are better suited for subsequently cannulating distal branches, making access to the middle cardiac vein more difficult.

In most cases, any technique—angiographic wires, contrast puffs or deflectable electrophysiological catheters—is successful. The advantage of angiographic wires is that their small size and soft tip allow repeated advancements into the appropriate radiographic planes; also, they engage and advance through tortuous, small-diameter, or partially dissected coronary veins. If the Eustachian ridge is prominent, the guiding sheath needs to be placed ventricularly to the ridge to allow the wire to engage the CS. This can be a difficult maneuver. Deflectable electrophysiological catheters have the advantage of recording electrograms, which should show a balanced atrial and ventricular signal to identify annular locations in the patient. Furthermore, catheters with bidirectional curves can be used to negotiate sharp bends over prominent Eustachian ridges and around near circumferential thebesian valves (Fig. 5.32). In choosing between techniques, the operator should make the decision on the basis of his or her experience. For example, an interventionalist may prefer trying various wires or contrast approaches, whereas an electrophysiologist is likely to be more comfortable with deflectable catheters.

**Fig. 5.28** Cannulating the coronary sinus with a guidewire. If a curved or angled sheath is employed, guidewire can be used to engage the coronary sinus and its tributaries. With the sheath rotated counterclockwise so that it points septally in the left anterior oblique projection, the wire is advanced gently, and it is seen to take the typical course of the vein in both the right and left anterior oblique projections. (From Chapter 4, Biventricular Device Implantation. In: Hayes DL, Wang PJ, Sackner-Bernstein J, Asirvatham SJ [eds] Resynchronisation and Defibrillation for Heart Failure: A Practical Approach. Oxford, UK: Blackwell Futura, 2004.)
Either straight or curved sheaths can be used with a deflectable catheter to engage the coronary sinus. The catheter is deflected just above the posterior fat pad in an end-on manner in the right anterior oblique projection and points septally and leftward in the left anterior oblique projection. Care should be taken that once the catheter has engaged the coronary sinus, the sheath should be advanced to the ostium of the coronary sinus with gentle pulling back of the catheter (see text). (From Chapter 4, Biventricular Device Implantation. In: Hayes DL, Wang PJ, Sackner-Bernstein J, Asirvatham SJ [eds] Resynchronisation and Defibrillation for Heart Failure: A Practical Approach. Oxford, UK: Blackwell Futura, 2004.)

Advancing the guiding sheath into the coronary sinus. After the sheath has been placed in the ostium of the coronary sinus, the deflectable catheter is advanced to the region of the desired vein. While pulling back on the catheter, advance the sheath and then advance the catheter again. Repeat this maneuver until the desired location in the venous system has been obtained. LAO, left anterior oblique; RAO, right anterior oblique. (From Chapter 4, Biventricular Device Implantation. In: Hayes DL, Wang PJ, Sackner-Bernstein J, Asirvatham SJ [eds] Resynchronisation and Defibrillation for Heart Failure: A Practical Approach. Oxford, UK: Blackwell Futura, 2004.)
Regardless, deflectable catheters are larger in diameter and may not allow cannulation in patients with coronary vein stenosis or spasm. It is imperative that minimal force be applied with slow and gentle movements, because CS dissection is more likely with stiff catheters, which should be avoided. This risk is minimized if the operator avoids advancing the sheath when the tip of the catheter is not free, avoids advancing into atrial coronary veins, and matches the French size of the catheter with the sheath.
Depending on operator preference and patient anatomy, one might choose ostial placement of the guiding sheath as opposed to subselection into a ventricular vein. The description of these techniques and advantages and disadvantages of each are beyond the scope of this text. However, multiple resources are available for in-depth description.22,23

**Coronary sinus venography**

After the guiding sheath has been placed at the ostium of the CS, coronary venography may be performed (Fig. 5.33). CS angiography can be performed effectively with end-hole balloon-tipped catheters (with injection of the contrast through the guiding sheath with or without a balloon on the sheath) or with deflectable electrophysiological catheters that allow the injection of contrast dye. The technique most commonly used is balloon occlusion angiography. Care should be taken to ensure that the tip of the balloon catheter is free; the catheter should be advanced approximately 1 cm beyond the guiding sheath tip. If further advancement is not possible, the catheter should be pulled back to a point where the tip is clearly free before the injection of contrast dye is contemplated. The balloon is then inflated and gently pulled back towards the guiding sheath. Complete deployment of the balloon aids visualization of the distal coronary venous tree (Fig. 5.34). The contrast agent is injected under cine fluoroscopy. The anatomy of the coronary venous tree is visualized in at least two orthogonal planes, usually the RAO and LAO projections. To obtain maximal anatomical information from balloon angiography, it is important that (i) complete occlusion is performed, (ii) injection and cine fluoroscopy are continued until more proximal veins are seen to fill through anastomoses, and (iii) fluoroscopy is continued after the balloon has been deflated, because the backwash of contrast dye often demonstrates the ostia of the middle cardiac vein, proximal posterolateral veins, and the small cardiac vein.

Performed in this way, contrast CS venography provides a map for further manipulation of the ventricular lead (Figs 5.35 and 5.36). However, CS angiography is not without limitations. First, if care is not taken to ensure free motion of the tip of the catheter before injection, CS dissection or perforation (or both) may

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Fig. 5.33 When performing coronary sinus angiography with balloon catheters, the distal coronary sinus, great cardiac vein, and ventricular veins are best visualized with complete inflation of the balloon and occlusion of the coronary sinus. More proximal branches and the middle cardiac vein can be visualized when collaterals reforming these veins are seen or with continuous imaging when the balloon is deflated and the ostia of the proximal veins are visualized with a backwash of contrast. (From Chapter 4, Biventricular Device Implantation. In: Hayes DL, Wang PJ, Sackner-Bernstein J, Asirvatham SJ [eds] Resynchronisation and Defibrillation for Heart Failure: A Practical Approach. Oxford, UK: Blackwell Futura, 2004.)
result. Second, in some patients, the contrast may adversely affect renal function or promote pulmonary edema. Third, the sheath may become dislodged during manipulation of the balloon catheter. Although CS angiography is helpful in some cases, effective lead deployment, especially with over-the-wire leads, can be accomplished without CS angiography. It is probably beneficial for an operator to use CS angiography for the first 20–30 implants to become familiar with coronary venous anatomy and to correlate this anatomy with fluoroscopic views and the “feel” of the lead engaging a particular vein. However, some experienced implanters prefer to attempt placing the ventricular lead without performing CS angiography initially; if unsuccessful, a venogram is performed.

**Technique for cannulating the coronary sinus**

The primary imaging modality used for cannulation of the CS is fluoroscopy. Any projection is potentially useful to effectively engage the ostium of the CS. However, because of the rotation of the heart in the chest cavity, the true orthogonal views along the cardiac axes are the

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**Fig. 5.34** At times during coronary sinus angiography, visualization is best with a graded pullback technique. Initially, the balloon is placed distally and the distal vessels visualized. The balloon is then deflated more proximally to visualize mid-level branches. Either during the backwash phase or with gradual deflation of the balloon while pulling back the catheter, proximal ventricular as well as atrial branches can be visualized. This technique can be useful with large coronary veins. (From Chapter 4, Biventricular Device Implantation. In: Hayes DL, Wang PJ, Sackner-Bernstein J, Asirvatham SJ [eds] Resynchronisation and Defibrillation for Heart Failure: A Practical Approach. Oxford, UK: Blackwell Futura, 2004.)
RAO and LAO projections (Fig. 5.37). The LAO projection is along the plane of the interventricular and interatrial septa. Therefore, the left-sided and right-sided cardiac structures are readily distinguished in this view, as are septal and free-wall positions. In the standard presentation, the right atrium and ventricle are seen to the left of the screen, and vice versa. Note that in the LAO projection ventricular and atrial structures cannot be distinguished. In most hearts, an LAO projection angle of approximately 30° will align along the interventricular septum. However, implantation of a biventricular device is often required for patients with grossly abnormal hearts, requiring a deviation from the standard approach. To define the septum in an asymmetrically enlarged heart, a catheter can be placed at the location where the His electrogram is recorded, or the RV pacing lead can be placed in the RV apex. After the septum has been defined by one of these techniques, the LAO angle
can be adjusted so that this septal catheter (His bundle or RV apex) is viewed end-on. In particularly difficult cases, echocardiography can be used to define the location of the interventricular septum. It is not unusual that an LAO angle > 80° is required to achieve the usual septal viewing plane. The natural viewing angle of the RAO projection is through the plane of the AV septum. With this viewing plane, it is easy to differentiate ventricular (i.e., toward the sternum) from atrial (i.e., toward the vertebral column) locations. Either of these viewing planes can easily distinguish superior and inferior positions.

To locate the CS fluoroscopically, the following steps are performed: (i) in the RAO projection (approximately 30°), the epicardial posteroseptal fat pad is visualized. This is usually seen near the angle of the right hemidiaphragm and the cardiac silhouette. If necessary, higher intensity fluoroscopy can be used briefly to visualize the structure. Often, the annulus can be visualized as a relatively radiolucent area. Occasionally, stents and coronary arterial calcifications define the annulus, as does a mechanoprosthetic cardiac AV valve; (ii) the angiographic wire, the guide catheter, or deflectable electrophysiological catheter is moved to a location just posterior and cephalad to the epicardial posteroseptal fat pad; (iii) mild counterclockwise torque is applied to the catheter, guide or sheath with wire, and the wire or catheter is gently advanced; (iv) now, the LAO viewing angle is used to ensure that the catheter or wire is advancing to the left side; (v) after the catheter or wire has advanced for approximately 1–2 cm into the CS (i.e., leftward in the LAO projection and along the AV groove in the RAO projection), the sheath is advanced gently to engage the coronary sinus (CS) ostium. (From Chapter 4, Biventricular Device Implantation. In: Hayes DL, Wang PJ, Sackner-Bernstein J, Asirvatham SJ [eds] Resynchronisation and Defibrillation for Heart Failure: A Practical Approach. Oxford, UK: Blackwell Futura, 2004.)
Complications associated with coronary sinus cannulation
Dissection of the CS may occur when engaging the CS or attempting to advance the sheath into the CS.\textsuperscript{24} Angiography from the CS will show staining of the CS musculature, the CS wall, or occasionally extensive staining of the entire coronary venous tree. Mild localized dissections of the CS are probably inconsequential, and the procedure can be performed in the usual fashion. More extensive dissections may result in closure of the coronary venous system, precluding placement of the lead (Figs 5.38 and 5.39).\textsuperscript{25} The natural history of these occlusions is not known. When a CS dissection is diagnosed, the pericardium should be carefully examined fluoroscopically during injection of dye.\textsuperscript{25} If no perforation is seen, certain techniques can be used to continue with the procedure.\textsuperscript{22,23}

True CS perforations are recognized by the extravasation of contrast dye injected in the CS. Also, the clinical or echocardiographic features of pericardial effusion are usually seen. Tamponade is unusual because of the low-flow coronary venous system. However, if a sheath is advanced inadvertently through a perforated segment, life-threatening tamponade may occur.

Left ventricular lead deployment
After the CS has been cannulated, the guiding sheath is used to advance the left ventricular pacing lead through the sinus into a ventricular vein. Considerations in choosing the left ventricular pacing site include lead stability and obtaining sufficient separation from the RV lead. Early acute intraoperative epicardial lead data suggest that maximal benefit is achieved with a mid-lateral positioning of the lead.\textsuperscript{26} Although a lateral wall position may be ideal for many patients, other issues that need to be considered include: viability of the tissue where the lead is being placed; pattern of mechanical dyssynchrony; and pattern of electrical dysynchrony and the location of the left phrenic nerve.

General considerations
Both stylet-driven and over-the-wire leads are available for clinical use. Operators are usually familiar with stylet-driven leads. The stylet can be preformed to various curvatures. Also, retracting and inserting the stylet at the tip of the lead can change the angulation at the tip so the lead can be maneuvered into a ventricular venous branch (Fig. 5.40). In many cases, over-the-wire leads are preferable because it is easier to negotiate more distal locations in the venous sys-

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*Fig. 5.38* Coronary sinus angiogram in a patient with highly tortuous distal great cardiac vein and ventricular veins. Forceful injection to visualize adequately the distal veins resulted in a pericardial blush (staining), which can result from coronary sinus dissection, pericardial infiltration, or contrast within the thebesian vein network. Cine fluoroscopy will show characteristic annular movement with coronary sinus dissection, but movement with the cardiac silhouette in pericardial and intramyocardial staining. (From Chapter 4, Biventricular Device Implantation. In: Hayes DL, Wang PJ, Sackner-Bernstein J, Asirvatham SJ [eds] Resynchronisation and Defibrillation for Heart Failure: A Practical Approach. Oxford, UK: Blackwell Futura, 2004.)
tem. If the over-the-wire system is used, the wire is first inserted through the sheath and placed in the vein of interest, and then the lead is loaded on the over-the-wire system and advanced into the vein (Fig. 5.41). Typically, small French-size leads are preferred so that the lead tip can be left in a sub-branch or tributary of a major ventricular vein, aiding stability. In some instances, however, the CS and ventricular veins may be grossly dilated and the small French-size lead cannot make adequate contact with the myocardial surface. In this case, larger stylet-driven leads can be used and placed more proximally in the coronary venous system to make better contact. Alternatively, leads with marked preformed bend may be used; withdrawal of the guidewire results in the development of complex tertiary geometries designed to stabilize the lead in a larger vessel. Similarly, larger stylet-driven leads may be useful when pacing thresholds are poor in the mid and distal portions of the ventricle or when a lead needs to be placed in the proximal venous system, where venous diameter is large and smaller leads may not make adequate contact with the myocardium (Fig. 5.41).
When the left ventricular lead is advanced, gentle pressure should cause mild buckling in the CS and proximal ventricular vein so that the lead conforms to the curvature of the venous system. Another technique to enhance stability is to advance the lead through a main ventricular venous branch, e.g., the posterolateral vein, and use the wire to subselect a tributary and advance this to a yet secondary tributary that is in parallel with the primary vein. This U-shaped placement is usually highly resistant to dislodgment during removal of the sheath. Care should be taken to prevent excess slack or buckling proximal to the ostium of the CS. After the sheath has been removed, the tricuspid annular region should be inspected carefully. If the lead prolapses beyond the tricuspid valve into the RV, the slack should be removed. Excess slack within a large dilated CS may cause coiling or looping of the lead in the CS. In our experience at Mayo Clinic, this has not affected lead stability and we have elected to leave the loop within the CS. Occasionally, excessive slack causes prolapse of the proximal portion of the lead into the inferior vena cava. It is probably best to pull back on the lead to minimize the slack (Fig. 5.42).

If the coronary venous system is very large (dilated and nearly variceal), obtaining contact and adequate...
stability is a problem with even larger leads. To overcome this challenge, two techniques may be used: (i) the over-the-wire lead system can be advanced through the large venous system from, for example, the posterolateral vein all the way to the apex and then advanced through either the anterior interventricular vein or middle cardiac vein to a more proximal location, where the lead can be placed in a smaller venous tributary; (ii) the left ventricular lead is purposely curled on itself and advanced as a loop into the dilated venous system. To do this, the wire is first advanced to engage a venous branch. Thus engaged, the lead is continuously pushed until the body of the lead begins to prolapse as a loop into the great cardiac vein. The wire is then retracted and the lead is advanced as a loop into another venous branch. Advancing the lead with this loop sometimes affords better myocardial contact and adequate thresholds when previous maneuvers were unsuccessful. This same maneuver can also be helpful in avoiding diaphragmatic stimulation when a proximal posterolateral or middle cardiac vein is used. When advanced as a loop, the tip of the lead can be manipulated to be oriented more toward the myocardium than the diaphragm.

Thresholds should be checked before and after the sheath has been removed. High output (at the highest output and pulse width setting) should be performed during both inspiration and expiration to check for phrenic or diaphragmatic stimulation. In our experience, if diaphragmatic stimulation occurs, it is preferable to obtain access in a different venous branch than to attempt repositioning the lead in the same vein, because diaphragmatic stimulation may occur subacutely with movement or change in respiration.

Deployment in the middle cardiac vein will be necessary in some patients, and the operator should be familiar with this special technique.22,23

Sheath removal
After the lead has been placed satisfactorily and preliminary thresholds have been checked to ensure local ventricular capture and to exclude extracardiac stimulation, the guiding sheath needs to be removed. Several methods are available for removing the sheath, including peel-away type sheaths and cutting-away systems. Also, with certain combinations of lead and sheath, the sheath can be pulled over the lead, although this type of system is rarely used. Regardless of the system used, certain principles need to be followed. A stylet should be reintroduced into the lead before the sheath is removed. Depending on the lead system, the stylet may be introduced all the way to the tip of the lead or into the main body of the CS. Despite these variations, the stylet should be of medium stiffness (in most cases) and placed at least 1 cm into the CS and preferably in the great cardiac vein. If the stylet is placed too close to the CS or in the right atrium, pulling back on the sheath may cause excessive inferior force and dislodge the lead. Whether the sheath is cut away, peeled away, or removed over the lead, the lead should be held firmly in place while the sheath is removed. In the case of the cut-away system, the cutting blade should be secured to the lead and the blade and lead held firmly onto the patient with one hand. The sheath should be pulled back against the blade and care taken not to change the existing rotational torque on the sheath. In other words, the sheath should be pulled back in the angle in which it lays and not be maneuvered to suit the operator. This is to avoid dislodging the lead as the sheath is cut back. The movement should be smooth and fluid, and the hand that stabilizes the lead should not be moved. Similarly, with peel-away or over-the-lead removal, the hand that stabilizes the lead should not move, and the tendency to push the lead further into the body should be resisted. After the sheath has been removed, the lead is secured to its sleeve and the sleeve to the underlying muscle. Most operators remove the stylet for suturing the lead and introduce the stylet up to the junction of the SVC and right atrium before manipulating other leads. Fluoroscopy should be performed after the stylet has been removed and the lead has been secured to ensure there is no excess slack, specifically slack that causes the body of the lead to prolapse into the RV.

Multiple approaches can be used to cannulate the CS and deploy a left ventricular lead. The choice of a particular approach depends on the training and background of the operator and the resources available. Also, it is desirable to know several solutions to any problem that may develop. No single sheath, curvature, deflectable catheter, wire, or technique is ideally suited for all patients. Although most operators become familiar with a particular set of techniques, they should be willing to try another technique that may be helpful in a difficult case.

Securing permanent leads
If pacing and sensing thresholds are satisfactory and there is no diaphragmatic stimulation measured with pacing at 10 V, the silicone rubber sleeve provided on the
lead is positioned over the lead at the point of entry into the vein. Synthetic nonabsorbable ligature is used to fix the sleeve to the lead and to the muscle or the vein itself. It is essential to use the sleeve and not affix the lead directly to the adjacent tissue (Fig. 5.43). Ligatures applied directly to the lead may damage the insulation and act as a fulcrum, with eventual lead fracture at the ligature site.

**Dual-chamber pulse generator implantation**

The introducer technique can be used to place two leads. Three variations of the technique can be selected. Two venepunctures can be made, one for each catheter to be inserted, the ventricular lead generally being placed first (Fig. 5.44). This technique reduces the potential for displacement of one lead while the other is being positioned, but requires two separate venepunctures. In the second variation, one venepuncture is made and one lead is introduced, and the guidewire is reintroduced or retained before the sheath is peeled away (Fig. 5.45A);
after positioning the first lead, an introducer is placed over the retained guidewire to accommodate the second lead (Fig. 5.45B). In an uncommonly used third variation, two leads (one for atrial and one for ventricular placement) can be advanced through the same introducer sheath into the right side of the heart (Fig. 5.46). (The size of the introducer required to introduce two leads depends on the additive size of the two leads.) Our preference is to position the ventricular lead and then pass the atrial lead. Alternatively, both leads can be passed into the right heart and the atrial lead held in a stable position in the right atrium while the ventricular lead is positioned in the RV apex. After stable ventricular placement is achieved, the atrial lead is positioned.

Although the atrial lead has most commonly been positioned in the right atrial appendage, satisfactory pacing can be achieved from multiple positions within the right atrium. In patients with previous cardiac surgery in whom the appendage has been cannulated or amputated, finding a stable position in the vicinity of the atrial appendage may be difficult and not always possible.

Fig. 5.45 Two leads can be placed without a second subclavian puncture and without simultaneously passing the leads. (A) As the dilator and guidewire are removed and the initial pacing lead is passed into the right heart, the guidewire is reinserted through the peel-away introducer alongside the pacing lead. The introducer is then peeled away, and the pacing lead and guidewire are left in place. (B) A second introducer is then passed over the reintroduced guidewire, and the second lead is placed. (From Holmes DR, Hayes DL, Furman S. Permanent pacemaker implantation. In: Furman S, Hayes DL, Holmes DR Jr, eds. A practice of cardiac pacing, 2nd edn. Mount Kisco, NY: Futura Publishing Co., 1989:239–87. By permission of Mayo Foundation.)
Technique varies, depending on whether a preformed atrial J lead or a non-preformed lead, i.e., a standard straight lead that can be used in either the atrium or the ventricle, is used. At this time, standard leads (non-preformed J) are much more commonly used than preformed J leads.

If a preformed J lead is chosen, a straight stylet is placed in the lead to straighten it, and the lead is passed into the middle to low right atrium (Fig. 5.47A). The straight stylet is withdrawn approximately 10 cm, and the lead assumes the J shape (Fig. 5.47B). The lead is gradually withdrawn in an effort to secure the lead tip against the endocardial surface (Fig. 5.47C). If the lead tip is securely against the atrial wall when the J begins to straighten, the lead should again be advanced slowly to allow the appropriate J to occur. If the lead has active fixation, the fixation mechanism should then be secured. Sensing and pacing thresholds should be checked. If they are adequate, the lead should be secured with the sleeve provided, as previously described.

Entry into the atrial appendage is indicated by a rhythmic to and fro medial and lateral motion of the J portion of the lead. The posteroanterior fluoroscopic projection may show that the lead is medial or lateral, and a lateral projection shows the lead to be anterior at approximately the same level as a lead in the RV apex.

If the atrial lead is being placed as part of a dual-chamber implant, the ventricular lead should be carefully observed so that it is not inadvertently displaced.

If a straight active fixation lead is used, a J curved stylet is needed to position the lead. J curved stylets are usually provided with the lead. Whether the lead is being positioned in the right atrial appendage or other right atrial site, a stylet with some degree of J shape will usually be required. The stylet is introduced into the atrial lead in the low right atrium, and the lead is pulled into the right atrial appendage. The active fixation lead is fixed in place, and the J guidewire is gently withdrawn to avoid displacing the lead from the point of attachment. Again, sensing and pacing thresholds should be
CHAPTER 5  Implantation and Extraction Techniques

179

checked. If they are adequate, the lead should be secured with the sleeve provided, as previously described.

Regardless of whether a preformed J or a straight lead is implanted in the atrium, with implantation in the right atrial appendage the J portion of the lead is slightly medial on the posteroanterior projection and anterior on the lateral projection. Optimally, the limits of the J should be no greater than approximately 80° apart. Redundancy proximal to the J within the atrium or SVC should not be seen.

As noted above, locations other than the right atrial appendage may be used for atrial lead positioning, and their use is increasing. There are some advocates of routine placement of the right atrial lead on the atrial septum. The right atrium can be explored to find optimal positioning for lead placement. With active fixation leads, the lead can be placed anywhere the lead is stable and good thresholds are obtained (Fig. 5.48).

Atrial leads are occasionally positioned in the CS and adjacent to the CS ostium (os) to prevent recurrent atrial fibrillation and flutter.\textsuperscript{27} (Atrial septal lead positioning\textsuperscript{26,27} or Bachmann’s bundle positioning\textsuperscript{30} is also favored by some for the prevention of paroxysmal atrial fibrillation or flutter.) The hemodynamic implications of dual-site atrial pacing are discussed in detail in Chapter 2.

The technical details of introduction of a CS lead have previously been described. Obviously, for atrial pacing, the CS lead needs to be positioned in a tributary of the CS that will afford adequate and stable atrial pacing and sensing. The ideal position is probably in the vein of Marshall (Fig. 5.49). Once again, because of

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**Fig. 5.47** Placement of a preformed atrial J lead. (A) The lead is at the middle of the right atrium with a straight stylet in place. (B) The straight stylet is removed; removal allows the catheter to assume the preformed J configuration. (C) The entire lead is pulled back, and the tip of the electrode is allowed to enter the right atrial appendage. The lead has a characteristic to-and-fro motion when positioned in the right atrial appendage. (From Holmes DR, Hayes DL, Furman S. Permanent pacemaker implantation. In: Furman S, Hayes DL, Holmes DR Jr, eds. *A practice of cardiac pacing*, 2nd edn. Mount Kisco, NY: Futura Publishing Co., 1989:239–87. By permission of Mayo Foundation.)
the individual variability in coronary venous anatomy, not all patients have a true vein of Marshall.

To position an active fixation atrial lead near the CS os, the lead is passed into the CS and then gently withdrawn and secured after exiting the CS. (A straight lead is required; a preformed J should not be used.) Fluoroscopic imaging in a RAO position may allow one to better visualize the posterior and septal position desirable (Fig. 5.50). The LAO fluoroscopic view is helpful subsequently to verify the location of the lead.

**Measurement of pacing and sensing thresholds**

Knowledge and measurement of pacing and sensing thresholds are integral parts in the placement of a permanent pacemaker, ICD or CRT system. The equipment used and measurements made vary from laboratory to laboratory. In most institutions, pacing system analyzers available from the pulse generator manufacturers are used. Most centers use an analyzer from a single manufacturer. (Although of historical concern, use of a mismatched pacing system analyzer and pulse generator does not result in any clinical problem.) Measurements necessary during device implantation are listed in Table 5.1.

**Determination of pacing threshold**

The pacing threshold is the minimal electrical stimulus required to produce consistent cardiac depolarization. It should be measured with the same electrode configuration (unipolar or bipolar) as the lead and pulse generator that are to be used. During pacing, the output of the pacing system analyzer is gradually decreased from 5V at 0.5 ms pulse width to the point at which loss of capture is documented. The pacing rate selected during this measurement is important. The rate should be just fast enough (approximately 10 ppm faster) to override the intrinsic rhythm. In some patients, pacing during measurement of thresholds suppresses intrinsic rhythm and results in lack of a stable ventricular escape focus, or even asystole, when pacing is discontinued. The lower the stimulation threshold, the better. Acceptable acute thresholds are generally considered to be < 1V for both ventricular and atrial leads at 0.5 ms pulse duration.

If active fixation leads are used, thresholds checked immediately after deployment of the fixation mecha-
nism may not reflect true thresholds. If the lead position looks good and the initial thresholds are high, it is worthwhile waiting for a short time, for example, 1 min, and repeating the measurements to see whether the thresholds are now acceptable. If the repeat threshold is lower but still not at the desirable level, it may be worthwhile waiting another minute or so and rechecking to see if the trend continues toward a lower threshold, as it often will.

The impedance of the pacing electrode is also measured. Measurement of voltage and current allows calculation of the lead resistance, which varies greatly depend-
ing on the lead used. A range from 300 to 1500 Ω may be seen, depending on lead type. Impedances should always be measured under standardized conditions of output and pulse duration. The finding of unsuspected low impedance raises the possibility of insulation failure in the lead and that of high impedance the possibility of a poor connection in the connector block, or lead fracture.

**Determinition of sensing threshold**
Measurement of sensing thresholds is equally important. Adequate sensing thresholds are essential to avoid the problem of undersensing or oversensing after implantation. The pulse generator senses intracardiac events, not the events seen on the surface electrocardiogram (Fig. 5.51). The intrinsic deflection is that component of the intracardiac electrogram that is sensed. It is the amplitude of this intracardiac signal in the chamber to be paced that is measured. The result is expressed as a voltage. The ventricular electrogram sensed for adequate long-term sensing should be > 4 mV. More commonly, the ventricular signal is 6–20 mV, a range that provides excellent sensing. Programmable options for ventricular sensing in permanent pacemakers may exist at ≤ 1 mV. However, the goal for a measured ventricular signal should be ≥ 5 mV during normal rhythm. This is particularly true for implantable defibrillators, to ensure that the small-amplitude signals during ventricular fibrillation are appropriately sensed.

For atrial sensing, a signal of at least 2 mV is desirable. However, with current pacemakers that offer programmable options for atrial sensing as low as 0.18 mV and with ICDs that incorporate autosensing to vary sensitivity values, lower atrial sensing thresholds can at times be accepted. Still, the goal should be a measured P wave of ≥ 2 mV. With an atrial lead placed in the appendage, it is common to see a far-field R wave on the electrogram. This results since the large mass of the ventricle generates an electric signal seen by the lead at a distance. The far-field R wave should be significantly smaller than the P wave (ide-
ally one-quarter the size or smaller) to ensure that it is not detected by the device. This is particularly true in defibrillators, since reliable sensing of P waves is important for rhythm discrimination.

In addition to peak amplitude, other aspects of sensing should be considered. The change in voltage with time (dV/dt), the slew rate, of the intrinsic deflection may be clinically important. Usually, this is most important in patients with borderline sensing voltages. In patients with low voltages (<5 mV), the slew rate measurement may be helpful and with current pacing system analyzers is easy to obtain. Some patients with a QRS of 3 mV but a slow slew rate may have undersensing, whereas other patients with a QRS of 3 mV but a normal slew rate may have adequate sensing.

Historically, some implanters would assess the current of injury at the time of lead placement but this is not commonly done at this time. The current, appearing as an increase in the electrical potential that immediately follows the intrinsic deflection, represents a small area of endocardium that reacts to placement of the lead (Fig. 5.52). This finding indicates adequate contact with the endocardium. A large current of injury may be mistaken for an adequate sensed intrinsic electrogram.

Additional measurements
Assessment of AV nodal conduction is necessary if an AAI pacemaker is to be implanted. In this situation, the atrial lead is positioned and the atrium is paced at rates nearly equal to the sinus rate and then at incremental rates up to approximately 150 ppm. A typical sequence might be 80, 100, 120, 140 and 160 ppm. The pacing rate at which Wenckebach, or higher grade, AV block occurs is recorded, as is the AR interval (paced atrial event to intrinsic QRS). To proceed with AAI pacing, the patient should have 1:1 conduction to rates of 130–140 ppm without any significant prolongation of the AR interval.

Epicardial systems
Epicardial (also called myocardial) systems account for a very small percentage of device implantation procedures. Three groups of patients still undergo placement of epicardial systems.
Patients undergoing cardiac surgery for another indication. In these patients, permanent epicardial leads may be placed at the time of surgery. Alternatively, some of these patients have temporary pacing until recovery from open-heart surgery. Before dismissal from the hospital, they may undergo placement of a transvenous pacing system. This latter approach is preferable, since transvenous leads have proven to be more reliable than epicardial leads.

Patients with a prosthetic tricuspid valve, a congenital anomaly, or atresia of the tricuspid valve without access to the CS. In these patients, epicardial ventricular leads are usually required. Bioprosthetic valves are, however, compatible with transvenous implantation. It is difficult to quantify adverse outcomes from placing a transvenous lead across a bioprosthetic valve, and it is certainly in the patient’s best interest to protect the bioprosthetic valve as much as possible. If a lead is to be placed across a bioprosthetic valve it is advisable to use the smallest lead possible, i.e., smallest French size.

Patients with ventricular septal defects or patients with right-to-left shunts in whom the possibility for systemic embolization exists.

Two surgical procedures have been described for the placement of epicardial leads: (i) subxiphoid, or left costal, approach, and (ii) left lateral thoracotomy. Such procedures obviously require a trained surgeon, and the reader is referred to cardiovascular surgical texts for details of these approaches.

In patients who have either tricuspid atresia or other conditions that preclude entering the RV through the tricuspid valve, the CS and ventricular vein may be used to pace the ventricle. In some types of Fontan correction, the CS continues to drain into the right atrium. If no other shunts at the atrial or ventricular level are present, the CS can be cannulated (as described under left ventricular lead placement) and the lead placed in a ventricular vein. Care must be taken to ensure that there are no insidious shunts (fenestration of the Fontan patch or unroofing of the CS). Because left ventricular leads tend to be less stable than endocardial screw-in leads, in patients with pacemaker dependence it may be best to proceed with epicardial pacing even when the CS is accessible.

Hardware adaptations

The “connector pin” of the implanted lead connector fits into an appropriate connector cavity in the “header” of the pulse generator to provide the permanent but reversible connection between the two. The connector cavity in the header holds the proximal (i.e., the extravascular) end of the lead.

Although a few bifurcated bipolar leads may remain in service, the vast majority of contemporary leads, whether unipolar or bipolar and whether coaxial or some other conductor design, employ “in-line” connectors. They conform to a formal, international connector standard, published by the International Standards Organization in Brussels, Belgium known as the “international standard,” or IS-1, design.

Older voluntary standard (VS) lead connector designs and the current/IS-1 pacing lead connectors are 3.2 mm in diameter, have sealing rings on the lead, and have a short (0.508 cm) connector pin (Fig. 5.53). The old ‘VS-1’ and the current IS-1 pacing lead connectors fit pacemakers that have 3.2-mm connector cavities. VS-1/IS-1 pacemaker header connector cavities are 3.2 mm in diameter, have no sealing rings, and have a short (0.508 cm) cavity bore for the lead’s connector pin, and accept only the VS-1/IS-1 pacing lead connectors.

A few pacemakers still exist that have header connector cavities with the designations ‘VS-1A’ and ‘VS-1B, and are a point of confusion (Fig. 5.54). Pacemaker connector cavities designated VS-1A are 3.2 mm in diameter, have no sealing rings, and have a long (0.851 cm) receptacle for the lead’s connector pin. Pacemakers with the VS-1B designation have sealing rings in the header’s connector cavities.

VS+1/IS-1

Long-pin with sealing rings

Long-pin without sealing rings

Fig. 5.53 VS-1/IS-1 connectors are intended to restore universal interconnection of all leads and pulse generators via the standardized 3.2-mm connectors. The pin is connected to the negative output and the ring to the positive terminal in the pulse generator header. Unipolar leads of similar configuration exist, but without a positive terminal, and are of the same size as and interchangeable with a bipolar receptacle. The ridges represent the sealing rings, which prevent the ingress of fluid into the header.
Implantation and Extraction Techniques

Chapter 5

Variations in pacemaker headers. See text.

Table 5.2 Pacemaker header connector cavity variations

<table>
<thead>
<tr>
<th>Pacemaker header connector cavity</th>
<th>Lead connector</th>
</tr>
</thead>
<tbody>
<tr>
<td>VS-1/IS-1</td>
<td>Accepts only VS-1/IS-1 lead connectors</td>
</tr>
<tr>
<td>VS-1A/IS-1</td>
<td>Accepts VS-1A or IS-1 leads and 3.2-mm in-line leads with a longer pin</td>
</tr>
<tr>
<td>VS-1B/IS-1</td>
<td>Accepts VS-1B or IS-1 leads and 3.2-mm in-line leads that have a longer pin and sealing rings</td>
</tr>
</tbody>
</table>

Table 5.3 Specific adaptor for specific combination

<table>
<thead>
<tr>
<th>Pulse generator connector cavity</th>
<th>Unipolar</th>
<th>In-line bipolar</th>
<th>Bifurcated bipolar</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unipolar</td>
<td></td>
<td>Low-profile adaptor sleeve (1)</td>
<td>End cap (2)</td>
</tr>
<tr>
<td>In-line bipolar</td>
<td></td>
<td>Low-profile lead to bifurcated pulse generator (5) and an indifferent electrode (4)</td>
<td>Bifurcated lead to in-line generator adaptor (3)</td>
</tr>
<tr>
<td>Bipolar with bifurcated connector</td>
<td></td>
<td>Indifferent electrode (4)</td>
<td>Low-profile lead to bifurcated pulse generator adaptor (5)</td>
</tr>
</tbody>
</table>

Connector that conducts to the distal pace/sense electrodes, and one or two DF-1 connectors that conduct to the defibrillation coils. At the time of writing, the soon to be available IS-4 connector, which will be a single in-line connector for both pacing and defibrillation, has not been incorporated into commercially available leads. Pacing and ICD lead selection is discussed in Chapter 4.

**Special considerations in pediatric patients**

Device implantation in the pediatric population raises specific issues, including the size and expected growth of the patients, whether congenital heart disease is associated, the need for long-term pacing, and whether the age and size of the patient should influence the selection of the system implanted.

Transvenous systems are used most frequently in the pediatric population, but epicardial systems remain very important for the pediatric age group, especially the neonate and infant.33

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*Fig. 5.55* Various adaptors used for pacing lead and pulse generator mismatches. From left to right: DF1 to LV1 lead adaptor; adapt two unipolar leads or a bifurcated lead to a bipolar in-line connector; Allen wrench used to place the small screws needed for certain lead adaptors; in-line lead to a bifurcated connector; end cap; bipolar in-line sleeve adaptor to convert to a unipolar generator.

*Fig. 5.56* Schematic representation of the DF-1 connector for implantable cardioverter-defibrillator leads. Dimensions in millimetres unless otherwise noted.
Older literature with much earlier designs of epicardial leads demonstrated that survival of endocardial leads was superior to that of epicardial leads in pediatric patients. However, with improvement in epicardial lead design there is probably less difference in lead longevity, although controversy persists. The lead for the pediatric patient, be it neonate, infant or older, should be chosen based on the individual patient.

In the pediatric patient with congenital cardiovascular anomalies who is undergoing device implantation, it is helpful to know before implantation whether the child has an associated persistent left SVC. This can usually be determined echocardiographically. If concern exists, angiography is diagnostic. Advancing the pacing lead into a persistent left SVC results in traversing the CS and entering the heart in the right atrium. This makes ventricular access more difficult to the position and angulation of the lead as it emerges from the CS. To avoid the problems associated with a persistent left SVC, the right subclavian vein should be used if there is any doubt about whether the patient has a persistent left SVC. Even with a left SVC, the patient usually has a right SVC.

For lead placement, there are two potential approaches. The first is to allow more lead redundancy than would usually be left in an adult patient, but otherwise to use standard techniques to place the lead, including securing the lead with the sleeve provided. However, instead of securing the sleeve with nonabsorbable suture, as is our usual practice, some believe that the use of absorbable suture may allow the lead to advance as the child grows. Whether or not this makes a long-term difference for the patient is difficult to prove. If the lead were to become fibrosed along the venous route or in an intramyocardial position, the lead could not advance. However, growth of the pediatric patient may minimize fibrosis.

The additional redundancy of the lead allows for growth of the pediatric patient (Fig. 5.57). If this approach is taken, the leads must be evaluated periodically during follow-up. If the child “outgrows” the lead, i.e., there is radiographic evidence of straightening of the lead, it is necessary to place a new lead. Because of entrapment of the lead in the venous system and cardiac chambers, the lead may not “advance” on its own, and lead advancement may not even be possible with a stylet in place after the lead has been implanted for a long period. Although a variety of clinical situations may develop when the patient truly “outgrows” the pacing system, most commonly, intermittent sensing abnormalities occur as tension develops on the lead at the electrode–tissue interface.

It is of particular importance to select the smallest pulse generator that serves the needs of the pediatric patient. The small weight and dimensions of current pacemakers allow implantation in a prepectoral position in a patient of almost any size. In very small infants, if there is concern that there is not enough subcutaneous tissue to protect the device, consideration may be given to placing the pulse generator in a subpectoral position. In our experience, this placement is not often necessary with pacemakers, but has been necessary at times for ICDs. Before the advent of very small pulse generators, the transvenous lead was occasionally tunneled subcutaneously from the pectoral entry site to an area in the abdomen or flank where the pulse generator could be placed more easily. This approach, too, is rarely necessary with the small size of currently available pulse generators.

Traditional venous routes can be used in the pediatric patient; that is, the axillary puncture technique with placement of one or two leads via the axillary vein is usually possible. Two leads can often be placed via the cephalic vein as well. Although rarely used, a single-pass VDD pacing system could be considered in the pediatric patient with AV block. Such a system minimizes hardware but still accomplishes AV synchrony and maintains P-synchronous rate adaptation if the sinus node is intact.

Although any standard pacing lead can be used in pediatric patients, active fixation leads are generally preferred for specific reasons. Active fixation may allow additional stability of the lead in the immediate postimplantation period, when it is difficult to control the activities of a pediatric patient. The pediatric patient may require several pacing systems during the growth years. Although a noninfected lead may be abandoned and left in place, it is reasonable to attempt removal of abandoned leads in the pediatric patient so that an excessive amount of hardware does not accumulate in the patient throughout a lifetime. Some preference has therefore been given to active fixation leads, because they are generally easier to remove than a long-term passive fixation (tined) lead.

Finally, active fixation leads can be placed in a greater variety of positions than passive fixation leads. This advantage is important in the patient with associated congenital heart disease, because the anatomy may be
quite distorted. An active fixation lead allows placement in all portions of the atrium, not the atrial appendage alone. Active fixation leads have been used in patients after the Mustard procedure, with the leads placed across the intra-atrial baffle and pacing in the left atrium.

A specific problem in pediatric pacing involves cardiac pacing after the Fontan procedure. Because the postoperative anatomy precludes transvenous endocardial ventricular pacing, dual-chamber pacing in these patients has been accomplished by placing a ventricular epicardial lead at the time of surgery and subsequently placing an atrial endocardial lead and tunneling the two leads to a common prepectoral position for attachment to a dual-chamber pacemaker (Fig. 5.58).

**Device implantation after cardiac transplantation**

There are special considerations for device implantation in the patient who has undergone cardiac transplantation. Although dependent on the surgical transplant

![Image of serial radiographs](image_url)
technique used, often after cardiac transplantation, the donor atrium can no longer receive stimuli from the intrinsic or native (recipient) sinoatrial node. The sinoatrial node remains in continuity with the recipient atrium and may drive the recipient atrium at a normal rate or at a rate more rapid than normal. The suture line between the free wall of the donor atrium and the recipient atrium is a barrier to the passage of
stimuli, which normally traverse the atrium to reach the AV node and bundle of His. After transplantation, the patient has two atrial rhythms, that of the donor atrium and that of the recipient atrium, both of which may be visible on the electrocardiogram.

Several approaches have been used for pacing in cardiac transplant recipients, some of which are quite complex. We have taken a conservative and simple approach. Because normal AV conduction usually exists between the donor atrium and ventricle, atrial pacing could be used both to preserve the AV sequence and to modulate the rate appropriately. Also, in many transplant recipients, sinus node dysfunction, a potential clinical problem from 1 month to 6 months after transplantation, often resolves. Nevertheless, our approach has been to implant a standard dual-chamber pacemaker and to position the atrial lead in the donor atrium. Even if there is no clinical manifestation of AV conduction disease, we are more comfortable placing a dual-chamber pacemaker for the unlikely event of late AV block.37,38

**Hospital stay after implantation**

The length of time the patient should be kept in hospital after pulse generator implantation varies among institutions. Patients are told to fast after midnight the night prior to the device implantation or pulse generator replacement. They are instructed to take their chronic medications with as little water as possible the morning of the procedure.

No medications are routinely withheld prior to elective pulse generator implantation. If the patient is on aspirin and/or clopidogrel (Plavix) the medications are continued. However, the patient is told that there may be significant ecchymoses following the procedure and there may be a higher risk of hematoma formation. This approach is taken because the risk of aspirin alone is minimal and does not merit discontinuation of the drug and waiting the length of time necessary for platelet function to return to normal. If consideration were to be given to discontinuation of Plavix, the risk of stopping the drug would have to be considered.

We do not reverse coumadin prior to pulse generator implantation. We prefer that the International Normalized Ratio (INR) be ≤2.5. If the patient is chronically anticoagulated and generally has an INR of >2.5, we suggest that the coumadin be held for 1–2 nights prior to implantation. An INR is rechecked the morning of the procedure to be certain that the INR is ≤2.5. Patients are allowed to resume coumadin the evening of the implant procedure. We have not appreciated any increase in hematoma formation or bleeding complications with this protocol. Others have described a similar

![Fig. 5.58 Posteroanterior (A) and lateral (B) radiographs from a patient with a dual-chamber pacemaker. The ventricle is paced from the epicardial position, and the atrium is paced from the endocardial position. The atrial lead is then tunneled subcutaneously to the site of the pulse generator.](image)
experience with implant procedures in the anticoagulated patient. Conversely, unfractionated heparin or low-molecular-weight heparin are always discontinued prior to device implant and ideally avoided for a minimum of 24 h post implantation.

We currently admit patients the morning of the procedure and usually dismiss them the next morning. The patient is monitored during the overnight stay. If the patient is pacemaker-dependent, we keep the patient at bed rest overnight after implantation unless a different duration is ordered as an exception. A posteroanterior and lateral chest X-ray is obtained after the bedrest restriction is completed. Before dismissal, thresholds are documented and the pulse generator is programmed to its final settings. When the patient’s pulse generator reaches battery depletion, the pulse generator is replaced on an outpatient procedure.

Many institutions perform initial device implantation as an outpatient procedure. Some physicians restrict outpatient implantation to nonpacemaker-dependent patients, whereas others perform outpatient procedures regardless of dependency status. Some third-party payers now insist that device implantation be accomplished as an outpatient procedure or that the hospital stay be < 24 h. If the implantation is done as an outpatient procedure, a mechanism should exist whereby the patient can be seen urgently by caregivers knowledgeable in device management should questions or problems arise.

**Pulse generator replacement**

Expected battery depletion is the most common cause of pulse generator replacement (Fig. 5.59 and 5.60).

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**Fig. 5.59** Reasons given for removing/ replacing pulse generators. (Total devices = 2652). (Reprinted from Heart Rhythm with permission.)

**Fig. 5.60** Battery longevity for pulse generators with vs. those without the capability of rate adaptation. The percentages represent proportion of pulse generators that failed prematurely, defined by this registry as ≤3 years. (Reprinted from Heart Rhythm with permission.)
When it has been established that a pacemaker or defibrillator has reached elective replacement indicators (ERI), the patient should be notified and plans made to bring the patient in for pulse generator change. Usually, the implanted device will perform reliably for approximately 2–3 months following ERI, although the actual time is a function of the device model, pacing frequency and other factors. In our practice, we electively replace devices within 2–4 weeks of ERI.

Having completed a focused history and physical, reviewed medications and checked basic preoperative laboratory work, i.e., potassium, sodium, creatinine, fasting glucose, complete blood count, the patient is told to fast after midnight the night prior to the procedure. The patient comes to the hospital early the following morning and every attempt is made to complete the pulse-generator replacements as the initial cases so the patient may recover early and be dismissed from hospital in the afternoon.

The patient is taken to the implant suite and, if pacemaker dependent, a temporary pacemaker is placed via the femoral vein. After infiltration with 1% lidocaine, the prior infraclavicular incision is reincised and dissection carried out to the level of the pacemaker, taking care to avoid the permanent leads. Parallel incisions (i.e., a new incision, parallel to the old one) are avoided due to compromise of the vascular supply between the new and old incisions; it is preferable to reincise the old scar. The device is explanted and disconnected from the chronic leads and thresholds checked via the pacing system analyzer. If the thresholds are acceptable (see below), the pocket is inspected for hemostasis, any bleeding areas cauterized, and the pocket is copiously irrigated with saline solution. The new pulse generator is connected to the chronic leads and the leads are gently “tugged” to be certain they are securely in the connector block. The device is then placed in the pocket and we routinely recheck thresholds via the programmer. This assures the implanter that the leads are making good contact in the connector block and the appropriate lead has been placed in the appropriate port. The incision is then closed in our standard fashion.

What is considered acceptable for chronic thresholds will depend on the patient and associated comorbidities. We generally prefer chronic pacing threshold to be < 2.0 V and there be no significant change in measured intrinsic P or R wave or impedance from the initial implant. In the patient who is very elderly, in whom the longevity of the pulse generator is not a major consideration, or the patient in whom there are comorbidities or other issues that make the patient less likely to tolerate or unwilling to consider any risks inherent in lead replacement, higher chronic thresholds may be accepted.

Whether the pulse generator is being changed for battery depletion or on the basis of a recall or advisory, patients should be informed of the risks of this relatively minor procedure. This is especially important when the pulse generator is being replaced on the basis of an advisory or recall, because the risk of device failure may be lower than the risk of pulse generator change-out; the patients need this information to make an informed decision. Operation-associated complications requiring intervention were noted in 1.24% of our population. Complications included five infections, three hematomas and one incisional dehiscence. Although this complication rate is lower than that reported in a multicenter survey of pulse generator complications that were seen in 8.1% of patients, generator replacement is not a benign procedure, and associated risks must be weighed in the context of other variables when this is considered on the basis of a recall or advisory.

**Postimplant order set**

We have established specific order sets that are used for patients following device implantation (Fig. 5.61). This approach not only ensures consistency in postoperative care and management, but also allows for greater efficiency and adds an extra safety measure. If any electronic order that is part of the order set in any way contradicts or significantly alters the standard order set, the RN entering the electronic order set must account for the discrepancy and when the MD responsible for issuing and signing the orders completes the sign-off, they will again need to recognize and explain any discrepancies. If the patient has an allergy listed in the electronic record, and medications ordered as part of the order set, e.g., antibiotics, include a medication that has been listed as an allergy or has cross-reactivity to a noted medication allergy, the orders cannot be completed until the discrepancy is reconciled or explained.

As noted in Fig. 5.61, we do routinely use antibiotics post device implantation. Although good sterile technique is the most important factor in avoiding an implant-related complication of infection and some
CHAPTER 5  Implantation and Extraction Techniques

controversy exists regarding postimplant antibiotic administration, we favor their use. Our antibiotic regimen is shown in Table 5.4.

**Homegoing instructions**

After implantation, the incision is covered with sterile gauze and tape or a “coverlet.” This is generally removed the next morning, and if the incision is dry it is left uncovered. Patients are allowed to bathe 48 h after implantation, but instructed not to scrub the incisional site but to simply allow water to run over the site.

Postoperatively, we recommend that the patient avoid lifting the arm on the side of the pulse generator higher than shoulder level for the first 4 weeks after

---

**Fig. 5.61** Order set used for patients post device implantation. The “inset” highlights a few of the specific orders included.
implantation. This restriction may be overcautious, but it serves to remind the patient to avoid aggressive activities while at the same time not significantly limiting arm or shoulder motion or activities of daily living. Use of a loose sling for the first 5–7 days may serve as a helpful reminder to limit arm and shoulder activities. Patients should be instructed not to overreact to this recommendation and completely restrict the movement of the ipsilateral shoulder. Movement should be encouraged, because immobility may cause pain later when full mobilization is attempted, with consequent further restriction of movement (frozen shoulder) or reflex sympathetic dystrophy. Movement of the shoulder should be encouraged on the first postoperative day. Early movement will not displace a well-placed and secure lead system.

For medicolegal reasons, we recommend that the patient not drive for 2 weeks after receiving a pacemaker or CRT implant. Return to driving after ICD implantation is a more complex issue and depends on the clinical history, rhythm disturbance requiring the ICD, and applicable state or country regulations. Briefly, when ICDs are implanted for prophylactic sudden death prevention, driving restrictions are similar to pacing restrictions. In the setting of syncope or ventricular tachyarrhythmias, patients are advised not to drive for 6 months. This is discussed in greater detail in Chapter 13.

A registered nurse with expertise in patient education and implantable devices meets with the patient and available family members prior to discharge. The RN instructs the patient in postimplant care and assessment of the implant site, basic device function, postimplant restrictions, transtelephonic and/or remote monitoring of the device, in-clinic follow-up schedule, how and when to contact caregivers in the event of concern related to the implantable device and any other questions posed by the patient or family. The patient is also given an instructional CD regarding the device and device management and follow-up, as well as educational brochures related to implantable devices.

**Lead extraction**

Lead extraction is a necessity in some patients, primarily those with infected device systems, but the procedure is not without significant potential risks, including death, and should not be undertaken lightly or without a thorough understanding of the technique and possible difficulties.

**Indications for lead extraction**

Although some controversy exists about indications for lead extractions, absolute indications can be categorized as class 1, conditions for which there is general agreement that leads should be removed; class 2, conditions for which leads are often removed but for which opinion differs somewhat on whether the benefit outweighs the risk of removal; and class 3, conditions for which there is general agreement that removal of leads is unnecessary. The following classification is taken from the Heart Rhythm Society (HRS) (formerly the North American Society of Pacing and Electrophysiology) Policy Statement on Recommendations for Extraction of Chronically Implanted Transvenous Pacing and Defibrillator Leads.

**Class 1**

- Sepsis (including endocarditis) as a result of documented infection of any intravascular part of the pacing system or as a result of a pacemaker pocket infection when the intravascular portion of the lead system cannot be aseptically separated from the pocket
- Life-threatening arrhythmias secondary to a retained lead fragment
- A retained lead, lead fragment, or extraction hardware that poses an immediate or imminent physical threat to the patient, e.g., Accufix atrial J

**Table 5.4 Mayo Clinic Rochester antibiotic recommendations for implantable device procedure**

<table>
<thead>
<tr>
<th>Antibiotic</th>
<th>Dosage</th>
<th>Administration</th>
<th>Indications</th>
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<tbody>
<tr>
<td>Cefazolin</td>
<td>1 g if &lt;80 kg or 2 g if &gt;80 kg, within 60 min before the initial incision and every 8 h for two doses in patients staying overnight. (For pulse generator change only the initial dose is given.)</td>
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<tr>
<td>Vancomycin</td>
<td>20 mg/kg intravenously within 2 h before the initial incision. If patient is staying overnight, 15 mg/kg once 12 h later. Dose is adjusted for renal insufficiency. (For pulse generator change only the initial dose is given.)</td>
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</tr>
<tr>
<td>Cefazolin</td>
<td>1 g intravenously if &lt;80 kg or 2 g if &gt;80 kg, within 60 min before the initial incision and every 8 h for two doses in patients staying overnight. (For pulse generator change only the initial dose is given.)</td>
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</table>
CHAPTER 5 Implantation and Extraction Techniques

- Clinically significant thromboembolic events caused by a retained lead or lead fragment
- Obliteration or occlusion of all usable veins, with the need to implant a new transvenous pacing system
- A lead that interferes with the operation of another implanted device (e.g., pacemaker or ICD).

Class 2
- Localized pocket infection, erosion, or chronic draining sinus that does not involve the transvenous portion of the lead system, if the lead can be cut through a clean incision that is totally separate from the infected area
- An occult infection for which no source can be found and for which the pacing system is suspected
- Chronic pain at the pocket or lead insertion site that causes significant discomfort for the patient, that cannot be managed by medical or surgical technique without lead removal, and for which there is no acceptable alternative means of relief
- A lead that, due to its design or its failure, may pose a threat to the patient that is not immediate or imminent if left in place
- A lead that interferes with the treatment of a malignant lesion
- A traumatic injury to the entry site of the lead that cannot be reconstructed without interference from the lead
- Leads preventing access to the venous circulation for newly required implantable devices
- Nonfunctional leads in a young patient.

Class 3
- Any situation in which the risk posed by removal of the lead is significantly higher than the benefit of removing the lead
- A single lead in a vessel that has become nonfunctional in an older patient
- A normally functioning lead that has a reliable performance history at the time of pulse generator replacement.

If the lead can potentially harm the patient, extraction should be considered. Infection and mechanical complications of retained leads have been the obvious lead complications with the potential to harm the patient. Of the multiple lead advisories issued in the past decade, most have been for leads with unacceptably high pacing or sensing failure rates due to insulation problems. Although these failed leads may have required abandonment and implantation of a new lead, extraction of the defective lead has not usually been necessary.

The recall of the Accufix atrial J lead that occurred in the 1990s differed significantly, because simply abandoning the lead and placing a new atrial lead did not protect the patient. This lead is largely of historical interest, but some centers, including ours, are still following patients with this problem lead. The Accufix lead incorporated a small wire to retain the J shape of the atrial lead. The wire has been shown to have the potential to fracture, and if the wire breaks and extrudes through the insulation, it can lacerate the aorta or perforate the atrial myocardium, injuries leading to fatal bleeding or cardiac tamponade.

The indications for lead extraction listed above are meant to be used as a guideline. Each patient’s situation must be individualized, and the procedure and potential complications should be discussed in detail with the patient. The HRS policy statement lists the following clinical factors that should be taken into consideration:

- Patient’s age
- Patient’s sex (published complication rates are higher in women)
- Patient’s overall health, both physical and mental
- Calcification involving the lead or leads
- Vegetations in the heart
- Number of leads in the intravascular space
- Length of time the lead or leads have been in place
- Fragility, condition and physical characteristics of the lead
- Experience of the physician
- Patient’s preference, i.e., extraction or not.

Facility requirements for lead extraction
Lead extraction should generally not be considered if the necessary equipment is not available, the patient is not a candidate for emergency thoracotomy should a complication require surgery, or there is known anomalous placement of the lead or leads through structures other than the normal venous and right-sided cardiac chambers (e.g., the leads are in an arterial position, left-sided cardiac chambers, pericardial space).

Who should perform extraction once the decision has been made to extract a lead? Less experienced operators have less successful outcomes, and the incidence of complications is higher, and the procedure time is longer. What qualifications are necessary for performance of lead extraction? Although guidelines are established for pacemaker implantation, they are
vague in referring to appropriate training for lead extraction. They state that it is preferable to have exposure to lead extraction techniques, and if a trainee cannot learn such techniques during the training period and wants to perform the procedure at a later date, that experience should be sought with someone expert in extraction.

Ideally, lead extraction procedures require specialized training in a center that frequently performs extraction. As noted above, stringent guidelines for training requirements for pacemaker implantation are in place, but similar guidelines for lead extraction are less precise. More rigorous guidelines for training requirements in lead extraction are needed. The HRS policy statement recommends that physicians being trained in lead extraction perform a minimum of 20 lead extractions as the primary operator under the direct supervision of a qualified training physician. The supervising physician, i.e., the one doing the training, should have performed more than 100 lead extractions with an efficacy safety record consistent with published data.

Complications of lead extraction
We generally quote patients a risk of potentially life-threatening complications of 2.1% and a risk of death of 0.6% from extraction. These numbers are based on extraction data gathered before the advent of laser-assisted extraction, and it is generally agreed that the risks of laser-assisted extraction are lower. In a single-center series of 200 extractions, death occurred in one patient, or 0.6%. Predictors of failed extraction in the multivariate analysis from this study were longer time from implant and hypertension. The only predictor of an acute complication was laser extraction from both the right and left sides during the same procedure.

Potential complications as outlined in the HRS policy statement are as follows:

**Major**
- Death
- Cardiac avulsion or tear requiring thoracotomy, pericardiocentesis, chest tube, or surgical repair
- Vascular tear requiring thoracotomy, pericardiocentesis, chest tube, or surgical repair
- Hemothorax or severe bleeding from any source requiring transfusion
- Pneumothorax requiring chest tube drainage
- Pulmonary embolism requiring thrombectomy or surgical intervention
- Respiratory arrest
- Septic shock
- Cerebrovascular accident.

**Minor**
- Pericardial effusion not requiring pericardiocentesis or surgical intervention
- Hemodynamically significant air embolism
- Pulmonary embolism not requiring intervention
- Vascular repair near the implantation site or venous entry site
- Arrhythmia requiring cardioversion.

Facility requirements have also been outlined by the HRS document. These include:
- An accredited cardiac surgery program on site
- An accredited cardiac catheterization program
- At least one physician who is properly trained and proficient in the technique of transvenous lead extraction
- Cardiothoracic surgeon on site and capable of initiating an emergency procedure within 5 min
- Anesthesiologist with working anesthesia equipment
- A full set of basic instruments for lead extraction
- High-quality fluoroscopy
- Transthoracic ultrasound and transesophageal ultrasound capability immediately available
- Physiological data acquisition equipment for arterial pressure monitoring and oxygen saturation monitoring
- Pericardiocentesis tray in the procedure room
- Thoracotomy tray in the procedure room
- Temporary pacing and defibrillation-cardioversion equipment in the procedure room
- Fluids, pressors, and other emergency medication available in the procedure room.

Extraction techniques
Several approaches to lead extraction that have been described. Multiple techniques can be used, including simple traction, locking stylet and telescoping sheaths with countertraction, an inferior approach with various catheter techniques to snare the lead, laser-lead extraction, extraction with electrosurgical dissection sheaths and open surgical techniques. Although individual operators may have definite biases about technique, it is important that the operator has a thorough understanding of the equipment and options at their disposal, allowing the approach to be defined by comfort level and expertise.
Before any attempt at extraction, the lead must be
freed from underlying tissue in the pocket, and any
sleeve around the lead securing it to underlying tis-
sue must be released. A stylet should be placed in the
lead before any traction is applied. Traction should
be gentle. Fluoroscopic observation may indicate the
extent of fibrosis holding the lead to the underlying
vascular structures. Fig. 5.62 shows how fibrous tis-
ue surrounds the lead. Fibrosis may occur in specific
areas or along the entire course of the lead.

As traction is applied (Fig. 5.63), the electrocardio-
gram should be monitored for ectopy, and the patient’s
blood pressure should also be observed for hypoten-
sion. Even if gentle traction is not initially successful,
continued steady traction for several minutes may be
successful.

If traction fails, our next approach is to transect the
lead just beyond the connector pin in a non-isodia-
metric portion of the lead, trying to leave as much as
possible with which to work. A locking stylet is placed
(Fig. 5.64). There are now several varieties of locking
stylets. Ideally, the locking stylet can be locked and un-
locked if necessary. Traction can again be attempted
with the locking stylet in place. If this is not successful,
either countertraction techniques or laser extraction
can be attempted. We tend to proceed straight to laser
Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach

extraction. Quite appropriately, however, others experienced in lead extraction prefer to place a countertraction sheath and attempt to pass this along the lead without laser. If successful, this is a more economical approach to extraction.

Results with the laser extraction and electrosurgical dissection techniques have been excellent\textsuperscript{47,49} and in experienced hands it can significantly decrease the time required for lead extraction. No extraction technique, including laser and electrosurgical dissection, is without potential morbidity and mortality, and all techniques require adequate training and a careful approach.\textsuperscript{49} Laser-assisted lead extraction has been the subject of a multicenter study, the Pacing Lead Extraction with the Excimer Laser System (PLEXES) trial. Randomization of patients to laser-assisted extraction technique or to standard extraction techniques demonstrated efficacy of laser-assisted lead extraction. Lead extraction can also be accomplished via an inferior approach, i.e., through the femoral vein. A variety of traction and snare techniques can be applied after vascular access is achieved. After placement of a relatively large sheath—16 Fr, a so-called workstation—a Dotter retriever, pigtail catheters, various sizes of snares, or other devices can be used to grasp and retract the lead.

Whoever is performing percutaneous lead extraction, cardiologist or surgeon, should not overlook the possibility of surgical removal of the leads. Although most leads can be extracted by a percutaneous technique, some cannot, and thoracotomy or some other limited surgical approach may be the best option for the patient.\textsuperscript{52}

The most recent challenge in lead extraction is the increasing requirement for experience in extraction of CS leads. Although clinical experience remains limited regarding extraction of chronically implanted leads in the coronary venous system, multiple techniques have been employed.\textsuperscript{53} At times, a combination of techniques is required to effect complete lead extraction. For example, laser extraction is required for the subclavian and superior vena caval portion of an adherent pacing or ICD lead. However, if the lead continues to be adherent within the ventricle itself, particularly at the tip, simple traction may
continue to be effective, and it is generally inadvisable to continue to lase into the ventricular myocardium. At this point, a femoral route can be used to snare the heel of the lead and apply traction from this femoral route. Any styles that were placed in the lead from above are removed and the leads cut in the infraclavicular pocket. With continued traction, the lead can often be removed in its entirety via the femoral vein. Another example of combined approaches is the application of radiofrequency energy through a standard ablation catheter for portions of retained lead fragments or intramyocardial adherent portions of the lead where laser is inaccessible. The lead now free can be snared and then removed via the femoral route.

Extraction techniques will continue to evolve and improve. For now and the foreseeable future, this procedure must be approached with great respect by personnel committed to developing expertise.

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CHAPTER 6

Implantation-related Complications

David L. Hayes, Paul A. Friedman

Although pulse generator implantation is usually straightforward and free of adverse events, there are multiple potential complications that can occur. Prior to device implant, not only should the procedure be explained in detail to the patient, but potential complications should also be discussed and that discussion documented in the permanent medical record. Our practice is to routinely discuss the complications that are the most common or that potentially carry the greatest threat to the patient. Specifically, this includes lead dislodgment, pneumothorax, infection, and cardiac perforation with tamponade. With internal cardioverter-defibrillator (ICD) implantation we also discuss complications associated with defibrillation threshold (DFT) testing as well as the potential need for additional hardware in order to achieve an adequate DFT. If a cardiac resynchronization therapy (CRT) system is being implanted, additional time is spent discussing problems that can arise with placement of the coronary venous lead.

Complications related directly to the implant procedure

Lead placement
Complications of lead placement result from cannulation of the vein, catheterization of the heart, and from placement of a permanent lead.

Lead dislodgment
Historically, the most common complication of transvenous pacing has been lead dislodgment. Improved fixation mechanisms have substantially reduced the frequency of this complication for both atrial and ventricular pacing leads. It is difficult to state precisely what rate of lead dislodgment is acceptable, but secondary intervention rates for all reasons should be <2% for ventricular leads and <3% for atrial leads. Some experienced implanters would argue that dislodgment rates should be even lower, e.g., ≤1% for ventricular leads and ≤2% for atrial leads. In the Pacemaker Selection in the Elderly (PASE) trial, lead dislodgment was the most common complication, occurring in nine of the 407 patients, or 2.2%. Slightly higher dislodgment rates may be tolerable in pediatric patients, whose activity is more difficult to control, and in patients with unusual anatomy, such as congenital cardiac anomalies.

Dislodgment is often classified as “macrodislodgment” or “microdislodgment.” Macrodislodgment is radiographically evident, and microdislodgment is not (Fig. 6.1). Adequate lead position is assessed by posteroanterior and lateral chest radiographs (see Chapter 11). Lead placement by chest radiography may appear excellent in the patient with a microdislodgment, but the tip has moved sufficiently to impair myocardial contact and function. Due to beat-to-beat variability in cardiac and lead position, only gross lead movement is identified as macrodislodgment.

The atrial lead dislodgment rate has traditionally been higher than the ventricular dislodgment rate. With current lead technology, the rate of atrial lead dislodgment should be <3%, and most would agree it should be <1–2% (Fig. 6.2). The dislodgment rates of active and passive fixation atrial leads are reported to be similar, and dislodgment is clearly related more to implant experience than to the fixation mechanism.

In the atrium, straight leads are more likely to dislodge than preformed J leads, but the preformed J leads are at greater risk for lead malfunction, so the overall performance between leads is similar.²
Fig. 6.1 Posteroanterior (A) and lateral (B) chest radiographs on the day after implantable cardioverter-defibrillator (ICD) implantation. The fluoroscopic image at the end of the implant had demonstrated a right ventricular apical position and adequate “J” on the atrial lead. However, on the X-ray shown, both atrial and ventricular lead positioning is suboptimal due to gross dislodgment. It appears that neither was adequately secured and have pulled back to a more shallow position. (C) The tracing was obtained when the ICD was programmed to the VVI pacing mode. Note complete failure to capture.
Fig. 6.2 Posteroanterior (PA) (A) and lateral (B) radiographs on the morning after implantation of a dual-chamber pacemaker. The active-fixation atrial lead appears to be somewhat shallow on the PA film but reasonable “J” configuration of the lead is noted on the lateral film. Atrial thresholds were excellent. PA (C) and lateral (D) radiographs obtained a few days later when the patient presented with vague fatigue. The atrial lead has clearly dislodged, being most evident on the lateral view.
In a prospective comparison of atrial passive and active fixation J-shaped leads, both performed well. However, passive fixation leads required less fluoroscopy at implantation, had better thresholds, and had a considerably lower rate of pericardial irritation or effusion. As newer, smaller leads become available, performance characteristics will further evolve.

The dislodgment rate of coronary sinus leads tends to be higher for several reasons. In addition to micro- and macrodislodgment giving rise to inadequate thresholds being made more likely with the complex venous system and the absence of active fixation, even with adequate thresholds, phrenic nerve stimulation or inadequate resynchronization may occur as a result of lead dislodgment. When counseling patients scheduled to receive a left ventricular lead, the importance of checking for adequacy of resynchronization (EKG, echo) in addition to the need for threshold testing and the higher incidence of lead revision requirement should be explained.

Complications of coronary sinus lead placement, including dislodgment, from MUSTIC, CONTAK-CD and MIRACLE-ICD have been summarized in an Expert Consensus Statement: Resynchronization Therapy for Heart Failure from the Heart Rhythm Society. A table from that consensus statement summarizing coronary sinus lead implantation success rates, implant problems, complications and thresholds is shown below (Table 6.1).

### Pneumothorax
Complications of venous entry, inherent in any approach to venous structures, include damage to associated arterial or neural structures, extensive bleeding, air embolism, and thrombosis. With the subclavian approach, the potential for pneumothorax also exists; it can be minimized by knowledge of the patient’s anatomy, attention to detail, and contrast venography (Fig. 6.3). In the PASE trial, pneumothorax occurred in 1.97% of patients and was more common in older patients and patients with lower body mass indices (< 20 kg/m²). It has also been reported that when experienced implanters use the subclavian puncture technique, the incidence of pneumothorax approaches 1%.

Although we have not prospectively analyzed our data, we believe that contrast venography lowers the incidence of pneumothorax. This technique is discussed in Chapter 5, Implantation techniques.

### Table 6.1 Complications of coronary sinus lead placement. Reproduced with permission from 4

<table>
<thead>
<tr>
<th></th>
<th>MUSTIC</th>
<th>CONTAK-CD</th>
<th>MIRACLE-ICD</th>
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<tbody>
<tr>
<td><strong>N</strong></td>
<td>64</td>
<td>286</td>
<td>421</td>
</tr>
<tr>
<td>Successful implantation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• First attempt</td>
<td>90%</td>
<td>87%</td>
<td>NA</td>
</tr>
<tr>
<td>• Total</td>
<td>92%</td>
<td>NA</td>
<td>88%</td>
</tr>
<tr>
<td>Implantation problems</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Failure</td>
<td>8%</td>
<td>13%</td>
<td>12%</td>
</tr>
<tr>
<td>• Coronary sinus trauma</td>
<td>NA</td>
<td>2%</td>
<td>4%</td>
</tr>
<tr>
<td>• Deaths</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>• Others</td>
<td>4.5%</td>
<td>15.2%</td>
<td>38%</td>
</tr>
<tr>
<td>Late complications</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Dislodgment</td>
<td>13.6%</td>
<td>6.8%</td>
<td>8.6%</td>
</tr>
<tr>
<td>• Extracardiac stimulation</td>
<td>12%</td>
<td>1.6%</td>
<td>3.0%</td>
</tr>
<tr>
<td>• Pocket infection</td>
<td>3.4%</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>• Loss of capture</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>• Deaths</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>• Other</td>
<td>3.4%</td>
<td>1.8%</td>
<td>1.3%</td>
</tr>
<tr>
<td>Pacing thresholds (Ldts)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• At implantation</td>
<td>1.36 ± 0.96</td>
<td>NA</td>
<td>1.5–1.7 (Model 4189)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1.7–2.3 (Models 2187/8)</td>
</tr>
<tr>
<td>• Chronic</td>
<td>2.4 (3 mo)</td>
<td>1.8 ± 1.2 (13 mo)</td>
<td>NA</td>
</tr>
</tbody>
</table>

NA – not available.
If a pneumothorax develops, it may manifest during the pacemaker procedure or as late as 48 h after implantation. Indications of pneumothorax are aspiration of air during subclavian puncture when the exploring needle is either introduced or removed, unexplained hypotension, chest pain, and respiratory distress.

After puncture of the subclavian or axillary vein, a chest radiograph should be obtained and inspected specifically for pneumothorax. A pneumothorax estimated to involve < 10% of the pleural space can probably be observed without chest tube placement. A chest tube should be considered if > 10% of the lung is involved,
the patient has continued respiratory distress, or hemopneumothorax is present.

If the subclavian artery is lacerated, hemopneumothorax may occur (Fig. 6.4) or bleeding may occur into the tissues, resulting in hematoma formation (Fig. 6.5). The regional anatomy must be considered before subclavian puncture is undertaken. In a patient with unusual anatomy of the chest wall or clavicle, the subclavian vein can be displaced and the usual landmarks used for subclavian puncture can be altered. Care must also be taken in the kyphotic patient in whom the venous anatomy may be displaced. As noted in Chapter 5, peripheral injection of contrast media and fluoroscopic guidance of the subclavian puncture may help to minimize complications (see Figs 5.6 and 5.7). Subclavian arterial dilation or aneurysm may also displace the targeted veins. Handheld ultrasound can also be used to identify vessel location.

Other potential complications of subclavian venous entry are air embolism, arteriovenous fistula, thoracic duct injury, and brachial plexus injury. Although all are uncommon, it is essential that the implanter using the subclavian puncture technique be familiar with the potential problems.

**Lead perforation**

Myocardial perforation during lead placement is an uncommon but potentially serious complication. Myocardial perforation is caused by improper force on the lead against the right ventricular (RV) free wall. Perforation of the coronary sinus may also occur as a complication of left ventricular lead placement.

The true frequency of lead perforation is difficult to determine and varies widely depending on the series and types of leads evaluated. Perforation has been shown to occur in 0.1–0.8% of patients undergoing pacemaker implantation and 0.6–5.2% in patients undergoing ICD implantation. In the PASE trial, perforations occurred in four of the 407 randomized patients, 0.98%. Perforations are more common in elderly patients in whom the RV wall may be thinner. Anecdotally, the risk is higher in elderly women than men. In patients in whom a post-implant pericardial effusion was present after implant and was believed to be a consequence of perforation, risk factors in a multivariate analysis included the presence of a temporary pacemaker, helical screw leads, and systemic steroid use (Fig. 6.6). The only protective factor was RV systolic pressure > 35 mmHg.

![Fig. 6.5 Posteroanterior chest radiograph of a patient following attempted pacemaker implantation. During the procedure the subclavian artery was inadvertently punctured. On the radiograph shown, there is fullness of the axillary and left lateral chest and a density can be appreciated above the left breast. This represents hematoma formation which corresponded to a substantial fall in the patient’s hemoglobin.](image)

Diagnosis of a perforation is usually based on clinical findings. Echocardiographic imaging may suggest perforation, but unless the lead is completely through the myocardium, the study may be inconclusive. More recently, computed tomography (CT) has been reported as a method of diagnosing myocardial perforation (Fig. 6.7). In a retrospective review of CT scans obtained in 100 patients with prior device implantation there was a surprisingly high incidence of perforations that could be radiographically identified. Overall, 15% of patients had a lead perforation. Rates of perforation...
were 15% for atrial leads and 6% for ventricular leads. There was perforation of 14% of RV ICD leads and 3% of RV pacemaker leads. Active fixation right atrial leads had a 12% perforation rate and passive fixation atrial leads had a 25% perforation rate. Of RV leads, 7% of active fixation and 5% of RV passive fixation leads demonstrated perforation. When correlated with measured pacing values, there was no difference in impedance between perforated and nonperforated leads. Only one perforated ventricular lead was said to have a “high” threshold. In an older autopsy study, lead perforation was seen in 27% of patients with right atrial leads.

Perforation may present in multiple ways. Perforation may potentially be asymptomatic and detected radiographically or by a rising stimulation threshold. In other patients, signs may include right bundle branch block (RBBB)-paced rhythm in a patient in whom the lead is placed in the RV (Fig. 6.8), intercostal muscle or diaphragmatic contraction, friction rub after implantation pericarditis, pericardial effusion, and cardiac tamponade. (Note: an RBBB pattern is typically seen with left ventricular pacing, since, as with the RBBB, the left ventricle is activated first. However, RBBB can be seen when a lead is placed apically in the RV and does not necessarily indicate perforation.) Hemodynamic deterioration may occur at the time of perforation, but a “slow” pericardial leak may also arise, and symptoms may not appear for 24–48 h. Delayed perforation, i.e., > 1 month, although rare, has also been described.

If the patient has mild symptoms or signs compatible with lead perforation, such as pericardial pain and friction rub, but a persistent perforation cannot be identified, observation is reasonable. If the symptoms or signs resolve within 24–48 h, lead repositioning may not be necessary. If an echocardiogram reveals a small pericardial effusion but no definite perforation, serial echocardiograms should be obtained to be certain that the effusion is not hemodynamically significant or enlarging.

Management of lead perforation depends, in part, on the clinical sequelae. Perforation associated with hemodynamic compromise must be dealt with as an...
emergency. If clinical and echocardiographic findings are consistent with tamponade, echocardiographically guided pericardiocentesis should be performed. Usually, placing an indwelling pigtail catheter is reasonable to avoid recurrent hemodynamic compromise and to measure drainage accurately. If neither significant additional drainage nor reaccumulation by echocardiographic imaging occurs, the catheter can be removed in 48–72 h and the patient managed by observation and reimaging. If no reaccumulation occurs, the leads may not have to be repositioned so long as thresholds remain stable. Any significant rise in threshold necessitates lead withdrawal and repositioning. Any time a lead suspected of perforation is withdrawn, there is the potential for pericardial bleeding. In our institution we do this in our usual pacemaker implant suite with the echocardiographer and equipment necessary for echocardiographic-guided pericardiocentesis standing by. Others prefer to pull back the lead suspected of perforation in an operating room with a cardiac surgeon on standby.

Another late complication of lead perforation occurs at the time of lead extraction if required. As discussed above, lead perforation is probably more common than realized, and patients may do well over the long term unless lead extraction is required. If there has been transmyocardial perforation or perforation/dissection of the venous system with re-entrance into the circulation at the time of implant, catastrophic results may occur when trying to extract these leads either with traction or a laser extraction system. Careful review of the patient’s history at the time of implant (however remote) and imaging data acquisition when questions arise just prior to performing lead extraction are recommended.

Perforation of the great vessels may also occur as a procedural complication. Management would be dependent on patient symptoms and hemodynamic stability (Fig. 6.9).

Pericarditis

Pericarditis, as mentioned, may be a presenting symptom of perforation. However, pericarditis may occur with or without any other clinical evidence of perforation.

Fig. 6.8 Twelve-lead electrocardiogram obtained immediately after VVI pacemaker implantation. The paced ventricular complex has a right bundle branch block configuration compatible with left ventricular lead placement.

Pericarditis

Fig. 6.9 Still-frame fluoroscopic image obtained at the time of upgrade from a dual-chamber pacemaker to a CRT-D system. As the new ventricular lead was passed through the venous system, the lead perforated, probably at the level of the superior vena cava. Because of uncertainty of the lead position, a contrast injection was performed and contrast is apparent in the mediastinum. The patient remained asymptomatic and hemodynamically stable. The lead was withdrawn and the procedure successfully completed.
tion. It is possible for the tip of an active fixation lead to irritate the pericardium, most commonly a right atrial active fixation lead.\textsuperscript{16,17} If there is no evidence of tamponade or symptomatic pericardial effusion, it is reasonable initially to treat the patient conservatively, i.e., observation and pain medications. Anti-inflammatory medications, e.g., nonsteroids or steroids, may relieve symptoms. However, if the medications cannot be withdrawn without symptom recurrence, it may be necessary to remove and reposition the lead.

**Arrhythmias**

A frequent complication during lead implantation is development of supraventricular or ventricular arrhythmias related to lead manipulation. These effects are usually transient, ending promptly when the lead position is changed. Rarely, they may be sustained. Atrial manipulation may rarely result in sustained atrial tachycardia, fibrillation, or flutter, which complicates placement of a permanent atrial lead. Atrial tachycardia or flutter may revert to normal sinus rhythm with gentle manipulation of the electrode against the atrial wall or by overdrive pacing. Commonly used pacing system analyzers have a “temporary” overdrive pacing mode available that allows rapid pacing. If the patient is in atrial tachycardia or flutter, burst overdrive pacing via the pacing system analyzer may interrupt the tachyarrhythmia and restore normal sinus rhythm.

Management of atrial fibrillation is more difficult and may require cardioversion to restore normal sinus rhythm during the implant procedure. Prior to cardioversion, the patient’s arrhythmia history and anticoagulation history should be reviewed to be certain that cardioversion is safe. We routinely place transcutaneous “pacing pads” which can be used for cardioversion in the event that cardioversion is necessary. Moreover, we routinely perform device implantation in anticoagulated patients, as long as the international normalized ratio (INR) is ≤2.5. Patients who are not anticoagulated and who have been in atrial fibrillation or flutter for >48 h are generally not cardioverted due to the potential risk of thromboembolism and stroke.\textsuperscript{16} Brief ventricular arrhythmias are also common, particularly during ventricular lead manipulation. They are usually easily controlled. However, in patients with a history of spontaneous sustained ventricular tachycardia, manipulation of the lead may initiate ventricular tachyarrhythmias. Occurrence is obviously more likely during implantation of an ICD. For this reason, all pacemaker and ICD recipients are monitored, and life-support equipment and an external defibrillator are immediately available.

Ventricular extrasystoles may occur in the early post-implantation period as a result of irritation at the electrode–myocardium interface. These premature beats, termed “tip extrasystoles,” are usually of the same morphology as the paced ventricular beat (Fig. 6.10). They usually subside within 24 h after implantation and rarely, if ever, require treatment.

In addition to tachycardia, bradyarrhythmias may occur during implant. In patients with intermittent atrioventricular (AV) block and left bundle branch block, catheter trauma to the right bundle may result in AV block. More commonly, bradycardia results from overdrive suppression of an escape ventricular focus during threshold testing. In a patient at high risk for development of asystole or complete heart block during the procedure, a temporary pacemaker may be placed before implantation at the discretion of the implanter. Alternatively, external-pacing pads can be placed during the procedure should temporary pacing be needed, and this method may obviate adjunctive transvenous temporary pacing. At our institution we routinely place external pacing pads.

**Pulse generator pocket**

Because local ecchymoses are common after pacemaker implantation, an ecchymosis, regardless of size, that is not expanding is treated by observation only. Ecchymoses occur particularly in patients receiving anticoagulants or antiplatelet agents. Aspirin and other inhibitors of platelet aggregation, specifically drugs such as clopidogrel, are not discontinued prior to device implantation.
implantation but may result in substantial bruising and bleeding. Careful local hemostasis is essential, and the patient should be told that bruising may be more substantial due to platelet inhibitors, aspirin, or warfarin.

In patients requiring oral anticoagulants (warfarin), we like the INR to be $\leq 2.5$ at the time of implantation. For most patients this may not require any alteration in therapy. If the patient is maintained at a higher INR, holding the warfarin for one to two nights prior to implantation will result in the INR being at acceptable levels to proceed.

As noted previously in this chapter, unfractionated heparin or low-molecular-weight heparin are always discontinued prior to device implant and ideally avoided for a minimum of 24 h post implantation.

For any patient, whether they are anticoagulated or on platelet inhibitors, or not, who seems to have excessive “oozing” within the pocket, special steps can be considered at the time of implant. Materials that can be used in patients with excessive oozing that cannot be stopped with electrocautery include Gelfoam™, thrombin-treated biodegradable mesh, and topical application of thrombin, which can be highly effective in stopping the bleeding. If topical thrombin is used, it may interfere with subsequent INR measurements.

Discrete hematoma formation at the site must be dealt with on the basis of its secondary consequences (Figs. 6.11 and 6.12). If bleeding continues, pain cannot be managed with mild analgesics, or the integrity of the incision is threatened, evacuating the hematoma should be considered. Aspiration of the hematoma should not be attempted, because it is often ineffective, and regardless of the care taken to maintain sterile technique, aspiration probably increases the risk of introducing an infection.

Administration of anticoagulants can be resumed within 48–72h after implantation if there is no evidence of substantial hematoma formation. Should a substantial hematoma occur, conservative treatment is preferred if possible. Needle aspiration or placement of a drain should be avoided to minimize the risk of infection. If evacuation of the hematoma is required to manage local pain or stop progression because of a threat to the integrity of the incision, the procedure should be thoroughly sterile.

Late complications, including erosion and migration, are often the result of suboptimal initial surgery or infection (Figs. 6.13 and 6.14). These can be minimized by careful technique at the time of initial pacemaker implantation and by the formation of an adequate pocket. Also, a painful pocket, as previously discussed, may result from inadequate positioning of the pacemaker below the subcutaneous tissues, and the pulse generator may have to be repositioned.

Pain

Patients should be told to expect some local discomfort at the pacemaker implantation site. This gradually subsides and can usually be managed with mild analgesics, such as acetaminophen. For several reasons, a patient could experience a painful pacemaker site, commonly called a “painful pocket,” and the complaint should be taken seriously. The differential diagnosis includes:

- Infection
- Pacemaker implanted too superficially
- Pacemaker implanted too laterally
- Pacemaker allergy.
An indolent infection may be signaled by a painful pocket long before any other signs of infection. This diagnosis may be difficult. Needle aspiration of a pacemaker site that is not obviously infected is not advised for fear of introducing infection. However, if a painful pocket is explored for any reason, specimens for culture should be obtained at that time.

The pacemaker pocket should be formed in the prepectoralis fascia, i.e., deep to adipose tissue in the subcutaneous space. If it is placed anterior to the adipose layer, i.e., within subcutaneous tissues, significant pain may result. This is one of the most common causes of a painful pocket and justifies revision of the pacemaker pocket.

If the pacemaker is positioned too laterally, impingement on the axillary space may cause discomfort (Fig. 6.15). Although there are published series on axillary pocket placement, substantial experience...
is required to position the pacemaker in such a way that there is no discomfort.

Allergic reaction to the pacemaker can or other components of the pacing system is a rare but reported complication. Pain at the pocket site may occur if the allergic reaction is to the pacemaker can or other component located within the pocket site. Proof of such an allergy requires sophisticated allergy testing, and correction of the problem may require changing certain components of the hardware. Some of the instances of “allergy” are, in reality, low-grade infections, which should be treated as infections rather than allergies. No

![Fig. 6.14 Erosion of a previously abandoned and capped lead.](image)

![Fig. 6.15 Posteroanterior radiograph from a patient with chronic pain and arm limitation after pacemaker implantation. An attempt had been made to place the pacemaker in an axillary position for cosmetic reasons.](image)

From this single view it also appears that both leads are “shallow,” i.e., suboptimal redundancy on the ventricular lead and suboptimal “J” on the atrial lead.
diagnosis of allergy should be made until infection has been ruled out.

Pacing system components to which there have been documented allergic reactions include: titanium, poly-chloroparaxylene, nickel, polyurethane, epoxy, mercury, cadmium, chromate, silicone and cobalt. If allergy is a real consideration in a patient with a painful pocket or other symptoms that may suggest an unusual allergic reaction, the manufacturer of the patient’s pulse generator and lead(s) should be contacted and a testing kit requested. The manufacturer should be able to provide a sample of all components that a dermatologist or allergist should be able to use for skin testing.

**Inadvertent left ventricular lead placement**

Inadvertent placement of the transvenous lead in the left ventricular cavity is not uncommon. This occurrence is most likely when a lead is passed across an atrial or ventricular septal defect (VSD) that is not known to exist (Fig. 6.16). It can also occur by inadvertent puncture and cannulation of the subclavian artery (Fig. 6.17A–C). A left-sided position of the lead can be suspected from an unusually high “takeoff” of the ventricular lead, that is, it begins to pass to the left side of the heart at a point higher than the lowermost portion of the atrial J. If lateral fluoroscopy or lateral chest radiography is done, the left ventricular position is fairly obvious because the lead is directed posteriorly.

The right anterior oblique (RAO) and left anterior oblique (LAO) fluoroscopic views performed either at the time of implant or requested at the time of later chest X-ray can be extremely useful in understanding the exact route of a ventricular pacing lead, especially when systemic circulation pacing is a concern. In the LAO projection, it becomes immediately evident whether or not the lead tip is located on the right side of the cardiac silhouette, and at implant excessive mobility of the pacing location. When the pacing lead in the RAO projection is not related to the region of the posterior fat pad/diaphragm, then either an ASD or VSD (or equivalent) is present. The distinction between these two entities can again be made with a quick perusal of the RAO view. ASDs will be found closer to the vertebral column (atrial) and VSDs closer to the sternum (ventricular). With myocardial perforations in either the RAO or LAO view, the lead will be seen traversing outside the cardiac silhouette, and at implant excessive mobility of the lead following perforation is characteristic.

The concern with left ventricular lead placement is the potential for thromboemboli. Small thromboemboli arising from the pacing leads on the right side of the heart are probably common, but are rarely of clinical significance. Conversely, a small thromboembolus in the systemic circulation could be catastrophic. Therefore, a lead in the systemic circulation is of clinical concern. If such a position is realized within the first few days after implantation, the lead should be withdrawn and repositioned if the patient does not have a right-to-left shunt across the defect that allowed the lead to cross. With a left atrial lead, epimyocardial lead placement is recommended.

If left ventricular lead positioning is not recognized in the very early post-implant period, it is not likely to be realized for some time. If months have passed, the approach must be individualized for the patient. If the lead is to be left in the system circulation, the patient should receive anticoagulation with warfarin and be told of the potential risk of embolic phenomena. Lead extraction can be considered, although controversy exists. Because of the potential for embolization of small clots during extraction, some physicians opt for removal of the leads only during an open chest approach. Those who are experts in extraction procedures believe that the risk of emboli is small and proceed with standard extraction techniques. All options should be discussed with the patient.

**Thrombosis**

Thromboembolic complications after permanent pacemaker implantation are uncommon, estimated to occur in 0.6–3.5% of implants. If thrombosis involves the superior vena cava, axillary vein, or area around the pacemaker lead in the right atrium or RV, several problems can develop (Fig. 6.18). These include occlusion of the superior vena cava and superior vena cava syndrome; thrombosis of the superior vena cava, right atrium, or RV, with hemodynamic compromise or pulmonary em-
bolism; and symptomatic thrombosis of the subclavian vein with an edematous painful upper extremity.

Partial or silent thrombosis is common and is usually clinically insignificant except at the time of pacing system revision; an alternative venous route may be required. Venoplasty has been used when partial thrombosis limits venous access and a new lead must be placed (Fig. 6.19). Venoplasty may also be performed in a coronary vein if a stenosis limits coronary sinus lead placement (Fig. 6.20).

If the patient presents with symptomatic venous thrombosis, several therapeutic approaches can be

Fig. 6.16 (A) Posteroanterior and (B) lateral chest radiograph obtained the day after pacemaker implantation. The lead has a “high takeoff” as it begins to cross to the left from the atrial position. This lead had been passed across an unknown patent foramen ovale and positioned in the left ventricle. (C) Posteroanterior and (D) lateral chest radiograph obtained the day after the lead had been withdrawn and repositioned in the right ventricular apex.
considered. The most common presentation is a mildly edematous arm and complaints of “aching” or a “heavy” sensation in the arm. Conservative treatment with bed rest, arm elevation, and intravenous heparin often results in relief of symptoms. There are reports of thrombolytic therapy for symptomatic thrombosis after device implantation. Although this method may work well, the patient should be advised that there is some risk of bleeding within the pocket if the procedure had been recently performed. Whether long-term anticoagulation is of benefit in patients with subclavian thrombosis is controversial. Although the information available is anecdotal only or consists of single case reports, we favor the use of warfarin for approximately 3 months after initial treatment with unfractionated heparin or low-molecular-weight heparin. In the patient with more extensive thrombosis, such as superior vena cava syndrome, other interventions may be required (Fig. 6.21).

Fig. 6.17 Posteroanterior (A) and lateral (B) chest radiograph of a patient with a single-chamber pacing system. Note that the lead takes an unusual course and remains to the left of the vertebral column and on the lateral image the lead is in a shallow ventricular position and oriented slightly posteriorly. (C) Still-frame from a 2D-echocardiogram of the same patient. The pacing lead (PM lead) noted by the arrow goes through the aortic (AO) valve and into the left ventricle (LV). The lead had been inadvertently placed by subclavian artery puncture.

Fig. 6.18 Venogram from a patient with an abandoned pacing lead on the left and a functional but failing pacemaker lead through the left subclavian vein. Extensive thrombosis is present in the subclavian vein with bilateral innominate vein occlusion.
Fig. 6.19  (A) Initial venogram reveals high-grade stenosis of the left innominate vein and large bridging collateral venous channels around the area of stenosis (arrow). (B) Venogram after venoplasty shows large opening in area of previously noted stenosis (arrow). Dilation was sufficient to allow passage of the pacemaker lead. (From Spittell et al.21 By permission of Futura Publishing Co.)

Fig. 6.20  Images obtained during coronary venous lead placement. (A) Initial coronary venogram reveals significant venous stenosis and inability to pass pacing lead. (B) Venoplasty performed and lead advanced without difficulty. (Figures courtesy of Seth J. Worley, Lancaster, PA, USA.)
Loose connector block connection

Intermittent or complete failure of output can occur because of a loose connection at the pacing lead–connector block interface. This failure usually occurs because the lead was inadequately secured at the time of pacemaker implantation. When there is a loose connection, manipulating the pacemaker may reproduce the problem. The poor connection may be evident radiographically (Fig. 6.22).

Lead damage

Lead damage during pacemaker implantation may be more common than is recognized. Pacing leads are easily cut by scissors or scalpel, although the damage may not be recognized. Polyurethane leads can be easily damaged by placement of a ligature directly around the lead itself. To secure the lead, the protector sleeve provided on most polyurethane leads or a “butterfly” sleeve that can be secured around the lead and then to the underlying support structures should be used (Figs. 6.23 and 6.24).

Rarely, the lead may be damaged by the stylet during implantation, that is, the stylet may be forced at an angle through the conductor and the surrounding insulating material. If this is recognized during the procedure, the lead should be removed and discarded.

Skin adherence

Adherence of the pulse generator to the skin strongly suggests an infection, and salvage of the site may not be possible. Impending erosion (skin thinned to the point of transparency) should be dealt with as an emergency. Once the skin is broken, infection is virtually certain; while it is still closed, the pacemaker is protected. If revision is accomplished before the pacemaker has fully eroded and become contaminated, the original pacemaker can be reimplemented if infection is not present. In this situation, the original site can be successfully revised and reused. Culture specimens should be obtained in all such circumstances.

Erosion

Although erosion of the pulse generator through the skin usually occurs long after implantation, it is most often related to the implantation technique (Figs. 6.13 and 6.14). Erosion is an uncommon complication that may occur in five situations:

• The patient has an indolent infection
• The pacemaker pocket formed at the time of surgery is too small for the implanted pulse generator
• The pulse generator is implanted too superficially, especially in children and small-framed adults, in whom lack of adipose tissue results in “tightness” of the pacemaker despite adequate pocket size
• The generator is implanted too far laterally in the anterior axillary fold
• The lead has been sutured to the subcutaneous or subcuticular tissue.

Infection is the most common cause of erosion, and the other causes are uncommon by comparison. When pacemaker erosion occurs, the only choice is surgical revision of the pacemaker site. If erosion is associated with infection, the entire system (both pulse generator and lead) must be removed and a completely new pacing system implanted at a clean site. It may be possible to revise the pacemaker site, enlarge the pocket, and fashion a satisfactory skin flap. Revision can be undertaken only if there is no infection. Infection may be present even without purulent material; therefore, cultures should be done and results proven negative before pocket revision.

Infection

The incidence of infection after pacemaker implantation...
Fig. 6.22  (A) Close-up of the pacemaker on a posteroanterior chest X-ray. Note that the atrial lead (top lead) is not completely inserted into the connector block. Comparing it with the ventricular lead, where the pin is visible coming through the connector block, the pin is not visible on the atrial lead. (B) The corresponding tracing shows substantial "noise" on the atrial lead, which is common if the lead is not secure in the connector block. (C) Photograph obtained at the time of revision. Again, the pin of the ventricular lead can be seen through the connector block, but the atrial pin cannot.
tion should certainly be < 2% and in most series has been < 1%. In the PASE trial, erosion and pocket infection occurred in 0.5% of patients. Careful attention to surgical details and sterile procedures is of paramount importance in avoiding pacemaker site infection. In a retrospective study to determine risk factors for developing device infection, the multivariable logistic regression model identified long-term corticosteroid use

Fig. 6.23 Close-up view from a posteroanterior radiograph shows a pacemaker and the proximal portion of a lead. At two sites (arrows), the insulation is compressed by ligatures placed around a securing sleeve. (Hayes DL: Pacemaker radiography. In A Practice of Cardiac Pacing. Edited by S Furman, DL Hayes, DR Holmes Jr. Mount Kisco, NY, Futura Publishing Company, 1989, pp 323–368. Copyrighted and used with permission of Mayo Foundation for Medical Education and Research.)

Fig. 6.24 An explanted lead. There is evidence of the contour of the lead being permanently altered by the ligatures placed on the sleeve, not on the lead directly. However, it cannot be determined from the photograph that there was evidence of loss of integrity of the lead insulation.
and the presence of more than two pacing leads vs. two leads as independent risk factors for pacemaker infection. (Although this study was restricted to patients undergoing pacemaker implantation, it is reasonable to think that these risk factors apply to other cardiac device implantations as well.) In the same study it was found that use of antibiotic prophylaxis prior to permanent pacemaker implantation had a protective effect. As noted in Chapter 5, Implantation Techniques, we do use antibiotic prophylaxis at the time of implant and details are described in Table 5.4.

Pacemaker infection must be recognized and be treated properly. It may appear as:

- Local inflammation and abscess formation in the area of the pulse generator pocket
- Erosion of part of the pacing system through the skin with secondary infection
- Fever associated with positive blood cultures with or without a focus of infection elsewhere.

The most common clinical presentation is infection around the generator; septicemia is an uncommon mode of presentation. Early infections are most commonly caused by *Staphylococcus aureus*, are aggressive, and are often associated with fever and systemic symptoms. Late infections are frequently caused by *S. epidermidis* and are more indolent, usually without fever or systemic manifestations. Treatment for both organisms almost always requires removal of the entire infected pacing system, pulse generator, and leads. Other organisms may be involved in either early, i.e., <4 weeks post procedure, or late, i.e., >4 weeks post procedure, infections.

In a retrospective review of all patients with infections of implanted cardiac devices at our institution, generator pocket infection (69%) and device-related endocarditis (23%) were the most common clinical presentations of infection. Coagulase-negative staphylococci and *S. aureus*, in 42% and 29% of cases, respectively, were the leading pathogens for infections of implanted cardiac devices, although multiple pathogens were identified (Fig. 6.25). Most patients (98%) underwent complete device removal. Duration of antibiotic therapy after device removal was based on clinical presentation and causative organism (median duration of 18 days for pocket infection vs. 28 days for endocarditis; 28 days for *S. aureus* infection vs. 14 days for coagulase-negative staphylococci infection [P > 0.001]) (Fig. 6.26).

Just as it is important to remember that infection may be strongly clinically suspected even when cultures of the pocket are sterile, it is also true that routine cultures of the pocket may also be misleading. Commensals as well as known pathogens may be frequently isolated in the absence of any known, or suspected, infection.

An algorithm for management of the patient with an infected pacing or ICD system is shown in Figure 6.27. Our guidelines for the diagnosis and management of device infections are listed in Table 6.2.

### Abandoned, nonfunctioning and noninfected leads

Whether abandoned, non-functioning, and non-infected leads should be extracted is controversial. Abandoned leads must be removed if they are part of an infected system. If the pacing system is infected, it is essential that the entire lead be removed. In other situations, extraction may be desirable. Specific situations include:

![Fig. 6.25 Microbiology of PPM/ICD infections. ICD, implantable cardioverter-defibrillator; PPM, permanent pacemaker. (With permission of: Sohail MR, Uslan DZ, Khan AH et al. Risk factor analysis of permanent pacemaker infection. Clin Infect Dis 2007; 45:166–73.)](image-url)
Tricuspid regurgitation felt to be due to or exacerbated by multiple leads across the valve (Fig. 6.28). The mechanisms of tricuspid regurgitation can vary. In a retrospective review of patients with implanted devices that underwent surgery for tricuspid regurgitation, the mechanism of the regurgitation in 41 patients was: lead impingement of the valve in 16 patients; lead adherence to the valve in 14 patients; valve perforated by the lead in seven patients; and lead entanglement in four patients.\(^{27}\)

- Symptomatic thrombosis\(^{28}\)
- When the abandoned lead(s) is an impediment to placement of a new pacing lead
- When there is an interaction between abandoned and active lead(s), e.g., the functioning lead senses "noise" from contact with the abandoned lead
- Pediatric patients in whom multiple lead changes will be required and in whom abandonment would result in excessive hardware.

If none of these conditions exists, it is possible to abandon leads in place without compromise to the patient (Fig. 6.29).\(^{29}\)

At the time of lead extraction, a portion of the lead, specifically, portions of the "tines," may be left in an endocardial position. A distal electrode also may not be removed and be left somewhere within the vascular tree (Fig. 6.30). A clinical problem usually does not result from these retained fragments. In a retrospective study of ICD recipients, we found that capping and abandoning leads does not generally affect sensing function or defibrillation thresholds.\(^{30}\)

**Twiddler’s syndrome**

Purposeful or absent-minded "twiddling"—manipulation of the pulse generator by the patient—has been named “twiddler’s syndrome.” Manipulation may cause axial rotation of the pacemaker, twisting of the lead, and eventual fracture or dislodgment of the lead. The syndrome commonly occurs when the pacemaker sits loosely in the pacemaker pocket (Fig. 6.31), either because the pocket is too large or because the pacemaker has migrated. Obesity may also predispose patients to this complication.

If the problem has occurred because of pacemaker migration or a poorly fashioned pacemaker pocket, the pocket should be revised. Avoiding the creation of an excessively large pocket, fixing the pulse generator by an anchoring suture, or anchoring the lead to the prepectoral fascia by a sleeve may prevent this problem, or placing the pulse generator in a subpectoral position. Another technique that is more of historical interest is to place the pacemaker in a snugly fitting Dacron pouch to reduce migration and torsion of the pacing system by promoting tissue in-growth and stabilization of the pacemaker. Although these Dacron pouches are still available, they are rarely used.

**New symptoms secondary to pacemaker placement**

**Extracardiac stimulation**

Extracardiac stimulation usually involves the diaphragm, pectoral muscle, or less commonly the intercostal muscles. Diaphragmatic stimulation may be
A

Suspected PPM/ICD infection

Blood and generator pocket cultures

Positive blood cultures or prior antibiotic treatment

TEE

Valve vegetation

Lead vegetation

Follow AHA guidelines for treatment of infective endocarditis*

Complicated, i.e. with septic venous thrombosis, osteomyelitis, etc.

Uncomplicated

Other

S. aureus

Treat with 4–6 weeks of antibiotics*

Treat with 2 weeks of antibiotics*

Treat with 2–4 weeks of antibiotics*

Negative blood cultures

Pocket infection

Generator/lead erosion

Treat with 10–14 days of antibiotics*

Treat with 7–10 days of antibiotics*

Reimplant once adequate debridement is achieved

Fig. 6.27 Mayo Clinic algorithm of cardiac device infection management. (A) Treatment algorithm based on blood and generator pocket cultures. This algorithm applies only to the patients with complete explantation of the implanted system. (* Duration of antibiotics should be counted from the day of device explantation.) (B) Algorithm for reimplantation of new pulse generator. (With permission from Sohail MR, Uslan DZ, Khan AH et al. Management and outcome of permanent pacemaker and implanted cardioverter-defibrillator infections. JACC 2007; 49:1851–9.)
due to direct stimulation of the diaphragm (usually stimulation of the left hemidiaphragm) or stimulation of the phrenic nerve (usually stimulation of the right hemidiaphragm). Diaphragmatic stimulation is particularly problematic with leads placed via the coronary sinus for left ventricular stimulation, due to the course of the left phrenic nerve along the left lateral cardiac border.

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Table 6.2 Mayo Clinic guidelines for the diagnosis and management of cardiac device infections

1. All patients should have at least two sets of blood cultures drawn at initial evaluation
2. Generator tissue, Gram stain and culture and lead tip culture should be obtained
3. Patients who either have positive blood cultures or have negative blood cultures but had recent antibiotics before obtaining blood cultures should have a transesophageal echocardiogram (TEE) to assess for device-related endocarditis
4. Sensitivity of TTE is low and is not recommended to evaluate for device-related endocarditis
5. Patients with negative blood cultures and recent antibiotics and valve vegetations on TEE should be managed in consultation with an infectious diseases expert
6. All patients with device infection should undergo complete device removal, regardless of clinical presentation
7. A large (>1 cm) lead vegetation is not a stand-alone indication for surgical lead removal
8. Blood cultures should be repeated in all patients after device explantation. Patients with persistently positive blood cultures should be treated for at least 4 weeks with antimicrobials even if TEE is negative for vegetations or other evidence of infection
9. Duration of antimicrobial therapy should also be extended to ≥4 weeks in patients with complicated infection (endocarditis, septic venous thrombosis, osteomyelitis, metastatic seeding)
10. Adequate débridement and control of infection should be achieved at all sites before reimplantation of a new device
11. Reevaluation for continued need of the device should be performed before new device placement
12. If an infected cardiac device cannot be removed, then long-term suppressive antibiotic therapy should be administered after completing an initial course of treatment and securing a clinical response to therapy. An infectious diseases expert opinion should be sought

The potential for diaphragmatic stimulation should be tested at the time of implantation. If any stimulation is noted with 10 V, the pacing lead should be repositioned. Because this testing is usually accomplished with the patient in a supine position, it does not eliminate the possibility of diaphragmatic stimulation when the patient is upright. Diaphragmatic stimulation occurring during the early postimplantation period may be due to either microdislodgment or macrodislodgment of the pacing lead or myocardial perforation. Stimulation may be diminished or alleviated by decreasing the voltage output or the pulse width. (An adequate pacing margin of safety must be maintained after the output settings are decreased.) Local muscle stimulation occurs much more commonly with unipolar than with bipolar pacemakers and is usually noted in the early postimplantation period.

In CRT devices pectoral muscle stimulation may be obviated by programmable change in pacing configuration. When a bipolar coronary sinus lead is utilized, multiple CRT systems offer an option by which multiple pacing configurations are programmable. This may be helping not only in identifying a lower pacing threshold, but also in avoiding phrenic nerve stimulations. In one study, phrenic nerve stimulation occurred in 12% of patients that had a device with a programmable pacing configuration and was correctable by a change in pacing configuration in all patients.31 Pectoral muscle stimulation may also be due to an insulation defect of the pacing lead, current leakage from the connector or sealing plugs, erosion of the pacemaker’s protective coating, or rapid high-amplitude atrial output in a unipolar dual-chamber pacemaker. If the problem is due to an insulation defect on either a unipolar pacemaker or the pacemaker lead, decreasing the voltage output or the pulse width (or both) may minimize the stimulation, but the defective portion of the system may have to be replaced. (If an activity-sensing rate-adaptive pacemaker is in place, muscle stimulation may result in sensor activation and inappropriately rapid pacing rates for a given level of activity.) Pectoral muscle stimulation is less common with bipolar than with unipolar pacemakers. If pectoral muscle stimulation occurs in a polarity-programmable pacemaker, reprogramming to the alternate polarity configuration may alleviate the problem.

Fig. 6.30 Posteroanterior chest radiograph (A) and close-up view (B) of a retained lead fragment, the distal electrode, after partial lead extraction. The fragment is wedged in an infraclavicular portion of the subclavian vein. The retained fragment has not produced long-term complications.
Fig. 6.31 (A) Posteroanterior (PA) chest radiograph from a patient with an earlier generation internal cardioverter-defibrillator. The X-ray was obtained the day after implant. (B) PA chest radiograph from the same patient obtained at the 3-month follow-up visit. Note that lead positions are now more shallow, and there is gross twisting of the leads above the pulse generator. This is consistent with “twiddler’s syndrome” regardless of the etiology of the lead entanglement.
Pacemaker syndrome
Pacemaker syndrome, described in Chapter 2, implies adverse hemodynamics associated with loss of AV synchrony. Pacemaker syndrome can occur as an implant or hardware-related complication if the atrial lead becomes nonfunctional for any reason and results in loss of AV synchrony. A classic example would be dislodgment of the atrial lead so that it either fails to capture or captures the ventricle; in either event, the patient has functional ventricular pacing only. However, the presence of a pacemaker does not diagnose a hardware-related complication, and it can occur with a technically appropriately functioning system.

Battery depletion
Battery depletion is expected, because the power supply of a pulse generator is consumable, and should not be considered a complication in most patients. Indeed, battery depletion is the most common cause of pulse generator removal (see Chapter 10, Troubleshooting, Figs 10.6 and 10.7). If the pulse generator displays end-of-life characteristics much earlier than expected, other potential problems should be explored. Early battery depletion may be due to inappropriate programming of unnecessarily high output; excess current drain caused by a loss of lead integrity; or internal current loss due to pulse generator component malfunction. The manufacturer should also be consulted for data on performance of the pulse generator, i.e., pulse generator longevity predicted or observed in other patients.

From a pacemaker registry at 8 years of prospective follow-up, battery depletion was the most common cause of pulse generator removal. Of the pulse generators displaying battery depletion indicators, 95% exhibited normal elective replacement, i.e., >3 years post implant. In the remaining 5% of patients, depletion occurred at <3 years post implant and was designated as “severe” battery depletion. These depletions presented primarily as loss of telemetry or no or low output.

If battery depletion is advanced, it may not be possible to program the pacemaker (see Chapter 10, Troubleshooting). At other times, attempting to program a pacemaker at an advanced stage of battery depletion may result in sudden complete loss of output (Fig. 6.32).

Implant or hardware-related complications that may result in recurrence of preimplantation symptoms (see also Chapter 10, Troubleshooting)

Loss of circuit integrity
Any abnormality that can permanently or intermittently interrupt the integrity of the pacing circuit can allow recurrent bradycardia and therefore recurrence of symptoms. Likewise, interruption of the circuit in an ICD can result in recurrent bradyarrhythmia or tachyarrhythmia, and circuit interruption in a CRT system could result in the lack of biventricular pacing and recurrent symptoms. This can occur with fracture of the lead conductor coil, breach of lead insulation, defect in a lead adaptor (Fig. 6.33), or loose connection where the lead pin joins the connector block (Fig. 6.22). Failure of the pacemaker circuitry, which would also allow recurrent bradycardia, is extraordinarily rare unless the pacemaker is exposed to some external source. For example, exposure to a strong electrical source, such as defibrillation, can result in circuit failure.

A component failure is a diagnosis of exclusion. In this situation the specific problem may not be clear until the device has been removed, returned to the manufacturer, and subjected to destructive analysis. Determining

Fig. 6.32 Electrocardiographic tracing from a patient with a 10-year-old pacemaker and failure to capture. The pacemaker was nearing total battery depletion and was generating insufficient voltage to maintain capture.
Fig. 6.33 Posteroanterior chest radiograph (A) and close-up (B) from a patient in whom a “Y-adaptor” has been used to adapt two unipolar epicardial leads to an in-line bipolar lead. The arrow on the close-up notes a defect (fracture) of the Y-adaptor which led to intermittent failure to pace.
CHAPTER 6 Implantation-related Complications

229

the cause of system malfunction is discussed extensively in Chapter 10, Troubleshooting.

Exposing a pacemaker, ICD, or CRT to therapeutic radiation may also result in unpredictable component failure and a “runaway” or “sudden no output” response. A pacemaker in or very near the field of therapeutic radiation should be moved to avoid damage to the circuitry and to prevent compromise of the field as defined by the radiation oncologist. This situation is most common in a female patient with a malignant tumor of the breast on the same side as the pacemaker or ICD. The simplest and least invasive approach is to explant the device, form a new pocket on the contralateral side, and tunnel the leads subcutaneously to the other side. If the leads are not long enough to reach, “lead extenders” can be connected to span the additional distance (Fig. 6.34). Alternatively, the pulse generator could be removed, leads capped, and a new system placed on the contralateral side.

Lead fracture and insulation defect

Lead malfunction due to fracture or insulation defect is most commonly seen in the late postimplantation period (Fig. 6.35). Lead fractures most often occur adjacent to the pulse generator or near the site of venous access, i.e., at a stress point, although fracture has also been reported of more distal portions of the pacing lead. Although uncommon, direct trauma may result in damage to the pacing lead. When lead fracture does occur, it is usually necessary to replace the lead. If the fracture is in a bipolar lead and the pacemaker is polarity programmable, it may be possible to restore pacing by reprogramming to the unipolar configuration. This is a short-term solution and should not be a substitute for replacing the lead (Fig. 6.36A,B). In ICDs, lead fractures often manifest as inappropriate shocks due to “make-break” noise at the fracture site detected as ventricular fibrillation.

Polyurethane and silicone are used as insulating materials for most permanent pacing leads. Insulation defects in polyurethane leads have also been described at stress points; with crush injury, specifically at the costoclavicular space after placement by the subclavian puncture technique (Fig. 6.37); and at the site of ligatures, even with a suture sleeve. In bipolar coaxial leads, the insulation defect often occurs internally, in the layer of insulation between coils, rather than externally, on the outer surface.

Exit block

Exit block has been defined in several ways. The most commonly accepted clinical definition is high pacing thresholds, often progressive, that cannot be explained by radiographic dislodgment or perforation. If normal thresholds are achieved and maintained after repositioning of the lead, the term “exit block” does not
Fig. 6.35 Posteroanterior chest radiograph (A) and close-up view (B) of a fractured atrial lead. What appears to be complete fracture of the conductor coil was accompanied by failure to pace in the atrium. Atrial sensing, however, remained intact. The most likely explanation is that intact insulation and a fluid column between the fractured ends of the conductor coil allowed maintenance of sensing function.

Fig. 6.36 (A) Tracing from a patient programmed to bipolar pacing and sensing at a pacing rate of 30 ppm. There is failure to sense of the third ventricular complex. Note that this beat is not labeled by the "marker channel" because it was not sensed. This is the first sign of an insulation effect. (B) The pacemaker is reprogrammed to unipolar pacing and sensing is normal.
Fig. 6.37 Posteroanterior chest radiograph in a patient with kinking or crushing of the lead as it passes under the clavicle. The patient presented with evidence of lead-insulation failure. Close inspection of the distal tip of the lead identified three poles, and inspection of the connector block of the pulse generator also identified a third port. The third “pole” was required for a special rate-adaptive sensor. (Reproduced with permission from Hayes DL. Radiography of implantable arrhythmia management devices. In: Kusumoto F, Goldschlager N, eds. Cardiac pacing for the clinician.)
apply.) In true exit block, stimulation thresholds are often excellent at the time of implantation, but instead of the usual early rise at 3–6 weeks with a subsequent decrease and plateau, the threshold remains high. Exit block is uncommon and appears to represent an abnormality at the myocardial tissue–electrode interface. The cause is controversial. Some believe that the problem is with the lead design, and others that it is intrinsic to the patient’s myocardium, resulting in excessive reaction to the electrode. Steroid-eluting leads are often effective in preventing exit block.

With ventricular pacing leads placed either epicardially or via the coronary venous system, exit block and exit delay become even more relevant. While classical exit block as a result of the lead being in the vicinity of where disease/ischemic left ventricular myocardium occurs, excessive delay from the pacing site despite adequate thresholds will result in failure of resynchronization despite simultaneous biventricular stimulation. When exit delay or block exists, pacing at higher than normal thresholds will result in failure of resynchronization despite simultaneous biventricular stimulation. When exit delay or block exists, pacing at higher than threshold output, setting an offset for the left ventricular lead to be paced ahead of the RV lead, and possibly varying the pacing vector should be attempted before considering lead revision.

References
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CHAPTER 7

Pacemaker and Cardiac Resynchronization Timing Cycles and Electrocardiography

David L. Hayes, Paul J. Wang, Samuel J. Asirvatham, Paul A. Friedman

An understanding of the basic concepts of cardiac pacing (Chapter 1) and comprehension of the pacemaker timing cycles of cardiac pacing are fundamental before one approaches the paced electrocardiogram (ECG). This chapter begins with a detailed description of “timing cycles” and approaches the subject by pacing mode and descriptions of other features that are used with some frequency that can alter the timing cycle. The second portion of the chapter outlines assessment of the paced ECG.

Paced electrocardiography must be approached systematically, much as nonpaced electrocardiography, chest radiography or any other diagnostic procedure. Knowing the type of pacemaker, the programmed parameters, and the underlying rhythm necessitating pacing is important in interpreting the paced ECG. Obviously, this information makes the interpretation much easier, but it is frequently not available.

A sensed event occurs when a pacemaker registers cardiac electrical activity; a paced event occurs when the pulse generator delivers current to pace the heart. Paced events are followed by blanking periods, during which amplifiers are switched off and no electrical activity is registered. Blanking periods are followed by refractory periods, during which cardiac electrical activity is normally sensed, but the device’s response to the event is limited. For example, atrial events occurring during an atrial refractory period will not be tracked (i.e., followed by a ventricular paced event), but are used to determine whether mode switching should occur. Blanking and refractory periods serve to prevent oversensing of physiological signals (such as T waves) and cross-chamber sensing. There is some variation among manufacturers as to how blanking and refractory periods are defined and implemented; their role in timing cycles is discussed further below. In defibrillators and in mode switch operation in pacemakers, a series of sensed events augment counters, leading to arrhythmia detection, which may then result in therapy delivery or mode switch. Defibrillator sensing and detection are discussed in greater detail in Chapter 8, Programming.

Pacemaker timing cycles include all potential variations of a single complete pacing cycle (Table 7.1). This could mean the time from paced ventricular beat to paced ventricular beat (VV); from paced ventricular beat to an intrinsic ventricular beat (VR), whether it be a conducted R wave or a premature ventricular contraction; from paced atrial beat to paced atrial beat (AA); from intrinsic atrial beat to paced atrial beat (PA); and so forth. Various aspects of each of these cycles include events sensed, events paced, and periods when the sensing circuit or circuits are refractory or blanked. Each portion of the pacemaker timing cycle should be considered in milliseconds and not in paced beats per minute (ppm). Although it may be easier to think of the patient’s pacing rate in paced beats per
CHAPTER 7 Pacemaker and Cardiac Resynchronization Timing Cycles and Electrocardiography

minute, portions of the timing cycle are too brief to be considered in any unit but milliseconds.

If one knows the relationship between the various elements of the paced ECG, understanding pacemaker rhythms becomes less complicated. Although a native rhythm may be affected by multiple unknown factors, each timing circuit of a pacemaker can function in only one of two states. A given timer can proceed until it completes its cycle; completion results in either the release of a pacing stimulus or the initiation of another timing cycle. Alternatively, a given timer can be reset, at which point it starts the timing period all over again.

### Table 7.1 Abbreviations for native and paced events and portions of the timing cycle

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>P</td>
<td>Native atrial depolarization</td>
</tr>
<tr>
<td>A</td>
<td>Atrial paced event</td>
</tr>
<tr>
<td>R</td>
<td>Native ventricular depolarization</td>
</tr>
<tr>
<td>V</td>
<td>Ventricular paced event</td>
</tr>
<tr>
<td>I</td>
<td>Interval</td>
</tr>
<tr>
<td>AV</td>
<td>Sequential pacing in the atrium and ventricle</td>
</tr>
<tr>
<td>AVI</td>
<td>Programmed atrioventricular pacing interval</td>
</tr>
<tr>
<td>AR</td>
<td>Atrial paced event followed by intrinsic ventricular depolarization</td>
</tr>
<tr>
<td>ARP</td>
<td>Atrial refractory period</td>
</tr>
<tr>
<td>PV</td>
<td>Native atrial depolarization followed by a paced ventricular event, P-synchronous pacing</td>
</tr>
<tr>
<td>LRL</td>
<td>Lower rate limit</td>
</tr>
<tr>
<td>URL</td>
<td>Upper rate limit</td>
</tr>
<tr>
<td>MTR</td>
<td>Maximum tracking rate</td>
</tr>
<tr>
<td>MSR</td>
<td>Maximum sensor rate</td>
</tr>
<tr>
<td>PVARP</td>
<td>Postventricular atrial refractory period</td>
</tr>
<tr>
<td>RRAVD</td>
<td>Rate-responsive atrioventricular delay</td>
</tr>
<tr>
<td>VA interval</td>
<td>Interval from a ventricular sensed or paced event to an atrial paced event</td>
</tr>
<tr>
<td>VRP</td>
<td>Ventricular refractory period</td>
</tr>
</tbody>
</table>

### Pacing modes

**Ventricular asynchronous pacing, atrial asynchronous pacing, and atrioventricular sequential asynchronous pacing**

Ventricular asynchronous (VOO) pacing is the simplest of all pacing modes, because there is no sensing and no mode of response. The timing cycle is shown Fig. 7.1. Irrespective of any other events, the ventricular pacing artifacts occur at the programmed rate. The timing cycle cannot be reset by any intrinsic event. Without sensing, there is no defined refractory period. Atrial asynchr-
nous (AOO) pacing behaves exactly like VOO except that the pacing artifacts occur in the atrial chamber.

Dual-chamber, or atrioventricular (AV) sequential asynchronous (DOO), pacing has an equally simple timing cycle. The interval from atrial artifact to ventricular artifact [atrioventricular interval (AVI)] and the interval from the ventricular artifact to the subsequent atrial pacing artifact (ventriculoatrial, or atrial escape, interval, VA interval) are fixed. The intervals never change, because the pacing mode is insensitive to any atrial or ventricular activity, and the timers are never reset (Fig. 7.2).

Ventricular inhibited pacing
By definition, ventricular inhibited (VVI) pacing (also referred to as inhibited demand pacing) incorporates sensing on the ventricular channel, and pacemaker output is inhibited by a sensed ventricular event (Fig. 7.3). VVI pacemakers are refractory for a period after a paced or sensed ventricular event, the ventricular refractory period (VRP). Ventricular events occurring within the VRP do not reset the ventricular timer (Fig. 7.4). Every pacemaker capable of sensing and all defibrillators must include a refractory period in their basic timing cycles. Refractory periods prevent the sensing of early inappropriate signals, such as the evoked potential and repolarization (T wave).

Atrial inhibited pacing
Atrial inhibited (AAI) pacing, the atrial counterpart of VVI pacing, incorporates the same timing cycles, with the obvious difference that pacing and sensing occur from the atrium and pacemaker output is inhibited by...
a sensed atrial event (Fig. 7.5). An atrial paced or sensed event initiates a refractory period during which electrical signals are ignored by the pacemaker. Confusion can arise when multiple ventricular events occur while there is atrial pacing. For example, in addition to the intrinsic QRS that occurs in response to the paced atrial beat, if a premature ventricular beat follows, it does not inhibit an atrial pacing artifact from being delivered. When the AA timing cycle ends, the atrial pacing artifact is delivered regardless of ventricular events, because an AAI pacemaker should not sense anything in the ventricle. If a ventricular event is sensed by the AAI pacemaker, the event is termed far-field sensing; that is, the ventricular signal is large enough to be inappropriately sensed by the atrial lead (Fig. 7.6). In this situation, the atrial timing cycle is reset, leading to a pacing rate slower than the programmed lower rate limit (Fig. 7.6). Sometimes this anomaly can be corrected by making the atrial channel less sensitive or by lengthening the refractory period. Interpretation can be made easier in some devices when a programmable ADI mode is available. This mode is operationally the same as AAI pacing mode, but ventricular events are recorded on the diagnostic channels (Fig. 7.7).

**Single-chamber triggered-mode pacing**

Initially developed as a way to defeat the problem associated with oversensing in the inhibited demand mode, the single-chamber triggered mode (AAT, VVT) has its own unique advantages as well as disadvantages. In single-chamber triggered-mode pacing, the pacemaker releases an output pulse every time a na-
tive event is sensed (Fig. 7.8). This feature increases the current drain on the battery, accelerating its rate of depletion. This mode of pacing also deforms the intrinsic depolarization, compromising interpretation of the ECG. However, it can serve as an excellent marker for the site of sensing within a complex. It can also prevent inappropriate inhibition from over-sensing when the patient does not have a stable native escape rhythm.

**Rate-modulated pacing**

**Single-chamber rate-modulated pacing**

Single-chamber pacemakers capable of rate-modulated (SSIR) pacing can be implanted in the ventri-

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**Fig. 7.6** In this example of AAI pacing, the AA interval is 1000 ms (60 ppm). The interval between the second and the third paced atrial events exceeds 1000 ms. The interval from the second QRS complex to the subsequent atrial pacing artifact is 1000 ms. This occurs because the second QRS complex (*) has been sensed on the atrial lead (far-field sensing) and has inappropriately reset the timing cycle. LR, lower rate (limit).

**Fig. 7.7** Although an AAI pacemaker is incapable of responding to ventricular events, interpretation is easier if the ventricular events are identified. One manufacturer has a programmable ADI mode in dual-chamber pacemakers. This mode is operationally the same as AAI, but events sensed in the ventricle are recorded on the diagnostics. (Reproduced with permission from Medtronic Adapta Technical Manual.)

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**Fig. 7.8** In the VVT mode, when an intrinsic atrial event occurs a pacing artifact is delivered at the point of sensing (TP). If the lower rate limit timer is completed, a pacing artifact is delivered with paced depolarization.
cle (VVIR) or atrium (AAIR). The timing cycles for SSIR pacemakers are not significantly different from those of their non-rate-modulated counterparts. The timing cycle includes the basic VV or AA interval and a refractory period from the paced or sensed event. The difference lies in the variability of the VV or AA interval (Fig. 7.9). Depending on the sensor incorporated and the level of exertion of the patient, the basic interval shortens from the programmed lower rate limit (LRL). An upper rate limit (URL) must be programmed to define the absolute shortest cycle length allowable. Some SSIR pacemakers incorporate a fixed refractory period; that is, regardless of whether the pacemaker is operating at the LRL or URL, the refractory period remains the same. Thus, at the higher rates under sensor drive, the pacemaker may effectively become asynchronous, i.e., SOOR, since the period during which sensing can occur is so abbreviated. Native beats falling during the refractory period do not reset timing cycles.

In the VVIR pacing mode the refractory period should be programmed to a short interval to maximize the sensing interval at both the low and high sensor-driven rates. In an AAIR pacing mode, the refractory period should be programmed long enough to avoid far-field sensing and short enough to allow sensing of native atrial events at rates up to the programmed upper sensor rate. Rate-variable or rate-adaptive refractory period as a programmable option—i.e., as the cycle length shortens, the refractory period shortens appropriately, analogous to the QT interval of the native ventricular depolarization—will probably become more commonly available in subsequent generations of SSIR pacemakers.

**Rate-modulated asynchronous pacing**

If rate modulation is incorporated in an asynchronous pacing mode, the basic cycle length is altered by sensor activity. In the single-chamber rate-modulated asynchronous (AOOR and VOOR) pacing modes, any alteration in cycle length is due to sensor activity and not to the sensing of intrinsic cardiac depolarizations. In the dual-chamber rate-modulated asynchronous (DOOR) pacing mode, the pacing rate changes in response to the sensor input signal, but not to the native P or R wave.

**Atrioventricular sequential, ventricular inhibited pacing**

AV sequential, ventricular inhibited (DVI) pacing is rarely used as the programmed pacing mode of choice, but it remains a programmable option in most dual-chamber pacemakers. It is helpful to understand the timing cycles for DVI pacing.

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**Fig. 7.9** The VVIR timing cycle consists of a lower rate (LR) limit, an upper rate (UR) limit, and a ventricular refractory period (VRP, represented by triangle). As indicated by sensor activity, the VV cycle length shortens accordingly. (The shaded area represents the range of sensor-driven VV cycle lengths.) In most VVIR pacemakers, the VRP remains fixed despite the changing VV cycle length. In selected VVIR pacemakers, the VRP shortens as the cycle length shortens.
By definition, DVI provides pacing in both the atrium and the ventricle (D), but sensing only in the ventricle (V). The pacemaker is inhibited and reset by sensed ventricular activity but ignores all intrinsic atrial complexes.

The timing cycle (VV) consists of the AVI and the VA interval. The basic cycle length (VV), or LRL, is programmable, as is the AVI. The difference, VV – AV, is the VA interval. During the initial portion of the VA interval, the sensing channel is refractory. After the refractory period, the ventricular sensing channel is again operational, or “alert.” If ventricular activity is not sensed by the expiration of the VA interval, atrial pacing occurs, followed by the AVI. If intrinsic ventricular activity occurs before the VA interval is completed, the timing cycle is reset.

Atrioventricular sequential, non-P-synchronous pacing with dual-chamber sensing (DDI)

AV sequential pacing with dual-chamber sensing, non-P-synchronous (DDI) pacing can be thought of as DDD pacing without atrial tracking. As opposed to the DVI mode just described, DDI incorporates atrial sensing as well as ventricular sensing, which prevents competitive atrial pacing. The DDI mode of response in the atrium is inhibition only; that is, no tracking of P waves can occur. Therefore, the paced ventricular rate cannot be greater than the programmed LRL in the non-rate-adaptive mode. (In the DDI mode there is only one programmable rate; in the DDIR mode there would be, by definition for any sensor-driven mode, a lower rate limit and an upper sensor rate.) The timing cycle consists of the LRL, AVI, postventricular atrial refractory period (PVARP), and VRP. The PVARP is the period after a sensed or paced ventricular event during which the atrial sensing circuit is refractory. Any atrial event occurring during the PVARP will not reset timing cycles. If a P wave occurs after the PVARP and is sensed, no atrial pacing artifact is delivered at the end of the VA interval. The subsequent ventricular pacing artifact cannot occur until the VV interval has been completed; that is, the LRL cannot be violated (Fig. 7.10).

It bears repeating that because P-wave tracking does not occur with the DDI mode, the paced rate is never greater than the programmed base rate (i.e., the LRL). A slight exception to this statement may occur when an intrinsic ventricular complex takes place after the paced atrial beat (AR) and inhibits paced ventricular output before completion of the programmed AVI; that is, AR < AV. In this situation, the cycle length from A to A is shorter than the programmed LRL by the difference between the AR and the programmed AVI.

Atrioventricular sequential, non-P-synchronous, rate-modulated pacing with dual-chamber sensing

The timing cycles for non-P-synchronous, rate-mod-
ulated AV sequential (DDIR) pacing are the same as those described above for DDI pacing, except that paced rates can exceed the programmed LRL through sensor-driven activity.

**Atrial synchronous (P-tracking/P-synchronous) pacing (VDD)**

Atrial synchronous (P-tracking/P-synchronous) (VDD) pacemakers pace only in the ventricle (V), sense in both atrium and ventricle (D), and respond both by inhibition of ventricular output by intrinsic ventricular activity (I) and by ventricular tracking of P waves (T). The VDD mode is available as a single-lead pacing system. In this system, a single lead is capable of pacing in the ventricle in response to sensing atrial activity by way of a remote electrode situated on the intra-atrial portion of the ventricular pacing lead. (Single-lead VDD systems are not commonly used at this time.)

The timing cycle is composed of LRL, AVI, PVARP, VRP and URL. A sensed atrial event initiates the AVI. If an intrinsic ventricular event occurs before the termination of the AVI, ventricular output is inhibited and the LRL timing cycle is reset. If a paced ventricular beat occurs at the end of the AVI, this beat resets the LRL. If no atrial event occurs, the pacemaker escapes with a paced ventricular event at the LRL; that is, the pacemaker displays VVI activity in the absence of a sensed atrial event (Fig. 7.11).

**Dual-chamber pacing and sensing with inhibition and tracking (DDD)**

Although it involves more timers, standard dual-chamber pacing and sensing with inhibition and tracking (DDD) is reasonably easy to comprehend if one understands the timing cycles already discussed. The basic timing circuit associated with LRL pacing is divided into two sections. The first is the interval from a ventricular sensed or paced event to an atrial event. This is the VA interval. The second interval begins with an atrial sensed (SAV) or paced event and extends to a ventricular event. This interval may be defined by a paced AVI, PR interval, AR interval, or PV interval. An SAV event that occurs before completion of the VA interval terminates this interval and initiates an AVI, and the result is P-wave synchronous ventricular pacing. If the intrinsic sinus rate is less than the programmed LRL, AV sequential pacing at the programmed rate or functional single-chamber atrial (AR) pacing occurs (Fig. 7.12).

In a DDD system, a sensed or paced atrial event initiates an atrial blanking period, a ventricular blanking period, an atrial refractory period (ARP) and also initiates the AVI (Fig. 7.13). The ventricular blanking period initiated by an atrial event prevents ventricular inhibition or initiation of ventricular safety pacing as a result of sensing the atrial event. During this portion of the timing cycle, the atrial channel is refractory to any sensed events; nor will atrial pacing occur during this period. A sensed or paced ventricular event initiates a ventricular blanking period, and a VRP. (A VRP is always part of the timing cycle of any pacing system with ventricular pacing and sensing.) The ventricular blanking period prevents any sensing on the ventricular sensing channel in the period immediately following the ventricular pacing output or sensing of

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**Fig. 7.11 The timing cycle of VDD consists of a lower rate (LR) limit, an atrioventricular interval (AVI), a ventricular refractory period, a postventricular atrial refractory period (PVARP), and an upper rate limit. A sensed P wave initiates the AVI (during the AVI, the atrial sensing channel is refractory). At the end of the AVI, a ventricular pacing artifact is delivered if no intrinsic ventricular activity has been sensed, i.e., P-wave tracking. Ventricular activity, paced or sensed, initiates the PVARP and the ventriculoatrial interval (the LR limit interval minus the AVI). If no P-wave activity occurs, the pacemaker escapes with a ventricular pacing artifact at the LR limit. PV, native atrial depolarization followed by paced ventricular event; TARP, total atrial refractory period.**
an intrinsic event. The VRP prevents sensing of the evoked potential and the resultant T wave on the ventricular channel of the pacemaker. A sensed or paced ventricular event also initiates a post-ventricular atrial refractory period (PVARP) and a PVARP. (By definition the sensing circuit is “off” during a blanking period, but conceptually the PVAB as the interval immediately after the ventricular event can be thought of as the “ab-
solute refractory” portion of the PVARP.) The PVAB prevents sensing of far-field R waves and ventricular pacing events on the atrial channel. The PVARP prevents atrial sensing of a retrograde P wave (see Endless-loop tachycardia, below) and also prevents sensing of far-field ventricular events.

A dual-chamber pacemaker can track the atrial rhythm to a defined maximum tracking rate (MTR). The combination of the PVARP and the AVI forms the total atrial refractory period (TARP) (Fig. 7.14). The TARP, in turn, is the limiting factor for the maximum sensed atrial rate that the pacemaker can reach. For example, if the AVI is fixed at 150 ms and the PVARP is fixed at 250 ms, the TARP is 400 ms, or 150 ppm. In this case, a paced ventricular event initiates the 250-ms PVARP, and only after this interval has ended can an atrial event be sensed. If an atrial event is sensed immediately after the termination of the PVARP, the sensed atrial event initiates the AVI of 150 ms. On termination of the AVI, in the absence of an intrinsic R wave, a paced ventricular event occurs, resulting in a VV cycle length of 400 ms, or 150 ppm. Programming a long PVARP limits the upper rate by limiting the maximum sensed atrial rate (Fig. 7.15, top). If the native atrial rate were 151 bpm, every other P wave would coincide with the PVARP, not be sensed, and hence not be tracked, so that the effective paced rate would be approximately 75 ppm, or half the atrial rate (Fig. 7.15, bottom). Pseudo-Wenckebach behavior is explained in Fig. 7.16. The pseudo-Wenckebach interval can be quantitated by mathematical equations. Figure 7.17 schematically displays the relationship of the effective paced ventricular rate and the atrial rate.

**Portions of pacemaker timing cycles**

**Atrioventricular interval**

The AVI is often poorly understood (Fig. 7.18A). The AVI is initiated by an SAV or paced event. A ventricular blanking period, usually programmable (ranging from 12 to 125 ms), accounts for the earliest portion of the AVI. If the atrial pacing artifact were sensed by the ventricular sensing circuit, ventricular output inhibition would result. This is termed “crosstalk.” To prevent this, the leading edge of the atrial pacing artifact is masked, or blanked, by essentially turning “off” the ventricular sensing circuit refractory during the very early portion of the AVI (Fig. 7.18B). The blanking period is traditionally of short duration because it is important for the ventricular sensing circuit to be returned to the “alert” state relatively early during the AVI so that intrinsic ventricular activity can inhibit pacemaker output if it occurs before the AVI times out. The potential exists for signals other than those of intrinsic ventricular activity to be sensed and to inhibit ventricular output. Even though the leading edge of the atrial pacing artifact is effectively ignored because of the blanking period, the trailing edge of the atrial pacing artifact can at times persist beyond the blanking period so that it is sensed on the ventricular channel. In a pacemaker-dependent patient, inhibition of ventricular output by crosstalk would result in asystole. A safety mechanism is present to prevent such an outcome.

If activity is sensed on the ventricular sensing circuit in a given portion of the AVI immediately after the blanking period (the second portion of the AVI has been called the “ventricular triggering period” or the “crosstalk sensing window”), it is assumed that crosstalk cannot be differentiated from intrinsic ventricular activity. To prevent catastrophic ventricular asystole, in most pulse generators a ventricular pacing artifact is delivered early, i.e., at a AVI of 100–120 ms, although in some pacemakers this interval is programmable for 50–150 ms (Fig. 7.18C). If the signal sensed is indeed something other than a ventricular event, a paced ventricular complex at the ab-

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**Fig. 7.14** The total atrial refractory period (TARP) is the sum of the AV delay and the postventricular atrial refractory period (PVARP). (SAV = sensed AV interval.)
briefed interval prevents ventricular asystole. If, on the other hand, intrinsic ventricular activity occurs during the crosstalk sensing window of the AVI, the safety mechanism results in delivery of a ventricular pacing artifact within or immediately after the intrinsic beat. This delivery is safe because the ventricle is still refractory, so that no depolarization results from the pacing artifact, and the pacing artifact is delivered too early to coincide with ventricular repolarization or a vulnerable period. This event has been referred to as “ventricular safety pacing,” “nonphysiological AV delay,” or the “110-ms phenomenon.” The actual safety pacing duration varies from approximately 70 to 120 ms depending on the device manufacturer and model. One manufacturer does not have “safety pacing” but instead uses a “noise-rejection” interval of 40–60 ms that begins with an atrial pace but is retrig-gerable and can be retrig-gered to a maximum of the programmed AV delay, i.e., at the end of that period, even if noise is being detected and retrig-gering the noise-rejection interval, a pacing output will be de-livered.

Differential atrioventricular interval

If there is a consistent difference between AVIs initiated by a sensed event and those triggered by a paced event, the most likely explanation is a differential AVI. This is an attempt to provide an interatrial conduc-tion time of equal duration irrespective of whether the atrial contraction is paced or sensed. The PV interval initiated with atrial sensing begins at the time of atrial depolarization. Conversely, the AVI initiated with atrial

Fig. 7.15 When the sinus rate exceeds the programmed maximum tracking rate, several upper rate (UR) behaviors can occur. In the top panel, pseudo-Wenckebach behavior is seen. If a P wave occurs outside the postventricular atrial refractory period (PVARP) and is sensed, the atrioventricular interval (AVI) is initiated. However, a ventricular pacing artifact cannot be delivered at the end of the programmed AVI if this would violate the programmed maximum tracking rate. Instead, the AVI would be lengthened and the ventricular pacing artifact would occur when the maximum tracking rate had “timed out.” For example, if the maximum tracking rate is 120 ppm, or an interval of 500 ms, the AVI is 150 ms, the PVARP is 250 ms and the P wave is sensed 10 ms after completion of the PVARP, or 260 ms after the preceding ventricular event, the next ventricular pacing artifact could not be delivered for 240 ms (500–260). In the bottom panel, 2:1 UR behavior occurs when every other sinus beat falls in the PVARP. ID, intrinsic deflection. (From Hayes DL. DDDR timing cycles: upper rate behavior. In: Barold SS, Mugica J, eds. New perspectives in cardiac pacing, 3rd edn. Mount Kisco, NY: Futura Publishing Co., 1993:233–57. By permission of the publisher.)
Fig. 7.16 Electrocardiographic tracing from a patient with pseudo-Wenckebach upper rate behavior. ARP, atrial refractory period; ESC, escape interval; PP, atrial rate; PVI, interval from P wave to ventricular stimulus; URL, upper rate limit; W1, interval from beat-to-beat prolongation of the P wave to ventricular stimulus; W2, URL-TARP (total atrial refractory period), representing the theoretical maximum increment in PV interval allowed; X, time from the ventricular stimulus to the first P wave that has fallen in the postventricular atrial refractory period. (From Higano et al. By permission of Futura Publishing Co.)

Fig. 7.17 Schema of the rate response of a DDD pacemaker with pseudo-Wenckebach type block at the upper rate limit (100 ppm). The dashed-dotted line represents the intrinsic atrial rate, and the heavy black line represents the ventricular paced rate, assuming pseudo-Wenckebach block as the atrial rate exceeds the maximum tracking rate. (Modified from Higano et al. By permission of Futura Publishing Co.)
The atrioventricular interval (AVI) should be considered as a single interval with two subportions. The entire AVI corresponds to the programmed value, i.e., the interval following a paced or sensed atrial beat allowed before a ventricular pacing artifact is delivered. The initial portion of the AVI is the blanking period. This interval is followed by the crosstalk sensing window. (B) If the ventricular sensing circuit senses activity during the crosstalk sensing window, a ventricular pacing artifact is delivered early, usually at 100–110 ms after the atrial event. This has been referred to as “ventricular safety pacing,” “110-ms phenomenon,” and “nonphysiologic AV delay.” (C) The initial portion of the AVI in most dual-chamber pacemakers is designated as the blanking period. During this portion of the AVI, sensing is suspended. The primary purpose of this interval is to prevent ventricular sensing of the leading edge of the atrial pacing artifact. Any event that occurs during the blanking period, even if it is an intrinsic ventricular event, as shown in this figure, is not sensed. In this example, the ventricular premature beat that is not sensed is followed by a ventricular pacing artifact delivered at the programmed AVI and occurring in the terminal portion of the T wave. PVC, premature ventricular contraction.
Pacing commences with the pacing artifact, not with atrial depolarization. The AVI following a sensed atrial event should therefore be shorter than that following a paced atrial event (Fig. 7.19). The differential between AV and PV is programmable.

**Rate-variable or rate-adaptive atrioventricular interval**

Most DDDR pacemakers may have the capability of shortening the AVI as the heart rate increases to allow for both tracking and sensor-driven operation.4–6 Rate-adaptive or rate-variable AVI is intended to optimize cardiac output by mimicking the normal physiological decrease in the PR interval that occurs in the normal heart as the atrial rate increases (Fig. 7.20). The rate-related shortening of the AVI may also improve atrial sensing by shortening the TARP and thereby giving more time for the atrial sensing window. Thus, use of a rate-adaptive AVI permits programming a higher upper rate limit.

There are many variations of rate-adaptive AVI, but linear shortening of the AVI from a programmed baseline AVI to a programmed minimum AVI is common (Fig. 7.21).

**Atrioventricular interval hysteresis**

This term is most commonly used to note an alteration of the AVI depending on the patient’s native AV conduction. The term is not used uniformly by manufacturers or caregivers.

Historically, the term “positive” AVI hysteresis was often used to describe a lengthening of the AVI in an effort to maintain intrinsic AV nodal conduction.7,8 It basically involves a gradual lengthening of the programmed AVI to determine if an intrinsic ventricular depolarization will occur within a certain interval. If criteria are met, the extended AVI persists unless there is lengthening of the AR or PR interval beyond preset limits, which would once again invoke the programmed AVI (Fig. 7.22). As discussed further below and in Chapter 8 (Programming), this feature is useful for minimizing the frequency of right ventricular (RV) pacing, thus minimizing the risk of heart failure in susceptible individuals.9

“Negative” AVI hysteresis is usually used to describe a shortening of the AVI in an effort to maintain paced ventricular depolarization (Fig. 7.23). This was at one time felt to have potential hemodynamic benefits for specific patients where it was important to achieve a high percentage of ventricular pacing, and remains useful in cardiac resynchronization devices to ensure that a high dose of therapy is delivered.

Table 7.2 lists the type of AVI hysteresis by manufacturer with a brief definition and programmable values.

**Atrial- and ventricular-based timing compared**

The way the timing of the pacemaker behaves in response to a sensed atrial and/or ventricular signal varies among manufacturers and among devices from the same manufacturer. Dual-chamber pacemakers may have a ventricular-based timing system, an atrial-based timing system, or a hybrid of these two systems.10,11 The
Fig. 7.21 Rate-adaptive atrioventricular delay response curves vary between manufacturers. This figure includes a combination of curve responses from various manufacturers. Reproduced with permission from: upper, Boston Scientific Corp.; bottom left, Medtronic, Inc.; bottom right, St Jude Medical.

Fig. 7.22 Simulated electrocardiographic tracing demonstrating “positive” atrioventricular interval (AVI) hysteresis. The AVI is prolonged to allow intrinsic atrioventricular nodal conduction.

Fig. 7.23 In the cycle labeled ‘B’, a sensed R-wave occurs 130 ms after the atrial pulse, i.e., intrinsic ventricular conduction. In the next cycle, the algorithm subtracts the programmed hysteresis value (30 ms) from the measured A-R interval (130 ms), i.e., 130 - 30 = 100 ms AV delay. This is labelled as ‘1’ because it is the first cycle with the shortened AV delay. After 32 cycles, at the shortened AV delay of 100 ms, the programmed AV delay is restored because no R-waves have been detected in the AV delay. Figure is courtesy of and copyrighted by St. Jude Medical.
difference between contemporary atrial- and ventricular-based dual-chamber pacemakers is of little clinical importance, although the difference may create confusion in interpretation of paced ECGs. Regardless of the timing system used, most manufacturers have modified the timing systems in such a way that the function and ECG manifestations are very similar.

In earlier pacing systems that were pure ventricular-based timing systems, the VA interval was “fixed.” In a pure ventricular-based timing system, a ventricular sensed event occurring during the VA interval resets this timer, causing it to start all over again. A ventricular sensed event occurring during the AVI both terminates the AVI and initiates a VA interval (Fig. 7.24). If there is intact conduction through the AV node after an atrial pacing stimulus such that the AR interval (atrial stimulus to sensed R wave) is shorter than the programmed AVI, the resulting paced rate accelerates. When this occurs at the LRL, the rate acceleration would be minimal, e.g., if the pacemaker is programmed to an LRL of 60 ppm (a pacing interval of 1000 ms). With a programmed AVI of 200 ms, the VA interval is 800 ms (VA interval = LRL – AVI). If AV nodal function permits conduction in 150 ms (AR interval = 150 ms), the conducted or sensed R wave inhibits the ventricular output. This, in turn, resets the VA interval, which remains stable at 800 ms. The resulting interval between consecutive atrial pacing stimuli is 950 ms (VA interval + AR interval). This

<table>
<thead>
<tr>
<th>Manufacturer</th>
<th>Terminology</th>
<th>Operation</th>
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<tbody>
<tr>
<td>Biotronik</td>
<td>AV hysteresis</td>
<td>Encourages intrinsic conduction and is programmable as a “repetitive” or “scan” option. In AV repetitive hysteresis the AVD is extended by a defined value when an intrinsic ventricular beat is sensed. When a paced ventricular event occurs, a long AV delay (AVD) is employed for a programmed number of pacing cycles. If an intrinsic event occurs during one of these cycles then the long AVD remains in operation, but if no intrinsic events occur then the original AVD is resumed. With AV scan hysteresis, after 180 consecutive cycles, the AVD is extended for a programmed number of pacing cycles and if an intrinsic event is detected when it is extended, the longer AVD remains in operation. If an intrinsic event is not detected the original AVD is resumed. A negative AV hysteresis can be initiated whereby the AVD is decreased by a programmed value after an intrinsic ventricular event. The normal AVD will resume after the programmed number of consecutive ventricular paced cycles has elapsed.</td>
</tr>
<tr>
<td>Boston Scientific</td>
<td>AV search hysteresis</td>
<td>When enabled, the AVI will lengthen periodically for up to eight consecutive cycles. It will remain active as long as the intrinsic PR interval is shorter than the hysteresis AV delay. When the first ventricular pace occurs at the hysteresis AV delay, or when the right-cycle search expires without sensing an intrinsic ventricular event, the device reverts to the programmed AV delay.</td>
</tr>
<tr>
<td>Medtronic</td>
<td>Search AV +</td>
<td>The pacemaker tracks the 16 most recent AV conduction sequences (start with nonrefractory atrial senses in tracking modes and start with atrial paces in DDI[R] modes) and adjusts PAV/SAV delays to keep intrinsic conducted events in an “AV” delay window that precedes scheduled paced events (by 15–55 ms). The AV delay window is set to promote intrinsic conduction to the ventricles, but ends early enough to avoid fusion or pseudo-fusion beats if pacing is necessary.</td>
</tr>
<tr>
<td>Sorin St. Jude Medical</td>
<td>AAISafeR (see text)</td>
<td>The pacemaker will search for intrinsic ventricular conduction every 256 cycles by adding or subtracting the AV/IVP hysteresis with search to programmed AV/IVP delay. If a ventricular event is not sensed during the extension the programmed AV/IVP delay is resumed for another 256 cycles. If it is sensed, the delay remains extended until the interval times out, i.e., no intrinsic event occurs and a ventricular pacing output is delivered. If a “negative” search is programmed then a sensed ventricular event initiates the hysteresis interval being subtracted from the programmed AV/IVP delay and the shorter interval will stay in effect for 256 cycles or until another sensed event occurs. [Newest generation of devices have Ventricular Intrinsic Preference (VIP); see text.]</td>
</tr>
</tbody>
</table>
is equivalent to a rate of 63 ppm, which is slightly faster than the programmed LRL (Fig. 7.24). When a native R wave occurs—for example, a ventricular premature beat during the VA interval—the VA interval is also reset. The pacemaker then recycles, and the result is a rate defined by the sum of the VA and AV intervals. This escape interval is therefore equal to the LRL. In both cases, the sensed ventricular event, an R wave, regardless of where it occurs, resets the VA interval.

By contrast, in a pure atrial-based timing system, the AA interval is fixed. This is in contrast to a ventricular-based system, in which the VA interval is fixed. As long as there is stable LRL pacing, there will be no discernible difference between the two timing systems.

In a system with pure atrial-based timing, a sensed R wave occurring during the AVI inhibits the ventricular output, but does not alter the basic AA timing (Fig. 7.25). Hence, the rate stays at the programmed LRL during effective single-chamber atrial pacing. When a ventricular premature beat is sensed during the VA interval, the timers are also reset, but now it is the AA interval rather than the VA interval that is reset. The pacemaker counts out an AA interval and then adds the programmed AVI, attempting to mimic the compensatory pause commonly seen in normal sinus rhythm with ventricular ectopy. For example, if the pacemaker was programmed to 60 ppm, 1000 ms cycle length and an AV delay of 200 ms, AV sequential pacing occurs and is followed by an atrial paced event at 1000 ms from the previous paced atrial event. However, if intrinsic ventricular conduction occurs at 150 ms, i.e., truncates the AV delay by 50 ms, it would result in an effective ventricular rate of 950 ms, or 63 ppm, i.e., an 800-ms VA interval plus a 150-ms AR interval. The next paced atrial event is still delivered at 1000 ms after the preceding paced atrial event, as defined by atrial-based timing. This time, the programmed AVI expires and a paced ventricular complex occurs. This results in an effective ventricular rate of 850 ms; the VA interval, which was lengthened by 50 ms because of the preceding intrinsic ventricular activity, and the 200-ms AVI, for a cycle length of 1050 ms, or 57 ppm.

Most contemporary pacemakers are hybrid timing systems. Although some published information is available on variations in timing systems, the best source is the technical manual for the specific dual-chamber pacemaker in question. A summary of the timing systems in the most recent generation of pacemakers from each company is shown in Table 7.3.
DDDR pacing systems further increase the complexity of the upper rate behavior because the pacemaker can be driven by intrinsic atrial activity to cause PV (native atrial depolarization followed by a paced ventricular event) pacing or by a sensor whose input signal is not identifiable on the ECG, or by both, to result in AV or AR pacing. The eventual upper rate also depends on the type of sensor incorporated in the pacemaker and how the sensor is programmed. Between the programmed LRL and the programmed URL, there may be stable P-wave synchronous pacing, P-wave synchronous pacing alternating with AV sequential pacing, or stable AV sequential pacing at rates exceeding the base rate (Fig. 7.26). AV sequential pacing rates may increase as high as the programmed maximum sensor rate (MSR).

Although the MSR and MTR are closely related, they are not identical. The tracking rate refers to the rate when the pacemaker is sensing and tracking intrinsic atrial activity. The MTR is the maximum ventricular paced rate that is allowed in response to sensed atrial rhythms. This may result in fixed-block, pseudo-Wenckebach, fallback, or rate-smoothing responses, depending on the design of the system. The sensor-controlled rate is the rate of the pacemaker that is determined by the sensor input signal. The MSR is the maximum rate that the pacemaker is allowed to achieve under sensor control. In some pulse generators in which the MTR and MSR can be independently programmed, the ventricular paced rate under sensor drive might exceed that attained when intrinsic atrial activity is being tracked.

Whether at the MTR or during rate acceleration below the MTR, the rhythm that results may be in part sensor-driven and in part sinus-driven (P-wave tracking) and not purely one or the other (Fig. 7.27). Which of these mechanisms predominates depends on the integrity of the sinus node and the sensor and how the pacemaker is programmed. If the sensor is optimally programmed, as the atrial rate exceeds the MTR, the RR interval will display minimal variation between sinus-driven and sensor-driven pacing (Fig. 7.28). If the rate-responsive circuitry is programmed to mimic the native atrial rate, the paced ventricular rate will not demonstrate the 2:1, or pseudo-Wenckebach-type, behavior. Conversely, if the rate-responsive circuitry is programmed in such a way that the sensor does not allow the patient to achieve sensor-driven rates above the MTR, upper rate behavior will be the same as with DDD pacing, i.e., the patient will

### Table 7.3 Summary of the timing systems in the most recent generation of pacemakers from each company

<table>
<thead>
<tr>
<th>Manufacturer</th>
<th>Timing system</th>
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<tr>
<td>Biotronik</td>
<td>In DDI mode the pacemaker uses ventricular-based timing and therefore R sensing which truncates the programmed AVI would start a VA timer. The result would be a rate of 62 or 63. When programmed to DDD or DDDR, the generators use atrial-based timing. Therefore, the lower rate timer and TARP begin with A sense, A pace or PVC sense. Atrial pacing is accomplished by expiration of the lower rate timer (there is no VA timer.)</td>
</tr>
<tr>
<td>Boston Scientific</td>
<td>If the ventricle is being paced, the escape interval timing occurs from one ventricular event to the next. If a sensed ventricular event occurs, i.e., the AV interval is truncated by an intrinsic R, the timing system switches to one of atrial-based timing to maintain rates that are true to the programmed rates</td>
</tr>
<tr>
<td>Medtronic</td>
<td>A-to-A timing is used on all current bradycardia treatment devices (IPGs). The pacemaker will lengthen the subsequent V-A timing to adjust for a prior shortened A-V delay due to native R waves being sensed or due to rate-adaptive AV adjustments, etc. This strategy will, by design, provide consistent A-to-A intervals at the expense of producing V-to-V delays that may be somewhat slower than the programmed lower rate [e.g., V-to-V rate may be as slow as 60 000/(lower rate interval + PAV – measured AV)]</td>
</tr>
<tr>
<td>Sorin</td>
<td>Modified atrial based timing system; basic intervals are determined by the atrial channel in tracking modes. PVCs (ventricular events not preceded by an atrial event) will reset all clocks as well as automatically add a nonprogrammable 500-ms atrial refractory period to prevent pacemaker-mediated tachycardia.</td>
</tr>
<tr>
<td>St Jude Medical</td>
<td>Modified* atrial-based timing if: DDD; DDD with PV &gt; MTR; DDD with safety pacing; VDD/DDD with PVC. *change to ventricular-based timing when atrial events are faster than MTR but still in alert</td>
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**Dual-chamber rate-modulated pacemakers: effect on timing cycles**

DDDR pacing systems further increase the complexity of the upper rate behavior because the pacemaker can be driven by intrinsic atrial activity to cause PV (native atrial depolarization followed by a paced ventricular event) pacing or by a sensor whose input signal is not identifiable on the ECG, or by both, to result in AV or AR pacing. The eventual upper rate also depends on the type of sensor incorporated in the pacemaker and how the sensor is programmed. Between the programmed LRL and the programmed URL, there may be stable P-wave synchronous pacing, P-wave synchronous pacing alternating with AV sequential pacing, or stable AV sequential pacing at rates exceeding the base rate (Fig. 7.26). AV sequential pacing rates may increase as high as the programmed maximum sensor rate (MSR).

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Whether at the MTR or during rate acceleration below the MTR, the rhythm that results may be in part sensor-driven and in part sinus-driven (P-wave tracking) and not purely one or the other (Fig. 7.27). Which of these mechanisms predominates depends on the integrity of the sinus node and the sensor and how the pacemaker is programmed. If the sensor is optimally programmed, as the atrial rate exceeds the MTR, the RR interval will display minimal variation between sinus-driven and sensor-driven pacing (Fig. 7.28). If the rate-responsive circuitry is programmed to mimic the native atrial rate, the paced ventricular rate will not demonstrate the 2:1, or pseudo-Wenckebach-type, behavior. Conversely, if the rate-responsive circuitry is programmed in such a way that the sensor does not allow the patient to achieve sensor-driven rates above the MTR, upper rate behavior will be the same as with DDD pacing, i.e., the patient will
Fig. 7.26 Diagram illustrating the rate response of a DDDR pacemaker and its behavior at both the maximum tracking and the maximum sensor rates. The dashed-dotted line represents the intrinsic atrial rate, and the diagonal dashed line represents the sensor rate during progressively increasing workloads. The heavy black line shows the ventricular paced rate, assuming complete heart block as it progresses from the P-tracking mode to atrioventricular (AV) sequential pacing through a period of pseudo-Wenckebach block. Note that the DDD pseudo-Wenckebach interval is shortened by sensor-driven pacing. Maximal shortening of the pseudo-Wenckebach period is accomplished by optimal programming of the sensor rate-adaptive variables. (From Higano et al.16 By permission of Futura Publishing Co.)

Fig. 7.27 Top panel, Electrocardiographic tracing from a patient with a DDDR pacemaker programmed to the DDD mode. During exercise, when the maximum tracing rate is exceeded, there are marked variations in VV cycle length as the pacemaker either waits until the ventriculoatrial interval "times out" to deliver an atrial pacing artifact or tracks an intrinsic atrial event that occurs. Bottom panel, Electrocardiographic tracing from the same patient, whose pacemaker is now programmed to the DDDR mode. Sensor-driven pacing during exercise minimizes the variation in VV cycle length. This effect has been called "sensor-driven rate smoothing." (From Hayes et al.13 By permission of Futura Publishing Co.)
experience pseudo-Wenckebach and/or 2:1 AV block when the programmed upper rate is reached.

Optimal programming of the rate-adaptive sensor is required to minimize cycle length variations between sinus-driven and sensor-driven pacing. As shown in Figure 7.28, the variation in RR interval is markedly lessened with the sensor “on” (DDDR) rather than “passive” (DDD). In the DDDR mode, the RR interval is allowed to lengthen only as much as the difference between the MTR and the activity sensor rate interval. For example, if a device is programmed to a P-wave tracking limit of 120 ppm and the patient’s atrial rate exceeds this, the pacemaker will operate in a Wenckebach-type block. If the sensor-indicated rate at this time is 100 ppm, the paced rate will drop from 120 ppm (500 ms) to an AV sequential paced rate of 100 ppm (600 ms) for the Wenckebach cycle and then return to P-wave tracking at a rate of 120 ppm. This situation usually shortens the DDD Wenckebach interval, but this interval depends on the atrial rate and the programmed values for the MTR and the TARP.

The portion of the RR cycle that is not part of the PVARP or the AVI is the period during which the atrial sensing channel is not refractory and atrial senses will inhibit scheduled atrial paces and start the SA V for tracking modes of operation. This interval can be designated as the atrial sensing window (ASW). Intrinsic atrial events in the ASW are tracked; atrial events that occur during the TARP are not tracked, but may affect mode switch, tachyarrhythmia detection and diagnostic information. If the PVARP or AVI (or both) is extended, the ASW may essentially be eliminated, so that a DDD pacemaker functions effectively as a DVI system.

Conversely, if a DDDR pacemaker is pacing at sensor-triggered rates faster than the MTR (also abbreviated at times as UTR for “upper tracking rate”), P waves falling into the ASW will inhibit the sensor-driven atrial pacing, giving the appearance of P-wave tracking at rates greater than the MTR (Fig. 7.29).

Although the MTR is programmed to a single value in DDDR pacing, it behaves as if it were variable and equal to the sensor-driven rate when the sensor-driven rate exceeds the programmed MTR (if a P wave occurs during the ASW to inhibit output of an atrial pacing artifact) (Fig. 7.30).

Mode switching
Mode switching refers to the ability of the pacemaker to change automatically from one mode to another in response to an atrial tachyarrhythmia. This may alter the ECG “timing” in a significant way. When the pacemaker is programmed to a pacing mode with ventricular tracking of atrial events, mode-switching algorithms automatically reprogram the device to a non-tracking mode when an atrial tachyarrhythmia occurs and meets detection criteria (Fig. 7.31). In the absence of mode switch, with the DDD or DDDR pacing modes a supraventricular arrhythmia may result in rapid ventricular pacing (Fig. 7.32). Perma-
nently programming the device to a nontracking mode, e.g., DDI, DDIR, VVIR, eliminates rapid paced rates during tachyarrhythmias, but also eliminates the ability to track normal sinus rhythm, which is disadvantageous in patients with paroxysmal arrhythmia. Mode switching avoids this limitation by permitting tracking of atrial events only during normal sinus rhythm.

Atrial flutter search is a related feature that may affect pacemaker timing. With the blanked atrial flutter search, if the algorithm determines that every other atrial event is being “blanked” the pacemaker will extend the PVARP and the VA interval in an effort to uncover any atrial sensed events that have been blanked. If criteria are met, a diagnosis of atrial flutter is made by the pulse generator and a mode switch will occur.

Avoiding atrial pace/sense competition

There are features intended for prevention of an atrial tachycardia being initiated by pacing within the atrium’s relative refractory period, e.g., “Non-Competitive Atrial Pacing” (API™; Medtronic, Inc.) This feature may affect both atrial and ventricular timing. If a refractory sensed atrial event occurs within the PVARP, a 300-ms interval is initiated during which no atrial pacing may occur (Fig. 7.33) If the timing cycle should have resulted in release of a sensor-driven atrial stimulus or lower pacing rate atrial stimulus during the 300-ms noncompetitive atrial pacing (NCAP) extension, the VA interval is extended until the NCAP expires. If timers do not indicate that an atrial paced event should occur during the 300-ms NCAP extension, atrial pacing occurs at the end of NCAP period. If an atrial sense occurs during the NCAP, a new NCAP is initiated.

If delivery of an atrial pacing stimulus is delayed by the NCAP, the pacemaker will attempt to keep the ventricular rate stable by shortening the PAV interval that would follow the NCAP. If the pacemaker is programmed so that the lower rate is relatively high and the PVARP relatively long, NCAP could result in ventricular pacing at a rate slightly below the lower rate limit.

Another manufacturer includes Atrial Protection Interval (API™), which shortens the PVARP for each interval where the pseudo-Wenckebach window is < 125 ms. This provides a noncompetitive pacing window of 125 ms.
Timing components of ventricular avoidance pacing algorithms

Data demonstrating the potential adverse hemodynamic effects of RV pacing have led to a variety of ventricular avoidance pacing algorithms (see Chapter 2, Hemodynamics).
The “timing” aspects of these algorithms vary significantly between manufacturers, best explained in a combination of manufacturer-specific and generic descriptions.

The most common feature used to promote intrinsic AV conduction is AV search hysteresis. Although each of the manufacturers that use this approach has slight differences in their algorithms, the basic operation is that of a programmed AV delay being extended to a programmed extension if a sensed ventricular event occurs. The extended AV delay persists until a paced ventricular event and the programmed AV delay is then reestablished. Depending on the algorithm, the pacemaker may periodically invoke the extended AV delay to “search” for intrinsic AV conduction. At the present time, variations of this approach are used by multiple manufacturers (Fig. 7.34).

There are other available algorithms used to promote intrinsic AV conduction that warrant more detailed description in order to understand their potential impact on the timing cycle of the pacemaker.

**Managed Ventricular Pacing (MVP) from Medtronic**

There are two available modes, AAIR ⇔ DDDR and AAI ⇔ DDD, meaning that atrial pacing is delivered allowing intrinsic AV conduction with continuous monitoring of AV conduction. If there is persistent loss of AV conduction, the mode will switch to the corresponding dual-chamber mode, i.e., DDDR or DDD (Fig. 7.35). When the pacemaker detects the return of AV conduction, the atrial pacing mode is resumed.

If there is transient loss of AV conduction, the pacemaker delivers a back-up ventricular pacing output.
in response to an A-A interval that does not contain a ventricular sensed event.

Persistent loss of AV conduction is defined as two of the four most recent nonrefractory A-A intervals missing a ventricular event. This results in a switch to the DDD or DDDR mode.

The pacemaker does periodic single-cycle assessments for AV conduction and, if present, will resume AAIR or AAI pacing mode.

**AAISafeR from Sorin**

An AV delay is not initiated after sensed or paced atrial events. The device will tolerate first-, second-, and third-degree AV block up to a programmed level before the mode changes to DDD or DDDR mode. PR and AR intervals are permanently monitored (Fig. 7.36). If the PR interval exceeds 350 ms or the PR interval exceeds 450 ms the mode switches to the DDD or DDDR mode and invokes the programmed AV delay.

The mode will return to AAI or AAIR mode if there is sensing of 12 consecutive R waves or after 100 cycles in DDD(R) mode. If there have been more than five switches to DDD(R) mode per day on three consecutive days or ≥ 15 switches occur within 24 h, a change back to the AAI(R) mode will not occur until the device is reprogrammed.

**Endless-loop tachycardia**

Endless-loop tachycardia (ELT) is not a portion of the timing cycle, but understanding the timing cycle of dual-chamber pacing is crucial to understanding ELT and vice versa. Endless-loop tachycardia, also referred to as “pacemaker-mediated tachycardia,” “pacemaker-mediated reentry tachycardia” and “pacemaker circus movement tachycardia,” is defined as a reentry arrhythmia in which the dual-chamber pacemaker acts as the antegrade limit of the tachycardia and the natural conduction pathway acts as the retrograde limit (Fig. 7.37).
Fig. 7.36 Example of AAI SafeR—in this example the algorithm allows two successive P waves without ventricular pacing in an attempt to allow intrinsic AV conduction. (AVD – atrioventricular delay.)

Fig. 7.37 Schematic diagram of endless loop tachycardia. When AV synchrony is lost for whatever reason with a dual-chamber pacemaker with atrial sensing, the paced ventricular beat may result in retrograde conduction. If the retrograde conduction results in a retrograde atrial event which is sensed by the device, the AV interval is again initiated resulting in another paced ventricular event, at or near maximum tracking limit. Reproduced with permission from the Mayo Clinic.
If A-V synchrony is uncoupled, i.e., if the P wave is displaced from its normal relation to the QRS complex, the subsequent ventricular event may result in retrograde atrial excitation if retrograde or VA conduction is intact. If the retrograde P wave is sensed, the AVI of the pacemaker is initiated. On termination of the AVI and MTR interval, a ventricular pacing pulse is delivered, which could once again be conducted in a retrograde fashion. Once established, this reentrant mechanism continues until interrupted or until the retrograde limb of the circuit is exhausted (Fig. 7.38). The paced VV interval cannot violate the programmed maximum limit, or URL, of the pacemaker, and the ELT often occurs at the URL.

There are two basic mechanisms that have been adopted to prevent or minimize ELT. If the pacemaker detects a programmable number of consecutive AS-VP cycles at the maximum tracking rate with a constant VP to AS interval, the pacemaker will extend the PVARP for at least one cycle. The next retrograde P wave will fall into the refractory and terminate the tachycardia. In some devices the detect rate may be programmed at a rate less than the MTR (Fig. 7.39).

Fig. 7.38 PMT (pacemaker mediated tachycardia) initiated by a premature ventricular event (VS).

Fig. 7.39 Schematic representation of pacemaker-mediated tachycardia termination algorithm. The intrinsic conduction system acts as the retrograde pathway and the pacemaker as the antegrade pathway. In this schematic, assumed to be from a patient at rest with a maximum tracking rate of 120 ppm (500 ms), with beats ‘1’ through ‘8’ the retrograde P wave occurs outside the PVARP or imitates the AV interval. On beat ‘9’ an algorithm to limit ELT extends the PVARP to 400 ms. The retrograde P wave after beat ‘9’ occurs within the PVARP and will terminate the ELT.
Because a premature ventricular event may commonly be the initiator of ELT, many pacemakers extend the PVARP when a premature ventricular contraction (PVC) is detected. Once again, by extending the PVARP, a retrograde atrial event that occurs as a result of a PVC would occur in refractory and be ignored.

**Timing cycles with algorithms responding to sudden bradycardia**

Algorithms used in patients paced for neurocardiogenic syncope with clinically significant cardioinhibition and vasodepression may alter timing cycles by introducing a sudden increase in pacing rate. If a patient’s heart rate occurs in a manner that meets the criteria of the algorithm, the device responds with pacing at a programmable faster rate, most commonly 90–100 ppm, in an attempt to blunt the patient’s symptoms (Fig. 7.40). Figures 7.41 and 7.42 demonstrate features from two manufacturers in a diagrammatic fashion.

**Timing cycles unique to biventricular pacing**

Programmable pacing modes in cardiac resynchro-

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Fig. 7.40 Schematic representation of the electrocardiogram with the onset of a sudden bradycardia response algorithm. The rate of 60 ppm at the onset of this tracing actually represents dramatic slowing which triggers the algorithm and results in a pacing rate of 100 ppm.

Fig. 7.41 Schematic diagram of "Rate-Drop Response." (Reproduced with permission from Medtronic.)
Pacemaker and Cardiac Resynchronization Timing Cycles and Electrocardiography

Cardiac resynchronization therapy (CRT) devices are the same as those described for standard pacemakers, and the general criteria for mode selection are similar to those for standard pacing.

Additional features that add complexity to biventricular pacemakers include the need to maintain a high frequency of ventricular pacing, and the need to tailor interventricular timing and pace/sense vectors in many patients. In contemporary cardiac resynchronization devices, ventricular sensing for the purpose of timing occurs only in the RV (Fig. 7.43). This occurs to prevent QRS double counting due to the long conduction time between the right and left ventricles in many patients receiving CRT devices. In implantable cardioverter-defibrillator (ICD) systems, double counting leads to inappropriate detections and therapies. Because the paced V-V interval is programmable, biventricular stimulation may occur simultaneously, or with an offset with one ventricle preceding the other. This must be considered when interpreting the timing cycle (Fig. 7.44).

**Achieving consistent biventricular pacing**

Benefits will not be realized from biventricular stimulation if a sufficient number of QRS complexes are not resynchronized. Resynchronization requires pacing and capture from the left ventricular (LV) lead. In many patients, the AV delay must be relatively short in order to maintain consistent ventricular pacing and avoid fusion with intrinsic conduction. However, the AV delay must be long enough to allow adequate ventricular diastolic filling (see Chapter 2, Hemodynamics).

Several algorithms have been designed to maintain biventricular pacing and prevent inhibition by intrinsic rhythm.

AV hysteresis, discussed above, is used to promote intrinsic ventricular conduction (positive AV hysteresis) in non-CRT systems. In CRT systems, negative AV hysteresis maintains a paced ventricular rhythm because a sensed ventricular event results in shortening of the subsequent AV interval. Although algorithms for negative AV hysteresis vary among manufacturers, most algorithms periodically lengthen the AV delay. If the ventricular event that follows is paced, the longer AV delay is maintained (promoting filling) until a sensed ventricular event occurs, at which time the AV delay is again shortened (Fig. 7.45 and 7.46).
Fig. 7.44  (A) Negative RV-LV. The LV paced event occurs before the RV paced event. (B) Positive RV-LV. The right ventricular paced event occurs before the left ventricular paced event. (C) Zero RV-LV. The right and left ventricular paced events occur simultaneously.29 (Reproduced with permission from Blackwell Publishing.)

\[ \text{AP-RVS > AP-RVP} \]

Fig. 7.45  Biventricular RV-LV hysteresis may be used to force biventricular pacing. RV, right ventricular; LV, left ventricular; LVP, left ventricular paced; RVP, right ventricular paced; RVS, right ventricular sensed.29 (Reproduced with permission from Blackwell Publishing.)
The Ventricular Sense Response™ algorithm maintains resynchronization by immediately pacing the LV in response to a sensed event in the RV (Fig. 7.47). Similarly, in some CRT devices the DDT mode allows triggered pacing when a ventricular sensed event occurs (Fig. 7.48).

In addition to lack of ventricular stimulation secondary to inhibition by intrinsic ventricular events, successful biventricular stimulation may also be lost if LV capture thresholds exceed the programmed output parameters. In one available system a feature is available that will periodically assess LV pacing thresholds and adjust the outputs to ensure LV capture.\(^3\) Loss of LV capture during testing, however, could lead to scenarios with transient inhibition of biventricular pacing.

**Lower rate behavior**

Lower rate behavior in CRT systems is similar to dual-chamber pacemaker function. Events may occur in either ventricle, but sensing for the purpose of “timing” occurs only on the RV lead in current CRT devices. There are devices that have “left ventricular” sensing, but it is for the purpose of electrograms and diagnostic information only. Theoretically, more combinations of sensing and pacing would be possible if LV events were to be used for the purpose of “timing.”

**Univentricular sensing and biventricular pacing**

Whenever pacing without sensing occurs in a specific cardiac chamber, there is theoretical concern that competitive pacing could result in the induction of a tachyarrhythmia. For example, VAT pacing (sensing only the atrium, pacing only the ventricle) was a predecessor of VDD pacing that was abandoned because of the potential for pacing the ventricle during the T wave, leading to ventricular arrhythmias.

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**Fig. 7.46** Biventricular hysteresis. Because conduction resulted in RVS event, negative hysteresis would be used to maintain biventricular pacing. LVP, left ventricular paced; RVP, right ventricular paced; RVS, right ventricular sensed.\(^2\)\(^9\) (Reproduced with permission from Blackwell Publishing.)

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**Fig. 7.47** Ventricular Sense Response™ is demonstrated in a telemetry tracing which demonstrates ventricular pacing occurring after the onset of the QRS. This is due to the Ventricular Sense Response™ algorithm, which introduces a triggered biventricular pacing pulse each time a sensed RV event is seen. (Reproduced with permission from Swerdlow CD, Friedman PA. Advanced ICD troubleshooting: Part I. PACE 2005; 28:1322–46.)
To date, this has not been a significant clinical problem with CRT devices. Physiologically, if activation of the LV occurs before LV pacing, the ventricle should be refractory and the LV pacing stimulus would be ineffective (Fig. 7.49). However, because there are situations where competitive LV pacing could possibly result in competitive pacing and induction of a ventricular tachyarrhythmia (Fig. 7.50), devices may incorporate a protective feature. A feature known as Left Ventricular Protection Period (LVPP™) is designed to prevent pacing during the LV vulnerable period (T wave). LVPP is the period after a LV event, either paced or sensed, when LV pacing is inhibited (Fig. 7.51).

**Upper rate behavior in biventricular pacing**

Specific to biventricular pacing is an understanding of how upper rate behavior could impact consistent ventricular stimulation. The majority of patients receiving biventricular pacing have intact AV conduction. Therefore, upper rate behavior that would result in pseudo-Wenckebach behavior should be avoided, because extension of the AV delay would allow a sensed ventricular event to occur and inhibit biventricular pacing (Fig. 7.52). Once ventricular sensing occurs, it is possible for inhibition to persist until the sinus cycle length becomes longer than the PR + PVARP, the so-

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**Fig. 7.49** Right ventricular sensing with biventricular pacing. The left ventricular event, resulting from conduction from the right to left ventricle, is not sensed. Therefore, since the RV-LV interval is positive, the LV paced event follows the LV event. In this theoretical schematic, it is unlikely that the LVP would result in effective capture. The second output pulse, i.e., the LVP in this example, would either encounter physiologically refractory tissue or in the setting of a very delayed interventricular conduction, true fusion would result. It is unlikely that this type of competitive pacing would induce a repetitive rhythm.29 (Reproduced with permission from Blackwell Publishing.)
CHAPTER 7 Pacemaker and Cardiac Resynchronization Timing Cycles and Electrocardiography

265

called intrinsic total atrial refractory period. The upper rate could also be affected by atrial tachyarrhythmias that are frequently seen in this population of patients. In order to maintain consistent biventricular pacing, ideally at or near 100% pacing, the TARP should be sufficiently short and the MTR should be sufficiently high. Sensor-driven pacing can also be used to permit continued biventricular pacing.

Atrial premature beats with conduction may cause a similar phenomenon by causing atrial undersensing and loss of biventricular pacing.

Premature beats and biventricular timing cycles

In a dual-chamber pacemaker a premature ventricular event (PVC) is usually defined as two consecu-

![Fig. 7.50](image_url) In this scenario a left ventricular sensed (LVS) event precedes the RV paced event. When a long RV-LV interval is present, and LV-RV conduction is very prolonged, the LV paced event may occur when the LV is no longer refractory. (Although theoretically possible, it is unlikely that a manufacturer would allow a RV-LV interval of such proportion to allow this scenario.) (Reproduced with permission from Blackwell Publishing.)

![Fig. 7.51](image_url) In this tracing there is a notation in the marker channel of “inh-LVP” which represents inhibition of left ventricular (LV) pacing. This is due to the LV protection period algorithm, which is designed to prevent pacing during the LV vulnerable period (T-wave). (Reproduced with permission from Swerdlow CD, Friedman PA. Advanced ICD troubleshooting: Part I. PACE 2005; 28:1322-46.)
tive ventricular events without an intervening atrial event. In a dual-chamber pacemaker the presence of an event identified as a PVC would reset the timing cycle and potentially uncouple AV synchrony. In addition, a PVC may trigger an extension of the PVARP to prevent the occurrence of pacemaker-mediated tachycardia.

In a CRT system a PVARP extension and the subsequent functional atrial undersensing that may occur along with uncoupling of AV synchrony may result in loss of biventricular pacing. Thus, the PVC-PVARP Extension function, if an option, should usually be deactivated in a biventricular system.

Refractory periods and biventricular pacing
Contemporary CRT devices sense only in the RV while pacing in both right and left ventricles. After pacing in one chamber, a cross-chamber refractory period may be created. If there is a programmed offset between LV and RV pacing, the total ventricular refractory period may be prolonged. If the device is programmed so that LV stimulation precedes RV stimulation, the total ventricular refractory period may be quite long. In a CRT-D system the prolonged sensing refractoriness could theoretically compromise detection of ventricular tachyarrhythmias.

Timing cycles in implantable cardioverter-defibrillators
The timing cycles of pacemakers apply to ICDs as well. However, there are timing cycle issues and "lock-outs" that are specific to ICDs. These features are discussed in detail in Chapter 10, Troubleshooting.

Initial electrocardiographic interpretation
In reviewing an ECG from a patient with an implanted pacemaker, one should carefully assess the underlying rhythm and its relationship to the pacemaker artifacts. The first step is to find any portion of the ECG during which the heart is not paced, i.e., identify the intrinsic cardiac rhythm. That portion of the ECG should be interpreted as any ECG would be: PR, QRS, and QT intervals; rate; axis; voltage; and so forth. If no intrinsic rhythm is apparent, the patient may be pacemaker dependent or the pacemaker may be programmed to stimulate faster (i.e., at a shorter cycle length) than the intrinsic rhythm. After determining the spontaneous atrial and ventricular rhythms, one should look for any relationship between the two; for example, does a P wave result in a QRS complex, indicating intact AV conduction? After the intrinsic rhythm has been carefully scrutinized, pacemaker activity should be assessed. If pacemaker activity is present, is there one stimulus or are there two stimuli? If only one stimulus is present, does it result in atrial (Fig. 7.53) or ventricular (Fig. 7.54) depolarization? Is there an apparent relationship between pacemaker activity and atrial activity or ventricular activity, or both? If pacing artifacts are occurring only in the ventricle, there is no relationship between the pacemaker stimulus and a preceding P wave and the pacemaker stimulus fol-
lows the intrinsic QRS complex at a consistent cycle length, ventricular sensing as part of ventricular inhibited (VVI) pacing is present (Fig. 7.54). If a pacemaker artifact is consistently found within intrinsic P or QRS complexes, a triggered pacing mode (AAT or VVT) exists (Fig. 7.55). Therefore, there is atrial far-field sensing, but because the events fall in refractory, they do not alter the timing cycle.

It is usually not possible to determine from the ECG whether the pacemaker is operating in a bipolar or a unipolar configuration. With analog recording systems, it may be possible to assess the size of the pacemaker stimulus in an effort to determine polarity. If the pacemaker artifact is large, it is most likely of the unipolar configuration; if a very small pacemaker artifact is present, it is most likely of the bipolar configuration. With the more commonly used digital recording systems, which artificially simulate the pacemaker artifact, the size of pacing artifacts is meaningless. There may even be situations in which all cardiac activity is paced and no artifacts are visualized or artifacts are only intermittently present even though all activity is paced (Fig. 7.56).
Fig. 7.55 Simulated tracing demonstrating AAT pacing mode with a pacing artifact occurring within each intrinsic atrial event. In the absence of an intrinsic atrial event, a pacing artifact is delivered at the same interval and atrial depolarization follows the pacing artifact.

Fig. 7.56 Twelve-lead electrocardiogram (insert is close-up of the area described) that demonstrates all ventricular paced events. However, in the area circled, there is a ventricular paced event without a discernible pacing artifact. The ventricular event is paced in this completely dependent patient and the absence of the pacing artifact is a function of the digital recording system.
Response to magnet application
Assessing the magnet response of the pacemaker provides additional information about pacemaker function and may be helpful in interpretation of the paced ECG. Magnet response may also help identify the pacing mode and often the specific pulse generator, and is equally useful for single- and dual-chamber pacing.

Application of a magnet to a single-chamber pacemaker always results in single-chamber asynchronous pacing (Fig. 7.57). In dual-chamber pacemakers, magnet application almost always results in asynchronous pacing in both the atrial and the ventricular chambers (DOO mode) (Fig. 7.58). Most pacemakers programmed to a VDD mode will display VOO pacing with magnet application. There are pacemakers that have a programmable option of turning the magnet response “off” and some with an option of having asynchronous operation for a specified number of beats followed by return of synchronous behavior. If magnet application fails to result in asynchronous behavior, programmed parameters should be checked to see if the magnet has been programmed “off.” Also, some pacemakers, when in a “reset” mode, will not display a magnet response. Historically there have
been exceptions, e.g., pacemakers with a VOO magnet response even when programmed to the DDD mode, but few, if any, remain in service.

The pacing rate should be determined during magnet application. Is the magnet rate faster or slower than or the same as the programmed pacemaker rate? If the pacemaker is a single-chamber pacemaker, does it result in atrial or ventricular depolarization? Having determined what chamber is being paced, one can assess the pacemaker artifact and subsequent depolarization to ensure proper capture. It should be remembered that pacemakers of different manufacturers respond differently to magnet application. Some continue to pace asynchronously for a specific number of beats after removal of the magnet and may do so at more than one rate. The magnet response of a particular pacemaker may vary depending on the programmed parameters, i.e., the mode, of the pacemaker. The individual specifics of magnet application must be known for each pacemaker to determine that behavior is normal during magnet application and after removal. For dual-chamber pacemakers operating in the DOO mode, the AV interval should be measured during magnet application.

It is important that few assumptions be made about the details of the magnet mode of operation and that one be aware of the specifics of the magnet response in a particular unit; otherwise, an erroneous interpretation of inappropriate operation may be made. The magnet mode is usually (but not always) free of sensing any events and is often at a specific rate independent of the programmed rate and sensitivity settings. It allows determination, with a puzzling or unusual ECG, of whether the pulse generator is capable of operating normally.

When a single cardiac chamber is being paced, the effect of the paced chamber on the remaining chamber should be determined. For example, if an atrial pacemaker is present, does atrial depolarization result in AV conduction and an intrinsic QRS complex, demonstrating intact AV conduction (Fig. 7.59)? Alternatively, if a ventricular pacemaker is present, is there retrograde activation of the atrium, resulting in retrograde P wave activity following the paced ventricular complex (Fig. 7.60)?

**Single-chamber pacemakers**

By following the preceding steps, one will have determined whether a single- or dual-chamber pacemaker is present and whether the pacemaker stimuli result in atrial or ventricular depolarization (or both). If a single-chamber atrial pacemaker is present, if stimulation produces atrial capture, and if the pacemaker artifact is inhibited by intrinsic P waves, the pacemaker is in the atrial inhibited (AAI) mode (Fig. 7.53). In the AAI mode, paced ventricular activity is never seen, with or...
without magnet application, and with normal function a pacemaker artifact never occurs within the intrinsic P waves. If a stimulus occurs that results in ventricular capture with inhibition by QRS complexes, the pacemaker is in the ventricular inhibited mode (VVI). If the pacemaker is pacing asynchronously without sensing or capture of either the atrium or the ventricle, the mode cannot be determined. Similarly, with a single-chamber pacemaker, either atrial or ventricular, if intrinsic activity is never seen and every complex is paced, either the patient is pacemaker-dependent or the pacemaker has been programmed to a rate faster than the intrinsic cardiac rate.

If a single stimulus falls consistently into the spontaneous P wave or QRS complex, the mode is of the triggered variety (AAT/VVT) (Fig. 7.55). Although this mode of pacing is available in many multimodal programmable pacemakers, it is rarely used as a long-term pacing mode. Programming a pacemaker to the triggered mode is sometimes helpful to determine exactly where on the surface ECG sensing occurs.

An exception to the rule of the timing cycles in AAI and VVI pacing and a long-standing source of confusion is hysteresis. This programmable feature allows the escape interval for the initial paced beat to be at a longer cycle length than subsequent paced intervals (Fig. 7.61).
For example, if a patient has sinus node dysfunction with episodes of sinus bradycardia or sinus arrest, the pacemaker can be programmed to pace continuously at an interval of 1000 ms (rate of 60), but hysteresis takes place at a rate of 40, i.e., 1500 ms without a paced event is allowed before pacing is initiated at the programmed rate. If one does not know that hysteresis is “on,” the two different intervals may give the appearance of oversensing. However, if the intervals are repetitive and the longer interval always follows an intrinsic beat, hysteresis is the most likely explanation.

**Dual-chamber pacemakers**

If a dual-chamber pacemaker is present, the steps already outlined should be followed, including determination of the AVI and the status of AV and VA conduction. The next step in interpretation of an ECG with dual-chamber pacing should be to determine the pacing mode. During the free-running (nonmagnet) pacemaker mode, it should be determined whether ventricular sensing, ventricular pacing, atrial pacing, or ventricular tracking of atrial activity occurs.

If P-wave activity is being sensed, does each P wave begin a pacemaker cycle? If each spontaneous P wave results in a paced ventricular complex at a consistent preset AV delay, the pacemaker is P-synchronous and may be in the DDD or VDD mode (Fig. 7.62). There are several ways to differentiate VDD from DDD pacing: Intermittent atrial pacing indicates DDD pacing; the absence of atrial activity followed by ventricular pacing at the lower rate or sensor-indicated rate is consistent with VDD pacing (Fig. 7.63). With magnet application, DDD pacemakers usually respond with DOO pacing and VDD pacemakers with VOO pacing.

If each sensed P wave inhibits pacemaker output but initiates synchronous ventricular pacing, the pacemaker is in the DDI mode (Fig. 7.64). Sensed atrial activity inhibits atrial output but does not result in a ventricular stimulus after the AV delay. AV sequential pacing at the programmed rate is provided if intrinsic activity is absent. Intrinsic ventricular activity occurring during the atrial escape interval or AV delay inhibits the pacemaker and resets the timing cycle.
Atrioventricular interval

The components and potential variations of the AVI have already been discussed. As the ECG is analyzed, the AVI should be assessed for any variations. The possible explanations for a variant AVI include:

- Ventricular safety pacing
- PV vs. AV interval (differential AV delay)
- AVI hysteresis—either “positive” or “negative”
- Rate-variable or rate-adaptive AVI.
Upper rate behavior
Descriptions have already been provided for 1:1 P-synchronous pacing at the MTR, pseudo-Wenckebach, and 2:1 upper rate behavior (Fig. 7.65). Other variations are fallback and rate smoothing.

Rate smoothing
Rate smoothing avoids abrupt changes in pacing rate, such as those that can occur during a sudden transition to pseudo-Wenckebach or 2:1 upper rate behavior, sinus pause or sinus arrest, premature ventricular and/or atrial contractions, paroxysmal supraventricular tachyarrhythmias, and may eliminate patient symptoms associated with sensed dysrhythmic events.33

Rate smoothing controls sudden changes in pacing rate by monitoring the interval between ventricular events (both paced and sensed) and storing the most recent RR interval in memory (Fig. 7.66). On the basis of this RR interval and the programmed rate-smoothing percentage, the pulse generator sets up two rate-control windows for the next cycle—one for the atrium and one for the ventricle (Fig. 7.67). For example, if the monitored VV interval is 800 ms and 6% rate smoothing is programmed, the algorithm allows the upcoming ventricular rate of the cycle to increase or decrease a maximum of 6%, or ±48 ms (752–848 ms).

![Fig. 7.65](image1.png) Electrocardiographic example of upper rate behaviors including transient pseudo-Wenckebach on the left, which rapidly progresses to 2:1 upper rate behavior as the atrial rate continues to increase.

![Fig. 7.66](image2.png) Example of how two rate-smoothing synchronization windows are calculated. If the heart rate is 75 ppm (800 ms) and rate-smoothing is programmed “on” at 6%, the next cycle length may vary by 48 ms, a range from 752 to 848 ms. The subsequent cycle would be calculated as ±6% of 752 or 848 ms, depending on whether the atrial rate was increasing or decreasing.
The rate-smoothing algorithm determines the atrial control window in a manner analogous to the basic ventricular timing cycle: \( VV = VA + AV \). To determine the VA interval from this equation if the VV interval and AVI are known, one simply subtracts the AVI from the VV interval. Rate smoothing does likewise by subtracting the AVI value from the ventricular control window, and the result is a "rate-controlled VA interval." Extending the previous example, if the AVI is 150 ms, the atrial control window is also ±48 ms (602–698 ms). Atrial pacing is observed at the maximum calculated VA interval of 698 ms if no sensed event occurs before the end of the VA interval.

**Ventricular rate regularization**

This feature is useful in patients with chronic atrial fibrillation or frequent atrial tachyarrhythmias. The purpose of the algorithm is to reduce variability in V-V cycle length when atrial arrhythmias are conducted. Pacing during AF may also modestly slow the ventricular response by means of concealed conduction into the AV node. Specific algorithms vary between manufacturers, but similar to "rate-smoothing" described above a regularization algorithm will calculate the differences between cycle lengths and pace as necessary to minimize cycle length variation.

**Fallback**

In a generic sense this feature can be thought of as a mechanism to decrement the paced rate gradually, but the specific "fallback" feature may vary between manufacturers. Fallback operation refers to the decrement in paced rate that occurs after criteria for mode-switch have been met. Once the mode has switched, the pacing rate will "fallback" to either the sensor-indicated rate or the programmed lower rate specific to the mode-switch algorithm (Fig. 7.68).

A different type of paced rate "fallback" may be an option for the patient’s hours of sleep. A "sleep" function, available in a number of pacemakers, allows a lower rate to be used during the sleeping hours. In some devices the patient’s usual hours of bedtime and arising are programmed into the pacemaker and the desired lower rate during "sleep." In other devices the sleep function may be tied to the rate-adaptive sensor. If the sensor detects no activity for a given period of time, the lower "sleep" rate would go into effect until activity is again recognized.
Approach to the biventricular paced electrocardiogram*

With the increasing use of and requirement for familiarity with biventricular pacing systems, several key questions are often asked:14–36
1 Is the LV lead capturing the ventricle?
2 Has the LV lead moved from its initial position at implantation?
3 What is the degree of fusion of ventricular activation between the left and RV pacing leads?37
4 Is there anodal stimulation present?
5 Is there likely atrioventricular synchrony?

Although a complete answer to these questions requires thorough analysis of the chest radiograph, ECG, and device interrogation, the ECG often provides immediate and reliable answers to these questions.38,39 This section outlines the ECG vector principles that underlie the ability to answer these questions. The characteristic ECG signature for sites in the RV and various LV lead positions and an approach to analyze the paced ECG to ascertain the extent and site of the pacing lead capture as well as the relative contribution of the two leads are presented.40

Principles of electrocardiographic and interpretation relevant to biventricular devices

The fundamental principles for interpreting biventricular device ECGs are as follows: (i) ECG leads generally represent widely spaced bipoles;41,42 and (ii) whenever a depolarization front, i.e., cardiac activation, proceeds toward a positive pole of an ECG lead, a positive deflection is inscribed. It follows that a negative deflection in a given ECG lead suggests electrical activation proceeding away from the positive electrode of that lead.43,44

On the basis of knowing the configuration of a given lead and understanding the significance of a positive or negative deflection, the vector of activation can be estimated. Therefore, a positive R wave in lead I suggests electrical activation proceeding from the right side of the ventricle to the left, because the positive electrode for lead I is located on the left arm. This leftward vector may represent activation from the RV toward the LV, or from the interventricular septum to the free wall of the LV.41,42 The principles for this type of analysis with biventricular devices are similar to those used to identify the site of earliest activation in Wolff–Parkinson–White syndrome or ventricular tachycardia.

Left and lateral leads

The positive electrode or equivalent for leads I, aVL, V5 and V6 are all located on the left side of the body. Therefore, a positive deflection in these leads suggests activation proceeding from right to left, and conversely, a negative deflection suggests activation.
proceeding away from the left and lateral portions of the ventricle (Fig. 7.69). However, there are important differences in the information provided within this set of leads. Lead aVL, in addition to having its positive electrode equivalent on the left side, is also superior compared with lead I. Therefore, a high superior site of activation that proceeds away from lead aVL toward the left lateral wall produces a negative deflection in aVL and yet is positive in lead I. The situation is similar for leads V5 and V6. Although their positive electrode is located on the left side of the body, it represents a more inferior and apical position than the positive electrode of lead I. Therefore, an apical origin of activation may be sharply negative in V6 but positive in lead aVL or lead I. Thus, a quick look at this set of leads will show that activation is proceeding from right to left or vice versa.41,42

A closer look at differences in the information or degree of positivity and negativity between these leads gives additional information about a superior or inferior leftward location. RV pacing from the apex always produces a positive deflection in leads V5 and V6. Although their positive electrode is located on the left side of the body, it represents a more inferior and apical position than the positive electrode of lead I. Therefore, an apical origin of activation may be sharply negative in V6 but positive in lead aVL or lead I. Thus, a quick look at this set of leads will show that activation is proceeding from right to left or vice versa.41,42

Inferior leads
Leads II, III and aVF have their positive electrode or equivalent located inferiorly. Therefore, a positive deflection in these leads suggests activation proceeding from a superior location toward the feet. Conversely, activation from an inferior site in the ventricle produces an S wave or negative deflection in these leads.43 There are some important differences in the information conveyed within this group of leads. The orientation of lead III is such that the positive electrode, although inferior, is more rightward than lead II. Therefore, RV pacing from an inferior location is more negative in lead III than in lead II. Conversely, pacing from a LV inferior site such as the middle cardiac vein is negative in both leads II and III; however, it is more negative in lead II than in lead III. Thus, the degree of negativity (i.e., depth of the S wave) compared between leads II and III will clarify further whether an inferior pacing site is rightward or leftward on the inferior portion of the heart.

Rightward chest leads—leads V1, aVR and III
These leads have their positive electrode or equivalent located on the right side of the body.46 Therefore, a
negative deflection in these leads suggests activation proceeding from right to left. There are important differences in the information provided within this set of leads. Although lead V1 represents a rightward positive electrode, it is placed on the anterior chest. Therefore, lead V1 is negative either with a right-sided stimulation site or an anterior stimulation site. Conversely, pacing from the left side or a posterior site produces a positive deflection (right bundle branch block pattern) in lead V1. Because the LV is posterior and to the left of the RV, most LV pacing sites produce a positive deflection in lead V1 (right bundle branch block). Most stimulation sites in the RV are rightward and anterior to the LV and thus produce a negative deflection in lead V1 (left bundle branch block pattern). However, a lead located deep in the RV apex, especially with clockwise rotated hearts, may produce a positive deflection in lead V1.

Lead aVR, with the location of its right anterior positive electrode equivalent to lead V1, is also a superiorly located positive electrode. Therefore, inferior and apical sites produce a positive deflection in lead aVR irrespective of a rightward or leftward orientation. Also, stimulation from anterior sites such as the basal anterior interventricular vein (see below) produces a negative deflection in lead aVR (as with lead aVL) regardless of rightward or leftward orientation.

Lead III, with its positive electrode located inferiorly and relatively rightward as described above, is negative in most typical RV pacing locations. Because the RV apex is both inferior and rightward, sharp negative deflections are seen in lead III. However, certain RV pacing sites such as the outflow tract, although still rightward, produce a positive deflection in lead III.

Chest leads

The positive electrodes of leads V1 through V6 are arranged sequentially from the right second intercostal space and “drape” the typical apical location. More apical locations for pacing stimulation produce a negative deflection in leads V4, V5 and V6, whose positive electrodes are located at the apex. Conversely, leads located more basally produce a negative deflection in lead V1. Analyzing the transition between negativity and positivity through the chest leads gives an idea of the apical to basal location of the pacing lead. For example, a lead placed in the posterolateral vein produces a negative deflection in lead I, signifying its left-sided pacing site. However, this lead alone will not provide information about whether the lead is located apically in this vein or more basally. In addition, if leads V4, V5 and V6 are negative, this suggests that the lead is very apical within the posterolateral vein. A positive deflection in these same leads suggests a more basal location within this vein.

Leads aVR and aVL

Both these leads have their positive electrode equivalent superiorly. Therefore, apical sites of stimulation produce positive deflections in both leads, and superior sites of stimulation produce negative deflections (S waves). The positive electrode for aVL is more leftward; thus, the positive electrode for aVR is more rightward. Consequently, a sharp deep negative S wave in lead aVR that is deeper than the S wave in lead aVL suggests a right superior pacing site.

Although at first glance it may appear complex to analyze ECGs to define the pacing site, the analysis consists of simple deductions based on the two basic principles. Understanding where the positive electrodes for the various leads are located and knowing that a pacing site near the positive electrode of a lead produces a sharp negative S wave in that lead will facilitate further analysis.

To identify the pacing lead site quickly, the ECG deflection in leads I and aVF can be considered. Lead I, if positive, suggests a right-sided or septal pacing site and, if negative, a left free-wall pacing site. A positive R wave in lead aVF suggests an anterior site (anterior interventricular vein in the high septal region), whereas a negative deflection (S wave) suggests an inferior or posterior pacing site (RV apex middle cardiac vein). These simple generalizations with just these two leads work in most instances.

Right ventricular pacing sites: ECG recognition

Typical RV pacing from an apical location is relatively straightforward to recognize on an ECG (Fig. 7.70). The key features include a left bundle branch configuration (negative in lead V1, a rightward lead), sharp deep S waves in leads II, III and aVF (negative in the inferior leads), and a positive deflection in leads I, aVR and aVL (positive deflection in the superior and leftward leads). Because this pacing location is the site most commonly used during implantation, it is important to recognize this pattern. This will allow the quick recognition of variations of this pattern. It is essential to realize that not all RV pacing sites produce this signature pattern. Not recognizing the variations of this pattern is a common reason for misidentifying which lead in the biven-
The QRS duration is used sometimes to distinguish between a RV septal pacing site and a RV free-wall pacing site (Figs 7.71 and 7.72). As a generalization, septal pacing sites give rise to more narrow QRS complexes, and free-wall pacing sites are strongly positive in lead I, with wider QRS complexes.

**Left ventricular pacing: ECG patterns and their recognition**

The three main ventricular branches of the coronary sinus used for LV pacing are the anterior interventricular vein, posterolateral vein, and middle cardiac vein (Figs 7.73 and 7.74). The typical ECG signatures of pacing from these venous sites are outlined below.52
Fig. 7.72 In this example, when the output voltage is decreased from 4 V to 3.75 V, there is intermittent loss of left ventricular lead capture. This is evidenced by the appearance of the sensed electrogram on the ventricular pacing lead. In addition, a change in QRS duration is seen intermittently. With further decrease in voltage at 0.5 V, complete loss of capture is noted. In this particular device, the presence or absence of the sensed electrogram is also useful in determining capture.
It should be noted that most patients also have several subsidiary branches between these three main branches. Pacing from one of these other branches produces a hybrid ECG morphology that can be deduced from the patterns typical with pacing from the three main veins.

**Anterior interventricular vein**

This vein runs along the left anterior descending artery in the anterior interventricular groove, and its branches follow the path of the septal and diagonal branches of the left anterior descending artery (Figs. 7.73–7.77). Typically, left lateral branches of the anterior inter-
ventricular vein interdigitate with the corresponding branches of the lateral cardiac veins. Pacing from the anterior interventricular vein produces a vector that proceeds from the anterior myocardial wall toward the inferior wall. The typical ECG pattern shows a positive deflection (R wave) in leads II, III, and aVF. Lead V1 shows a positive deflection suggestive of a right bundle branch block pattern. If one of the lateral tributaries of the anterior interventricular vein is used for pacing, then lead I will be negative and lead III will show a taller R wave than lead II. To distinguish whether the lead is placed more apically or basally in this vein, the apical leads V4, V5, and V6 as well as lead aVR can be analyzed. With apical locations in the anterior interventricular vein, leads V4, V5, and V6 are typically negative and lead aVR is positive. With the more basal locations in the vein, lead aVR is negative and leads V4, V5, and V6 may be positive.

**Lateral and posterolateral cardiac vein**

The lateral and posterolateral cardiac venous tributaries follow the obtuse marginal or posterolateral coronary artery (or both). Pacing from this vein produces a negative deflection (S wave) in leads II, III, and aVF and lead I. This results from the posterolateral origin of the cardiac impulse and, therefore, conduction away from the posterior and inferior leads (leads II, III, and aVF) and from the lateral lead (lead I). In a more straight lateral position, lead III will be positive, as explained.
above, because of the relatively rightward orientation of the positive electrode in lead III. In more posterior and septal locations, lead III will tend to be negative. Thus, although leads II and aVF for posterolateral vein pacing are almost always negative, a more septal site will also produce negativity in lead III; however, a more lateral site will produce negativity in lead I and positivity in lead III.

Middle cardiac vein
The middle cardiac vein runs in the posterior interventricular groove and follows the course of the posterior descending artery (Fig. 7.78). Tributaries to this vein drain both at the posterior RV and posterior LV. Near the apex, the middle cardiac vein often communicates with apical branches of the lateral veins and the anterior interventricular vein. Pacing from this vein results in a vector that proceeds away from the inferoposterior wall of the heart. This produces a sharp negative deflection (S wave) in leads II, III and aVF. Lateral branches of the middle cardiac vein can often be used for pacing. These lateral branches may allow the lead to be passed to the lateral wall, where lead I will show a sharp negative deflection.

Differentiating middle cardiac vein pacing from RV pacing may be difficult. First, rightward branches of the middle cardiac vein drain the RV, and if a pacing lead is wedged in one of these branches, there is no discernible difference on the electrocardiogram between pacing from that site and an endocardial RV pacing location. Usually, however, middle cardiac vein pacing occurs either in one of its left lateral branches or in the main vein itself. Close analysis of the QS complex in leads II and III can help identify such pacing locations. While both RV endocardial pacing and middle cardiac
vein pacing give rise to negative deflection in leads II and III, the extent of negativity is greater in lead III compared with II with RV pacing. whereas lead II is more negative than III in middle cardiac vein pacing. This is because lead III is a relatively rightward lead compared with lead II. In addition, middle cardiac vein pacing, particularly from the left lateral branches, will almost invariably give rise to an R wave in lead VI (right bundle branch block pattern).

**Biventricular pacing: ECG characteristics**

The ECG in biventricular pacing, with both RV and LV stimulation sites, represents a summated vector of the individual access of activation (RV + LV). The QRS duration is typically shortened compared with stimulation at either a RV or LV site alone. However, this is not always true, and QRS duration alone cannot be used to determine whether stimulation is biventricular or from a single site. In the follow-up of patients with biventricular pacing systems, it is often necessary to analyze the ECG to judge whether biventricular stimulation is occurring. This requires a thorough understanding of the typical ECG signatures of various LV and RV sites. Furthermore, when certain characteristics of the ECG suggest RV apical pacing and yet others suggest LV stimulation (e.g., a negative deflection in lead III and also in lead I) or a biphasic pattern in certain leads, particularly lead I (see below), two different stimulation sites are being used. The situation is more complicated when the RV pacing site is not the RV apex, and with certain ECGs this distinction cannot be made unless the LV and RV anatomical sites of stimulation are known beforehand or a previous ECG demonstrates biventricular stimulation (Fig. 7.79).

**Right ventricular apical* and anterior interventricular site**

Lead V1 usually shows an atypical right bundle branch pattern.

* It is assumed that RV pacing is from a typical apical location. If the RV pacing site is elsewhere, differences are noted.
pattern with an R wave preceded by either a small negative or isoelectric segment. Leads II, III and aVF are either biphasic or isoelectric because the anterior interventricular site causes a positive deflection in these leads and RV apical pacing produces a negative deflection. If either LV conduction is slow or RV timing is ahead of LV timing (see below), the inferior leads will be more typical of a RV apical pacing site alone (deep S waves). Lead I will be either isoelectric or negative, suggesting an anterolateral site of stimulation from the LV lead.

**Right ventricular apical and lateral venous stimulation**

Biventricular pacing from these sites typically causes a negative deflection in lead I or a right bundle branch block pattern in lead V1, with a negative deflection (S wave) in leads II, III and aVF. Biventricular pacing from these two sites can be difficult to distinguish from posterolateral venous pacing alone. Typically, with posterolateral venous pacing alone, lead II is more negative (deeper S wave) than lead III. With RV pacing alone, lead III has deeper S waves than lead II. Biventricular stimulation from these sites results in nearly equal negativity in leads II and III. This pattern in conjunction with a negative deflection in lead I, which cannot occur with RV pacing alone, suggests biventricular stimulation and capture.

**Other right ventricular sites along with left ventricular stimulation**

The ECGs in this situation are difficult to analyze for biventricular capture without previous knowledge of the RV site. This situation is a frequent cause of error in misdiagnosing which lead is failing to capture. For example, when a high RV septal site along with a middle cardiac vein site for LV stimulation is used when the LV lead is failing to capture, then positive R waves in leads II, III and aVF will be seen from the high septal RV stimulation site. The positive deflection in lead III and the other inferior leads will give the mistaken impression of LV pacing and wrongly suggest failure to capture the RV lead. Present devices that allow separate stimulation of the LV and RV leads have greatly simplified diagnosing appropriate ventricular lead capture during threshold testing. During routine follow-up with only the ECG available, it is vital to have previous ECGs or documentation of the exact site where the leads were placed at implantation.

**Assessing ventricular synchrony with the ECG**

The initial premise of biventricular pacing was that simultaneous stimulation of the right and left ventricles would lead to mechanical synchronization of ventricular function. It is clear that simultaneous stimulation from disparate sites in the right and left ventricles will shorten the total duration of the QRS complex (ventricular depolarization) and predict a shortened synchronization. However, there are several situations in which electrical synchronization does not predict mechanical synchronization. There may be significant capture latency from the pacing site, that is, a time delay from the delivery of the pacing stimulus to ventricular capture diminishes the contribution to ventricular depolarization from that pacing site. In an extreme situation, there is no difference between single-site pacing and dual-site stimulation with one site having extreme capture latency. Capture latency as well as exit delay from a particular pacing site may be manifest only at more rapid pacing rates because of the decremental properties of the myocardium in certain diseases. Similarly, if stimulation is performed from a site, e.g., in the left ventricle, that is close to scarred or diseased myocardium, the relative contribution to global ventricular depolarization from that pacing site will be minimal (Fig. 7.80).

The contribution of one pacing site to overall ventricular capture can be estimated by comparing the 12-lead ECG with biventricular pacing with the ECG of pacing from the individual stimulation sites. If the biventricular-paced ECG (with capture confirmed with individual lead testing) is similar to right ventricular pacing alone, it suggests that the left ventricular lead is not contributing to ventricular depolarization. This could be due to capture latency or extreme delay in exiting from the local myocardial capture at the site of the lead. If this situation is recognized at implantation, an attempt should be made to move the lead to a different site. If this is found at follow-up after implantation and the capability for varying the ventricle-to-ventricle stimulation timing is available, the timing should be set to pace earlier in the lead that is contributing less to ventricular depolarization. For example, if the biventricular-paced ECG approximates the right ventricular pacing ECG, then left ventricular timing should be made to precede right ventricular timing.

The QRS duration has been used to determine whether ventricular synchronization is occurring (Fig. 7.71), but using the QRS duration alone has pitfalls. RV sep-
tal pacing may produce a more narrow QRS complex than the addition of a LV lead. This may be due to functional refractoriness of the LV caused by LV stimulation producing an overall delay in total ventricular activation.

In addition to the above-mentioned caveat for using QRS duration as a surrogate for ventricular synchronization, the following need to be considered. Portions of the QRS may be isoelectric with biventricular pacing because of the summated vectors of different sites such as the RV apex and an anterior interventricular vein site. This may give a false impression of a narrow QRS complex when in fact portions of the QRS complex are not identified on the 12-lead ECG. This error is compounded when one or only a few leads are used to measure the QRS complex. Perhaps more importantly, it is well recognized that mechanical synchronization is not necessarily implied by electrical synchronization, because electrical activation at a particular site variably produces contraction depending on the electromechanical coupling interval. This interval, in turn, depends on various factors, including ischemia, scar tissue, dysplasia, and fiber orientation. Despite these limitations, if the ECG is analyzed accurately, it can give an approximate idea of resynchronization (Figs 7.81 and 7.82).

In summary, the 12-lead ECG should be analyzed first for a decrease in QRS duration with biventricular pacing. This suggests that global depolarization is being shortened with the use of the two sites. Next, the biventricular ECG should be compared with the ECG from pacing at individual sites. The biventricular paced ECG should be the sum vector of the individual pacing site ECGs. If the biventricular ECG is similar to either right or left ventricular pacing alone, it suggests that the lead that is not manifest with biventricular pacing is not contributing to global depolarization significantly. Either lead repositioning or ventricle-to-ventricle timing should be adjusted to produce better evidence of a balanced biventricular contribution to ventricular depolarization.

Biatrial pacing

The ECG principles outlined above are equally applicable to atrial pacing. The P wave vector from right atrial appendage pacing results in a positive P wave in lead I, a negative wave in leads aVR and V1, and a positive wave in leads II, III and aVF. Pacing from the coronary sinus ostium, as with dual-site atrial pacing, typically results in a biphasic or negative P wave in leads II, III and aVF and a biphasic initially positive and then negative deflection in lead V1. Left atrial
pacing, for example, from a lateral atrial vein such as the vein of Marshall, produces a negative P wave in leads I and aVL.

Coronary sinus musculature

The musculature of the coronary sinus is continuous with the right atrium. Very proximal locations for attempting ventricular pacing in a ventricular vein occasionally may stimulate the local atrial myocardial extension into the coronary sinus, resulting in atrial activation that in turn may activate the ventricle. This may be mistakenly interpreted as local ventricular capture. Although this situation is rare, ventricular activation may also occur because of coronary sinus musculature via a closely related but different ventricular vein than where the pacing lead is located. This will result in slight, usually subtle, changes in the ECG pattern during pacing from a particular location. This may be mistaken interpretation can be avoided by paying careful attention to all details of the ECG (Figs 7.83–7.86).

Atrioventricular synchrony on the electrocardiogram

As mentioned above, the atrial site of pacing can be deduced with much the same principle as explained in detail for determining the ventricular location of stimulation. Further analysis of the dual-chamber paced electrocardiogram can be used to obtain information on electrical atrioventricular synchrony. When atrioventricular synchronization is in question or needs optimization, usually the pulsed wave Doppler of the mitral valve is used to ascertain that atrial emptying is complete (without diastolic mitral regurgitation) prior to the onset of ventricular contraction. By similar reasoning, if the P wave has not yet been fully inscribed prior to the ventricular
pacing spike and onset of the paced QRS, it follows that some atrial mechanical activations (contraction) would not yet have completed prior to ventricular contraction and associated closure of the mitral valve.

Similarly, if after the paced P wave there is a prolonged isoelectric period prior to the onset of ventricular pacing, a probably unnecessary lengthening of the A-V interval with the potential for diastolic mitral regurgitation exists. Because of differences in electromechanical coupling, electrocardiographic analysis of the A-V interval cannot supplant echocardiographic evaluation, but can provide a quick overview to recognize situations with obviously inappropriately long or short A-V intervals.

One manufacturer has developed a method for optimization of the AV/PV and VV delay values which is programmer based using intracardiac electrograms. Studies have been performed demonstrating a good correlation with echocardiographic optimization of these programmable intervals.

**Anodal stimulation**

When pacing in a bipolar configuration, stimulation typically occurs from the negative electrode or cathode (tips). With biventricular systems, a commonly used configuration is to pace from the tip of the left ventricular lead to the ring or coil of the right ventricular ICD lead. In some patients when pacing either at high output (twice or thrice threshold) or close to threshold (just prior to loss of capture), a marked change in the QRS morphology can be seen even though LV pacing with the same configuration (LV tips negative) is occurring. The change in morphology is between the typical pattern seen with LV pacing to a morphology more similar to RV pacing. This phenomenon is known as anodal stimulation where despite pacing with the LV
Fig. 7.83 The classic pattern with right ventricular pacing from the right ventricular apex is shown in (A). A left bundle branch block morphology with negative deflections in leads II, III and aVF and a tall R wave in leads I and aVL all signify right ventricular apical pacing. Biventricular capture is seen in (B) (after the first beat). There is a dramatic shortening in the QRS complex and a marked change in the vector. Lead I now becomes negative, suggesting lateral ventricular stimulation, and leads II, III and aVF become positive, suggesting an anterior location. Note that despite the anterolateral rather than straight lateral location, excellent shortening of the QRS duration is seen. The 9th, 11th and 12th beats in (B) are premature atrial contractions with biventricular capture. Note that the QRS complex is slightly more prolonged with the faster pacing rate triggered by the premature atrial contractions in patients with ventricular conduction abnormalities or those taking antiarrhythmic drugs. This effect can be pronounced. This is one of the limitations to biventricular pacing in patients with atrial fibrillation and relatively rapid ventricular responses necessitating rapid pacing rates. (Continued)
Fig. 7.83 (Continued.)
Fig. 7.84 The same example as in Fig. 7.15 showing the effect of increasing the pacing output (A) and then decreasing it (B). Because of the given lead position in this example, any one of the leads can be used to quickly see whether biventricular stimulation is occurring. For example, the negativity in lead I and the positivity in lead III are easily observed when increasing the output and obtaining left ventricular stimulation. (Continued.)
Fig. 7.84 (Continued)
This example illustrates the importance of systematic analysis of biventricular (BiV) tracings. With right ventricular (RV) pacing, a relatively characteristic morphology is seen, with a left bundle branch block pattern and negative deflections in leads II, III and aVF. However, careful analysis shows an early small positive deflection in lead V1 and varying degrees of QRS widening, especially evident in lead V2. Both these findings suggest conduction abnormalities at the RV exit site. With left ventricular (LV) pacing, a characteristic right bundle branch block pattern with negative deflections in leads I and aVL is seen. The QRS vector is upright in the inferior leads (leads II, III and aVF). This suggests that the LV pacing lead has been placed in a lateral vein in a slightly anterior location. BiV pacing results in an ECG that is very similar to LV pacing alone. This suggests that the RV lead is contributing little to the overall pacing vector. Thus, there is no added shortening of the QRS complex duration, a surrogate for resynchronization when adding RV pacing to LV pacing alone. In such instances, the availability of varying the ventricle-to-ventricle stimulation interval (VV timing) will allow better QRS shortening. In this instance, stimulating the RV earlier than the LV lead will approximate simultaneous stimulation of the ventricles. This ability to vary the VV timing may be more important when there is LV delay and the BiV-paced QRS morphology resembles RV pacing alone. This is because QT dynamic data show similar benefits with LV pacing and BiV stimulation.
Fig. 7.86 (A–C) This series of ECGs illustrates the difficulty with using a single lead to assess left ventricular capture thresholds. Biventricular stimulation is seen in (A). (B) shows that with decreasing the output of this biventricular system, a change in morphology occurs after the first two QRS complexes. The question is, which lead has lost capture? Is it the right ventricular or the left ventricular pacing lead? (C) shows that further decrease in the output voltage results in the loss of capture altogether, and intrinsic rhythm with left bundle branch block ensues. (Continued.)
Fig. 7.86 (Continued.) If one is asked to assess whether the right or left ventricular lead has lost capture (B), it would be nearly impossible to answer this question from analysis of lead III alone, because lead III continues to be positive. This suggests that the right ventricular lead (usually resulting in negative complexes in lead III) has lost capture. However, in lead V1, the resulting QRS complexes are negative (left bundle branch block pattern). In fact, the loss of capture was in the left ventricular lead. However, because the right ventricular lead had been placed in the high interventricular septum, the QRS complex is positive in lead III. Accurate knowledge of the position of the right ventricular lead and analysis of the entire 12-lead ECG are necessary in atypical situations. (Continued.)
Fig. 7.86 (Continued)
lead tip as the cathodes, the activation wave front proceeds from the RV anode. This type of change in pacing vector (anodal stimulation) has profound implications not only for EKG interpretation, but for ventricular synchronization, since it is essentially represents RV pacing. This phenomenon is important to recognize at implant whenever the pacing configuration adopted is or may be (future option) a widely spaced configuration such as LV tip to RV ring, etc. At implant, even if the pacing threshold appears excellent, if the QRS morphology suggests RV pacing, the programmed output should be higher than threshold and higher than the threshold at which true cathodal stimulation with a desirable QRS morphology (reflecting LV pacing) is visualized.

**Conclusion**

Although the paced ECG may have infinite variations depending on the type of device therapy, pacing mode, pacemaker, ICD or CRT model, combination of programmed parameters, and whether function is normal or abnormal, understanding basic timing cycles and a systematic approach to the ECG should allow successful interpretation. When difficulty in ECG interpretation persists despite a systematic approach, the technical manual and/or the manufacturer should be consulted for assistance.

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Programmability is defined as the ability to make noninvasive, stable but reversible changes in pacemaker function. Using the pulse generator programmer, one can change many aspects of device function. Although it is technologically possible to completely reprogram the software of a pulse generator, there are no approved mechanisms by which this can be done at present. The first modern programmable pacemakers were introduced in 1972. In this pacemaker, a magnetic code was introduced from an external programmer to manipulate four levels of output and six rates. Since 1972, innumerable changes have evolved in programming capabilities. Radiofrequency signals are now exclusively used to communicate between the pacemaker and the programmer. The number of programmable features and the variability of each feature have expanded to the point that the programmable combinations are extraordinary and offer the ability to alter the programming to meet the specific needs of the patient.

**Pacemaker programming**

All contemporary pacemakers manufactured by major manufacturers are programmable. The North American Society of Pacing and Electrophysiology/British Pacing and Electrophysiology Group (NASPE/BPG) code designates the degree of programmability and rate modulation in the fourth position of the code. In practice, only the designation "R" is used in this position. An "R" in the fourth position indicates that the pacemaker has a special sensor to control the rate independent of intrinsic electrical activity of the heart. Virtually all pacemakers with a sensor also have extensive telemetric and programmable capabilities. Designations of "O," "P," "M," and "C" are described, but rarely used. "O" indicates that none of the parameters of the pacing system can be noninvasively altered. "P" is simple programmability; one or two parameters can be changed, but this code does not specify which ones. "M," multiparameter programmability, indicates that three or more parameters can be changed. "C" reflects the ability of the pacemaker to communicate with the programmer; namely, it has telemetry. By convention and in actual operation, it also means that the pacemaker has multiparameter programmability.

It is important to have a systematic approach to programming. Many devices provide a methodical way of checking programmed and measured data. Although the manufacturer-specific approaches differ, a programming sequence should include specific assessments, data collection and interpretation and optimization if needed.

Most programmable parameters have been discussed in Chapter 7, "Pacemaker and Cardiac Resynchronization Timing Cycles and Electrocardiography" and it is not the purpose of this chapter to discuss each programmable value individually. Nor is there an attempt to cover nuances of specific manufacturers or...
models. As always, it is important to consult the technical manual and/or the manufacturer helpline for technical services for further information about a specific pulse generator or specific feature. Table 8.1 attempts to include most available programmable features and a range of the programmable values for each parameter.

The chapter is structured by going through major programmable parameters and programming considerations for each. Programmer “screen shots” will be used to demonstrate the type of information available and steps to assessment and optimization when needed.

Interrogation

When the programming head is placed over or near the pulse generator, if the device is equipped with a radiofrequency link that allows programming to be done without the programmer head directly over the device, most contemporary pulse generators are automatically recognized by the programmer and either a full interrogation or a display of programmed parameters will be generated.

The initial screen usually displays key programmed parameters, alerts that have occurred since the last interrogation and options to access other data (Fig. 8.1).

Emergency programming

If, during the course of programming in a pacemaker-dependent patient, the pacemaker is programmed in such a way that ventricular asystole occurs, every programmer is equipped with an emergency or “stat set” button that restores nominal pacing parameters if activated. However, there may be a delay between activation of the stat set parameters and actual restoration of nominal pacing parameters. It is important that the

<table>
<thead>
<tr>
<th>Table 8.1 Pacing modes*</th>
</tr>
</thead>
<tbody>
<tr>
<td>VOO Ventricular pacing; no sensing</td>
</tr>
<tr>
<td>VVI Ventricular pacing; ventricular sensing and inhibition</td>
</tr>
<tr>
<td>VVT Ventricular pacing; ventricular sensing and triggering</td>
</tr>
<tr>
<td>VVIR Ventricular pacing; ventricular sensing with inhibition; rate-modulated pacing</td>
</tr>
<tr>
<td>VOOR Ventricular pacing; no sensing; rate-modulated pacing</td>
</tr>
<tr>
<td>AOO Atrial pacing; no sensing</td>
</tr>
<tr>
<td>AAI Atrial pacing; atrial sensing and inhibition</td>
</tr>
<tr>
<td>AAT Atrial pacing; atrial sensing and triggering</td>
</tr>
<tr>
<td>AAIR Atrial pacing; atrial sensing with inhibition; rate-modulated pacing</td>
</tr>
<tr>
<td>AOOR Atrial pacing; no sensing; rate modulation</td>
</tr>
<tr>
<td>OOO Pacemaker is programmed “off” (allows assessment of underlying rhythm)</td>
</tr>
</tbody>
</table>

AV, atrioventricular.

*Reference is sometimes made to the SSI, SSIR, SST, or SOO mode. Manufacturers use “S” in both the first and the second positions of the pacemaker code to indicate that the device is capable of pacing a single cardiac chamber. Once the device is implanted and connected to a lead in either the atrium or the ventricle, “S” should be changed to either “A” or “V” in the clinical record to reflect the chamber in which pacing and sensing are occurring.

healthcare professional performing the threshold determination be familiar with the specific programmer and the steps necessary to activate stat set or emergency back-up parameters.

**Programmed parameters**

One or more screens specifically display programmed parameters. Drop-down menus or other paths display more obscure programmable parameters or "advanced" programmable parameters.

Parameters may be reprogrammed either directly from the "parameter" screen or from "temporary programming" screens that permit routine programming sequences or troubleshooting without permanently changing programmed values.

**Measured data**

Measured data are displayed in most pacemakers. Some of these data are used to modify or verify the appropriateness of programmed parameters and are therefore discussed in this chapter.

- **Impedance** – Impedance values for atrial and/or ventricular leads depending on type of pacemaker in place should be obtained. When the values are displayed, some pulse generators will also display prior value or values or a "trend" of impedance measurements (Fig. 8.3).
  - P and/or R wave amplitude – The size, in mV, of intrinsic activity should be measured and compared with prior values when available (Fig. 8.4).
  - Capture threshold(s) – There is usually a link on the same screen to options for measuring the capture (stimulation) threshold. There are usually multiple ways the autothreshold can be "set-up," depending on how one prefers to obtain and monitor capture threshold. At our institution we generally do capture thresholds with a fixed pulse width and decrement the voltage amplitude. (In some devices this may not be an option.) Some manufacturers require that a touch-sensitive area on the programming screen be held down in order for the autothreshold to be done, with the rationale that as soon as capture is lost the operator simply has to remove the pressure, be it stylus or finger touch. Others simply require that the autothreshold be initiated and the threshold search will end automatically upon detection of loss of cap-
In some pacemakers a screen may also be available that will display prior values for capture thresholds and other previously measured data (Fig. 8.6).

**Specific programmable parameters to consider in all patients**

As noted, the degree of programmability varies significantly among pulse generators. In this chapter, pacing mode options are listed in Table 8.1 and Table 8.2 lists commonly programmable parameters, a definition and potential programmable values. The degree of programmability varies not only between manufacturers but between various generations of pulse generators from the same manufacturer. The chapter is written with regard to current generation pulse generators, realizing that the degree of programmability and the sophistication of programmable parameters may be significantly less on earlier generations of pulse generators that remain in service. Although an attempt is made to discuss programming generically, this is not always possible. Specific programmable variables may be protected by trademark and available from only one manufacturer. Therefore, it is necessary at times to refer to specific manufacturers and specific algorithms.

**Mode programming**

Pacing modes have been discussed in detail in Chapter 7, "Pacemaker and Cardiac Resynchronization Timing Cycles and Electrocardiography."

Of the many programmable mode options listed in Table 8.1, there is an appropriate time for every one of these modes to be used either permanently or temporarily. For programming purposes, consider the potential uses for the following modes:

- **VVI**—determination of ventricular pacing threshold
- **VVT**—determination of ventricular sensing threshold; determine site of ventricular sensing; for temporary diagnostic use in the evaluation and management of arrhythmias performed by triggering the device output through chest wall stimulation
- **AAI**—determination of atrial pacing threshold

Temporary programming allows testing and specific programming sequences to be accomplished without having to alter the prior settings.
Fig. 8.3 A follow-up screen from a specific pacemaker. Measured impedance values are provided, in this example atrial impedance = 498 Ω; ventricular impedance = 476 Ω. In addition, the measured unipolar intrinsic P and R waves are shown. In the “box” to the right there is a list of events that have occurred.

Fig. 8.4 An intrinsic amplitude test has been performed which measured a 3.1-mV unipolar P wave and a 4.8-mV bipolar R wave.
Fig. 8.5 Programmer screen from which threshold testing can be accomplished. In this example ventricular pacing threshold is being performed by decrementing the voltage amplitude at a fixed pulse width of 0.4 ms.

Fig. 8.6 Programmer screen demonstrating stored follow-up data. Information is available from three programming episodes and includes lead impedances, lead polarity, intrinsic P- and R-wave amplitude, atrial and ventricular pacing thresholds and battery status.
Table 8.2 Common programmable options for pacemakers

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
<th>Potential programmable values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atrial refractory period</td>
<td>An interval of the atrial channel timing cycle during which the atrial sensing amplifier is designed to be unresponsive to input signals. In some pacemakers, however, the sensing circuitry is alert for extraneous or noncardiac signals during a portion of the atrial refractory period. In single-chamber atrial pacing modes, the atrial refractory period occurs after a sensed or paced atrial event.</td>
<td>150–500 ms</td>
</tr>
<tr>
<td>Atrioventricular interval</td>
<td>Period between the initiation of the paced or sensed atrial event and the delivery of a consecutive ventricular output pulse.</td>
<td></td>
</tr>
<tr>
<td>Circadian lower rate limit or sleep rate</td>
<td>Reduces the lower rate limit during sleeping or resting hours</td>
<td>PAV (Paced AV) also “AV” – 25–350 ms; SAV (Sensed AV) also “PV” – 25–325 ms.</td>
</tr>
<tr>
<td>Differential AVI</td>
<td>Feature that permits a longer AVI after a paced atrial event (PAV) than after a sensed AVI (SAV). In many pacemakers these are independently programmable (see immediately above). In some the differential is fixed; and in others, it is programmable as a single AV interval with an “offset”</td>
<td>Offset from 0 to 100 ms</td>
</tr>
<tr>
<td>Fallback</td>
<td>An upper rate response in which the ventricular paced rate decelerates to, and is maintained at, a programmable fallback rate that is lower than the original programmed MTR. Fallback mechanisms vary among pacemakers. Fallback may also be a programmable parameter related to the mode-switch algorithm indicating the paced rate that will be implemented when mode-switching criteria are met.</td>
<td>May be programmable on or off; if on, the rate to which the fallback occurs may be fixed or programmable, i.e., 50–80 ppm</td>
</tr>
<tr>
<td>Lower rate limit</td>
<td>Preset or programmed rate at which a pacemaker emits an output pulse without intrinsic cardiac activity.</td>
<td>30–170 ppm (options faster than 150 ppm available in some pulse generators)</td>
</tr>
<tr>
<td>Magnet response</td>
<td>The response of a permanent pacemaker to magnet application is generally asynchronous pacing, and the behavior of an implantable cardioverter-defibrillator varies among manufacturers and possibly among models or generations of a specific manufacturer</td>
<td></td>
</tr>
<tr>
<td>Maximum sensor rate (MSR)</td>
<td>The fastest sensor-driven pacing rate that can be achieved in a rate-adaptive pacing system. In a single-chamber demand pacemaker with rate-adaptive capability (SSIR), the maximum sensor rate is the same as the programmed upper rate limit. In a dual-chamber rate-adaptive pacemaker (DDDR), the maximum sensor rate is not necessarily equal to the maximum tracking rate.</td>
<td>80–180 ppm</td>
</tr>
<tr>
<td>Maximum tracking rate (MTR)</td>
<td>The fastest atrial rate at which consecutively paced ventricular complexes maintain 1:1 synchrony with sensed atrial events. The maximum tracking rate is a function of dual-chamber pacing modes and can be defined as a preset or programmable value. The maximum tracking rate is limited to the total atrial refractory period (TARP).</td>
<td>80–210 ppm</td>
</tr>
</tbody>
</table>
### Mode (see Table 8.1)

Preset or programmed response from a pacemaker with or without intrinsic cardiac events.

### Modes switch

Capability of a dual-chamber pacemaker to automatically switch from an atrial tracking (P-synchronous) mode to a non-atrial-tracking mode when an atrial rhythm occurs that the pacemaker determines to be pathological. When the atrial rhythm meets the criteria for a physiological rhythm, the mode switches back to an atrial-tracking mode.

### PMT (Pacemaker Mediated Tachycardia)

A function of dual-chamber pacemakers that minimizes the initiation or continuation of a sensed premature ventricular contraction, and a dropped ventricular output pulse after a predetermined number of beats at a specified rate or upper rate limit.

### Polarity

Stimulating electrode typically is the cathode, which has negative polarity relative to the indifferent electrode (anode). If the anode is the “ring” of the pacing lead then a “bipolar” configuration is in use. If the anode is the pulse generator “can,” then a “unipolar” configuration is present.

### Postventricular atrial blanking period (PVAB)

A programmable feature in some dual-chamber pulse generators. During the period specified (in milliseconds), the atrial events are blanked from the atrial channel and therefore not considered when the atrial rate interval is calculated. The postventricular atrial blanking period is initiated with a ventricular paced or sensed event.

### Postventricular atrial refractory period (PVARP)

In dual-chamber pacemakers, that portion of the timing cycle during which the atrial channel is refractory after a paced or sensed ventricular event. The PVARP prohibits the atrial channel of the pacemaker from sensing the far-field ventricular depolarization or the afterpotential of the ventricular pacing impulse. If the PVARP is sufficiently long, it can prevent pacemaker-mediated tachycardia by prohibiting sensing of premature atrial beats or retrograde atrial depolarizations after ventricular ectopic or paced ventricular beats. However, extension of the PVARP limits the maximum tracking rate unless there is a rate-related shortening of the PVARP.

### Pulse amplitude

Magnitude of the voltage level reached during a pacemaker output pulse, usually expressed in volts.

### Pulse width

Duration, in milliseconds, over which the voltage output is delivered.

### PVARP extension

Lengthening of the PVARP after a sensed premature ventricular contraction to prevent sensing of a retrograde P wave.
Rate hysteresis: Extension of the escape interval after a sensed intrinsic event. Off, 30–130 ppm. (Most commonly used is in SSI device with rates 30–60 ppm. However, in some, if N = the base rate, hysteresis is available at N – 5 or 10 ppm up to as the programmable base rate)

Rate smoothing: Prevents atrial or ventricular paced rate from changing by more than a programmed percentage from one cardiac cycle to the next. This prevents large cycle-to-cycle intervals that can be seen at the upper rate limit or during rapid acceleration of atrial rate. On or off; when on options of 1% smoothing, i.e., 3%, 6% and 24% change per cycle length allowed; may also have option of being on or off for rate increments or decrements, or both.

Rate-adaptive AVI: Shortens the AVI as the heart rate increases. On or off only in some devices; in others, able to set the minimum AV delay to as short as 30 ms and in others there is a manufacturer-determined “scale,” e.g., low, medium, high that determines how aggressive the AVI shortening will be.

Reaction time: A programmable parameter in some rate-adaptive pacemakers which determines how quickly the pacing rate will increase via sensor activation. 15–60 s.

Recovery time: A programmable parameter in some rate-adaptive pacemakers that determines how quickly the sensor-driven pacing rate will decrease once the sensor is no longer activated. 2.5–16 min.

Sensitivity: Ability to sense an intrinsic electrical signal, which depends on the amplitude, slew rate, and frequency of the signal. Atrial: 0.1–8 mV; ventricular: 0.5–14 mV.

Sensor slope: A programmable value that determines the pacing increment over the base rate which will occur with different levels of sensor signal input. Usually a scale unique to the manufacturer, e.g., 1–10, 1–16.

Sensor threshold: A programmable value for rate-adaptive pacemakers which determines, in part, the level of activity necessary to activate the sensor. Programming the sensor threshold is not consistent across manufacturers. In some devices, the higher the sensor threshold is set, the greater the level of activity required to increase the pacing rate, and vice versa in others. Low, medium, high.

Ventricular blanking: 20–50 ms.

Ventricular refractory period: An interval of the timing cycle following a sensed or paced ventricular event. The ventricular channel is totally unresponsive to incoming signals or waveforms during the majority of the ventricular refractory period. However, in some pacemakers, the sensing circuitry is alert for extraneous signals during a portion of the ventricular refractory period. The ventricular refractory period also may be referred to as the ventricular refractory interval. 125–500 ms.

Ventricular safety pacing (VSP): Delivery of a ventricular output pulse after atrial pacing if a signal is sensed by the ventricular channel during the cross-talk sensing portion of the AVI. On or off in most pulse generators. When on the VSP interval is usually in the range of 90–120 ms. One manufacturer does not have VSP as a programmable option, but as a function of a noise detection algorithm.

Table 8.2 (Continued.)

<table>
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<th>Parameter</th>
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<td>Rate smoothing</td>
<td>Prevents atrial or ventricular paced rate from changing by more than a programmed percentage from one cardiac cycle to the next. This prevents large cycle-to-cycle intervals that can be seen at the upper rate limit or during rapid acceleration of atrial rate</td>
<td>On or off; when on options of 1% smoothing, i.e., 3%, 6% and 24% change per cycle length allowed; may also have option of being on or off for rate increments or decrements, or both.</td>
</tr>
<tr>
<td>Rate-adaptive AVI</td>
<td>Shortens the AVI as the heart rate increases</td>
<td>On or off only in some devices; in others, able to set the minimum AV delay to as short as 30 ms and in others there is a manufacturer-determined “scale,” e.g., low, medium, high that determines how aggressive the AVI shortening will be</td>
</tr>
<tr>
<td>Reaction time</td>
<td>A programmable parameter in some rate-adaptive pacemakers which determines how quickly the pacing rate will increase via sensor activation</td>
<td>15–60 s</td>
</tr>
<tr>
<td>Recovery time</td>
<td>A programmable parameter in some rate-adaptive pacemakers that determines how quickly the sensor-driven pacing rate will decrease once the sensor is no longer activated</td>
<td>2.5–16 min</td>
</tr>
<tr>
<td>Sensitivity</td>
<td>Ability to sense an intrinsic electrical signal, which depends on the amplitude, slew rate, and frequency of the signal</td>
<td>Atrial: 0.1–8 mV; ventricular: 0.5–14 mV</td>
</tr>
<tr>
<td>Sensor slope</td>
<td>A programmable value that determines the pacing increment over the base rate which will occur with different levels of sensor signal input</td>
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<tr>
<td>Sensor threshold</td>
<td>A programmable value for rate-adaptive pacemakers which determines, in part, the level of activity necessary to activate the sensor. Programming the sensor threshold is not consistent across manufacturers. In some devices, the higher the sensor threshold is set, the greater the level of activity required to increase the pacing rate, and vice versa in others</td>
<td>Low, medium, high</td>
</tr>
<tr>
<td>Ventricular blanking period</td>
<td>A short preset or programmable interval in dual-chamber pacemakers during which the ventricular sensing amplifiers are disabled. Ventricular blanking is initiated by an atrial output pulse and is designed to eliminate ventricular sensing of the atrial stimulus (cross-talk)</td>
<td>Ventricular blanking: 20–50 ms</td>
</tr>
<tr>
<td>Ventricular refractory period</td>
<td>An interval of the timing cycle following a sensed or paced ventricular event. The ventricular channel is totally unresponsive to incoming signals or waveforms during the majority of the ventricular refractory period. However, in some pacemakers, the sensing circuitry is alert for extraneous signals during a portion of the ventricular refractory period. The ventricular refractory period also may be referred to as the ventricular refractory interval</td>
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</tr>
</tbody>
</table>
• AAT – determination of atrial sensing threshold; determine site of atrial sensing
• DDI – allow intrinsic ventricular activity to occur, e.g., during an automated look for underlying ventricular rhythm, DDI may allow the appearance of intrinsic ventricular rhythm
• ADI* – display ventricular diagnostics when programmed to the AAI mode
• VDI* – display atrial diagnostics when programmed to the VVI mode
• DAT* – potentially useful when there is a need for dual-chamber stimulation in the absence of ventricular activity
• ODO, OVO, OAO – temporary diagnostic evaluation of underlying rhythm and when a record of the intrinsic activity is needed

Rate programmability
During programming for pacemaker follow-up, if the patient’s intrinsic rate is greater than the programmed rate, the pacing rate is increased to assess the threshold of stimulation. If pacing is at the programmed lower rate, the rate should be decreased to determine the status of the patient’s underlying conduction. Ideally, this should be known prior to checking stimulation threshold; for example, if the patient is pacemaker-dependent and has no reliable ventricular escape rhythm, loss of capture during threshold determination could have clinical consequences. If thresholds are being obtained with an automated method this is less of a concern than if thresholds are determined manually (Fig. 8.7).

The nominal lower rate, i.e., the lowest ventricular rate allowed of most pacemakers, single and dual, is frequently 60 ppm. Programming to a slower rate may be helpful in an attempt to allow a patient with rare episodes of bradycardia to remain in sinus rhythm rather than in paced rhythm. Programming a rate of 50 ppm or even one as low as 40 ppm may allow the patient’s intrinsic rhythm to exist much of the time, with pacing only in the event of a more profound sinus bradycardia.
dia or asystole. Hysteresis, see below, and ventricular avoidance pacing algorithms (see Chapter 7) allow an even greater ability to promote intrinsic rhythm.

More rapid “lower” pacing rates, i.e., > 70 ppm, are used most commonly in pediatric patients and are sometimes useful when faster pacing rates may be necessary to enhance cardiac output, e.g., postoperatively. In an occasional patient, a faster rate may be used to suppress an atrial or ventricular arrhythmia.

An option for “circadian response,” or “sleep rate,” is available in many pacemakers (Fig. 8.8). This feature allows a lower rate to be programmed for the approximate time during which the patient is sleeping. A separate, potentially faster lower rate limit may then be programmed for waking hours. (For example, the lower rate limit may be programmed to 60 ppm during waking hours and 40 ppm during sleeping hours.) In some pacemakers, this feature is tied to a “clock,” and the usual waking and sleeping hours are programmed into the pacemaker. In other pacemakers, the sleep rate is also set on the basis of waking and sleeping hours, but verification by a sensor is required to allow rate changes to occur.

In any rate-adaptive pacemaker and in dual-chamber devices capable of atrial tracking, an upper rate limit must also be programmed. The upper rate defines the fastest paced ventricular rate allowed. Determining the appropriate upper rate depends on the patient’s exercise requirements and associated cardiac and other medical problems. The total atrial refractory period (TARP), which is the postventricular atrial refractory period (PVARP) plus the atrioventricular (AV) interval, effectively determines the maximum achievable tracking rate (see Chapter 7). In dual-chamber rate-adaptive pacemakers, the upper rate limit may be a single programmable value, or independent programming of the maximum tracking rate and maximum sensor-driven rate may be required (see Chapter 7).

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Fig. 8.8 Programmer screen from which the “Basic rate” and “Night rate” can be programmed. In this example the base rate is 60 ppm and the sleep rate is programmed to 50 ppm. Tied to the “clock” in the pulse generator the lower rate will change to 50 ppm at 22.00 h and back to 60 ppm at 06.00 h.
The nominal upper rate limit, i.e., the upper rate programmed by the manufacturer, is often in the range of 120–130 ppm. For many active individuals a more aggressive upper rate limit should be considered. In Chapter 9, “Rate-adaptive Pacing,” we discuss exercise assessment for patients with rate-adaptive pacemakers. At times it will be helpful to assess the patient’s rate with an ambulatory monitor, informal exercise or a formal treadmill exercise test. Identifying symptom correlation to pseudo-Wenckebach or 2:1 upper rate behavior would lead to alteration of the upper rate limit and other programmable features that might require adjustment to reach a faster upper rate. For example, if the patient has normal sinus node function but the upper rate limit is limited by the TARP, reprogramming the AV interval and/or the PV ARP may be required (Fig. 8.9). [The sensed AV interval (SAV) is often the most commonly altered parameter to adjust this issue.]

**Programming hysteresis, AV search hysteresis, ventricular pacing avoidance algorithms and sudden bradycardia response algorithms**

Programming hysteresis is discussed separately because this feature, one that has been available for decades, remains a source of confusion. Hysteresis permits prolongation of the first pacemaker escape interval after a sensed event. A pacemaker programmed at a cycle length of 1000 ms (60 ppm) and a hysteresis of 1500 ms (40 ppm) allows 500 ms more for another sensed QRS complex. Should another QRS complex not be recognized, the pacemaker stimulates continuously at the programmed rate of 60 ppm, an escape interval of

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**Fig. 8.9** Electrocardiographic tracing obtained during a treadmill exercise test in a 17-year-old patient complaining of sudden onset fatigue during intense exercise. In this example the patient had reached the programmed maximum tracking rate and displays pseudo-Wenckebach behavior. The sensor-indicated rate was not fast enough to prevent a significant alteration in V–V cycle length. The effective sudden decrease in ventricular rate paired with the irregular cycle length resulted in the patient’s exertional symptoms.
1000 ms (see Chapter 7, “Pacemaker and Cardiac Resynchronization Timing Cycles and Electrocardiography,” Fig. 7.62), until a sensed event restarts the cycle. The advantage of hysteresis in a single-chamber pacing mode is the ability to maintain spontaneous AV synchrony as long as possible. In patients with VVI pacing and pacemaker syndrome, hysteresis may prevent symptomatic retrograde ventriculoatrial (VA) conduction and increases the potential for maintaining the patient’s intrinsic rhythm.

Several types of AV search hysteresis and ventricular pacing avoidance algorithms are programmable options in dual-chamber pacemakers. These are detailed in Chapter 7. The purpose of such algorithms is to minimize ventricular pacing, i.e., maintain intrinsic ventricular conduction (Fig. 8.10). There is an increasing body of literature demonstrating the advantages of minimizing ventricular pacing whenever possible.

When programming, it is reasonable to extend the AV interval to the maximum to assess intrinsic conduction or, if available, to utilize a device-directed feature to optimize intrinsic conduction (Fig. 8.11). If the patient displays intrinsic AV nodal conduction it is reasonable to program “on” the algorithm available to minimize ventricular pacing. The often asked question is “How long can the AV interval be extended before there are adverse consequences?” There is no evidence-based answer to this question of which we are aware. With most available methods and algorithms for minimization of ventricular pacing, criteria can be established that put a limit on how far the paced atrial event (PAV)/SAV may be extended. We will frequently allow PAV/SAV intervals to 300 ms. At longer intervals one needs to be certain that AV synchrony is not compromised. Occasionally, it is helpful to use echocardiographic techniques to optimize the AV interval in pacemaker patients. The goal of echocardiographic guidance is to optimize atrial contribution to ventricular filling and to minimize or at least limit diastolic mitral insufficiency.

It is important to remember that available AV search hysteresis and ventricular pacing avoidance algorithms are well-designed and can be safely turned on in pa-

![Fig. 8.10 Programmer screen demonstrating that the pacing mode is "AAISafeR," which is a mode to minimize ventricular pacing. In addition the patient has a "rest rate" of 55 ppm and a basic rate of 70 ppm. The lower "rest rate" should promote less atrial pacing rate during sleeping hours.](image-url)
In addition to minimizing ventricular pacing, there are patients who will benefit by allowing their native sinus activity to have preference over paced atrial activity. Several manufacturers have algorithms to maximize the presence of native atrial rhythm.\(^\text{11}\) Criteria are programmed that periodically slow the paced rate to a preset level to determine whether or not sinus activity is present. If sinus activity is present, and if the intrinsic atrial rate meets criteria, atrial pacing would be inhibited until the patient’s intrinsic rate fell to a specific rate that would again trigger atrial pacing (Fig. 8.12). Algorithms are also available to allow the converse, i.e., a preference for atrial pacing\(^\text{12}\) (Fig. 8.13).

Another feature that may impact the pacing rate is one that responds to a sudden bradycardia. Most manufacturers have some type of algorithm whereby if a significant decrease in heart rate occurs, the pacemaker intervenes with pacing at an increased rate in both chambers for a specific, programmed duration (Fig. 8.14). At the conclusion of the programmed duration of more rapid pacing, the pacing rate gradually returns to the programmed lower rate. This feature varies between manufacturers and sometimes has programmable options in a specific pacemaker. In one algorithm available the pacemaker monitors a drop in heart rate that must satisfy a programmable degree of rate decrease, the number of beats the rate must fall, and the duration of time over which the drop in rate occurs. The lower rate limit needs to be programmed slow enough to measure the magnitude of rate drop required for intervention at the faster paced rate. Setting the parameters in a very liberal manner may result in frequent triggering of the algorithm and annoying and unnecessary symptoms (see Chapter 7, “Pacemaker and Cardiac Resynchronization Timing Cycles and Electrocardiography,” and Figs 8.45 and 8.15).\(^\text{13–15}\)

**Fig. 8.11** Programmer screen that depicts the outcome of intended ventricular pacing avoidance, i.e., promotion of intrinsic atrioventricular conduction. This example summarizes a 5-day period during which a total of 340 sensed ventricular events occurred, with further detail regarding the heart rates at the time these relatively rare sensed events were noted.
In another algorithm therapy is triggered when pacing occurs at the programmed lower rate for the programmable consecutive number of “detection beats”\(^{16}\) (Fig. 8.16).

**Programming output (pulse width and voltage amplitude)**

Programming the output of the pacemaker is one of the most important programmable features. There are two goals to programming output: (i) ensure reliable capture by an adequate safety margin, preferably measured in voltage (less preferably in pulse duration); (ii) maximize pulse generator longevity to the extent this is consistent with the first goal. This requires getting the best safety margin at a pulse duration that provides minimal energy drain from the battery.
Pacing pulses approximate rectangular pulses. Based on these considerations, the optimal pulse duration for rectangular pulses is near the chronaxie on the hyperbolic strength–duration.

Programming output can be used to extend pulse generator life by reducing output or to solve the clinical problem of increased or increasing stimulation thresholds. Contemporary pulse generators provide significant flexibility in pulse width and voltage amplitude. Voltage amplitude is programmable from 0.5 to 8.1 V and pulse width from 0.05 to 1.9 ms, with variations between manufacturers and to some degree between models of a given manufacturer.

With good implantation technique and low-threshold lead designs, e.g., steroid-eluting leads, it is common to program the voltage output to values of ≤ 2.5 V.
nominal voltage for many pacemakers). (The voltage amplitude program is also affected by the programmed pulse width, see below.) By programming the output at an efficient but safe level, the projected battery life can be increased significantly. A decrease in output can also be used to eliminate extracardiac (diaphragmatic or pectoral muscle) stimulation.

Conversely, in some patients, thresholds may increase after implantation. Although a transient and mild increase in thresholds is not uncommon in the first 4–6 weeks after implantation, higher outputs can be programmed until thresholds return to a stable level. Although this threshold evolution is largely avoided with steroid-eluting leads, it is reasonable to program higher outputs for the first 2–3 months postimplantation. We generally leave output parameters at nominal values until we see the patient back for threshold measurement and then subsequently reprogram to lower outputs. If an auto-capture algorithm is being used that will check stimulation thresholds on a relatively frequent basis and respond automatically to a loss of capture, it may not be necessary to leave the initially programmed output parameters at the higher values.

High thresholds may be transient or permanent (Fig. 8.17) and output programmability is useful in both situations. In patients with a transient elevation, higher outputs can be used until thresholds return to a stable chronic level. In patients with chronically high thresholds, the pulse generator can be programmed to higher output to permit reliable pacing (albeit with reduced pulse generator longevity). Programming to higher outputs may be done to temporize until a solution is sought to achieve lower thresholds, or it may be the permanent solution, despite the additional drain on the pacemaker battery, if there is some reason not to proceed with lead revision.

The output function to be programmed for the most effective control of a rising threshold depends on the actual threshold. Both pulse duration and output voltage (amplitude) are programmable. Programming the pulse duration at or > 1.0 ms approaching rheobase—
the lowest voltage threshold at an infinitely long pulse duration—does not provide much additional pacing margin of safety, and results in high current and energy drain. If pulse duration programmability defines a threshold lasting > 1.0 ms, increasing the output voltage is a better option.

Experts disagree about the optimal method to program the safety margin once the stimulation threshold has been established. Manual options that have been advocated include:

- Double the voltage amplitude.
- Triple the pulse width.
- Determine the capture threshold in microjoules required at threshold and program the voltage amplitude and pulse width to achieve three times the threshold in microjoules. (This technique is rarely used with contemporary devices.)

Again, if the threshold is high, the rheobase may be above a specific voltage setting no matter how long the programmed pulse duration. If pulse duration threshold is high, output voltage is more useful. Conversely, if pulse duration threshold is very low, i.e., in the range of 0.05–0.1 ms at a given output voltage, reducing the voltage and modestly prolonging the pulse duration could be considered. It is possible to program output on the basis of “microjoules” delivered at capture threshold. Output programmability should not, at any time, be a substitute for proper lead placement.

How much safety margin is enough, especially for contemporary leads? There is no universally agreed upon answer to this question. Although this depends in part on how often you measure the threshold and options that are operational for intervening in the event of a change in threshold, the two extremes are beat-to-beat with automatic adjustment and once a year with operator adjustment.

As general guidelines, a larger safety margin is often considered in patients who are pacemaker dependent, in patients placed on medications that may alter pacing thresholds and patients on dialysis or other metabolic abnormalities where there may be wide swings in electrolytes and/or metabolic status. Maintaining less of a safety margin may be possible in patients with steroid-eluting leads and in patients with a cardiac resynchronization therapy (CRT) system with reliable capture on the left ventricular lead.

Determination of the stimulation threshold should be a part of routine pacemaker follow-up. Determining stimulation threshold is now fairly easy with auto-threshold measurements that are optional in most pacemakers, implantable cardioverter-defibrillators (ICDs) and CRT devices. This is often accomplished by programming the output values—voltage amplitude and pulse width—at which the threshold determination is to begin and then observing the electrocardiogram and electrogram during decrement of the output variables until capture is lost. The programmer for the specific device should provide clear directions on how to respond when capture is lost, for example, move the programming head or release pressure from the programming screen (Fig. 8.19).

Despite the relative ease with which thresholds can be done, it must be remembered that the risk of loss of capture includes not just asystole but bradycardia-dependent ventricular fibrillation (VF). Thus, in pacemaker-dependent patients, equipment for external defibrillation should be available whenever thresholds are measured. Ideally, all thresholds should be measured on a temporary programming screen rather than a permanent screen, unless there is a specific reason to use a permanent screen. This is especially important when the device is near battery depletion.

Thresholds can still be determined by manually reprogramming the output variables until the threshold is determined. In the non-pacemaker-dependent patient manual thresholds can be performed by first programming to the VVI mode and decreasing the rate until the patient’s intrinsic rhythm is observed. The pacing is then increased to a rate exceeding the intrinsic ventricular rate. Thresholds can be performed by using a fixed pulse width and decrementing the voltage amplitude or using a fixed voltage amplitude and decrementing the pulse width. We generally program the pulse width to 0.5 ms and decrement the voltage. The point at which capture is lost is noted, and the threshold should be the last voltage amplitude where capture was maintained. Another rapid method for checking thresholds in the nondependent patient can be performed with simultaneous changes in rate and output variables. For example, with the pacemaker programmed to VVI at 40 ppm, if the intrinsic rate is 70, the next programming step could include VVI at 80 ppm, and very low output variables, e.g., voltage amplitude of 1.0 V and pulse width of 0.12 ms. If pacing is reestablished, the stimulation threshold is ≤ 1.0 V, 0.12 ms. If capture is not reestablished at 1.0 V and 0.12 ms, one of these two variables can be increased until capture occurs (Fig. 8.19). Whether one increases pulse width
or voltage amplitude for determination of stimulation threshold is in large part personal bias.

Other methods to provide some sense of stimulation threshold, or perhaps more appropriately stated as some sense of pacing margin of safety, have also been used for many years. One manufacturer incorporates a Threshold Margin Test with magnet application. Another approach is a proprietary feature in a rate of 100 ppm for three beats, followed by asynchronous pacing at the programmed rate. The first and second pacing artifacts at a rate of 100 ppm are of normal, i.e., programmed, pulse duration. The third pacing artifact at a rate of 100 ppm is at 75% of the programmed pulse duration.Loss of capture on the third beat indicates a narrow pacing margin of safety (Fig. 8.20). Another approach is a proprietary feature

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**Fig. 8.18** Printouts from two pacemaker programmers displaying autothreshold testing. (A) Atrial voltage amplitude threshold test, i.e., pulse width is held constant and voltage amplitude is decreased. Atrial capture is lost at 0.5 V. (B) Ventricular voltage amplitude threshold test. Ventricular capture is lost at 1.0 V.
Fig. 8.19 Determination of ventricular stimulation threshold during routine pacemaker follow-up. (A) The pacemaker is programmed VVI, rate of 75 ppm, voltage amplitude of 2.5 V, and pulse duration (PW) of 0.05 ms (upper tracing) and 0.1 ms (lower tracing). Capture is consistently lost at 0.05 ms but restored at 0.1 ms. The stimulation threshold is 2.5 V, 0.1 ms. (B) In the upper tracing, the pacemaker is programmed VVI at 70 ppm, 2.5 V, and 0.1 ms pulse width. There is intermittent failure to capture. In the lower tracing, the pulse width has been increased to 0.3 ms and there is consistent failure to capture. However, one should not be confused by the variation in QRS morphology. The fourth and sixth beats represent fusion beats, and the fifth beat is a pseudofusion beat. (A, Modified from Hayes DL. Programmability. In: Furman S, Hayes DL, Holmes DR Jr, eds. A practice of cardiac pacing, 3rd edn. Mount Kisco, NY: Futura Publishing Co., 1993:635–663. By permission of Mayo Foundation.)

Fig. 8.20 Electrocardiographic tracings from a patient with a VVI Medtronic pacemaker programmed to a rate of 70 ppm. The pacemaker is capable of a Threshold Margin Test (TMT) during magnet application. With magnet application the rate goes to 100 ppm. The first pacing artifact fuses with the QRS and it is difficult to tell if there is a QRS complex present. However, there is a definite "T" wave that follows so by definition, “if there is repolarization there had to be depolarization.” The second artifact results in definite capture. At the 3rd pacemaker artifact there is failure to capture and a native QRS follows approximately 240 ms later. It is on the 3rd output after magnet application when the pulse width is decreased to 80% of the programmed value.
called “Vario.” This is a programmable option that remains available on some pacemakers still in service. In the Vario mode, magnet application results in 16 asynchronous beats at a specific magnet rate followed by 16 asynchronous beats during which the voltage output is reduced progressively until zero output or very low output is reached (Fig. 8.21). Removal of the magnet returns full output at the next stimulus. The Vario mode can be activated for the test procedure only or programmed “on” permanently.

Features that allow the pacemaker to adjust output to threshold alterations are able to conserve battery consumption and prolong device longevity as well as protect patients from increasing stimulation thresholds. Automatic output adjustment and management is available in many pacemakers and, as with any algorithm, operational features vary between manufacturers. The proprietary AutoCapture system confirms capture on a beat-by-beat basis by monitoring the “evoked response” (ER) associated with the ventricular pacing output. (More specifically, the “evoked response” is verification on the intracardiac electrogram that indicates that capture has occurred.) To detect the ER signal, a bipolar low polarization pacing lead must be used. Each paced ventricular beat is assessed for capture by the system through monitoring of the ER signal. When no ER signal is detected, the AutoCapture system delivers a 4.5 backup safety pulse within 80–100 ms. This backup safety pulse functions as the “safety margin.” With two consecutive loss-of-capture events followed by backup safety pulses, the AutoCapture system automatically increases the output of the primary pacing pulse until capture is regained from the primary pacing pulse (Fig. 8.22).
the beginning of this increment, the first paced event is increased by 0.25 V. If capture is not confirmed with this paced event, the pulse amplitude continues to increase in 0.125-V steps* until capture is verified from the primary pacing pulse for two consecutive paced events. Once verification occurs, the pacemaker automatically initiates a threshold search. If capture for two consecutive events is not confirmed by the time the pacemaker output reaches 3.875 V, the pacemaker automatically reprograms to 4.5 V and 0.5 ms pulse width, or “high output mode” pacing.

A threshold search is initiated whenever the programmer head is placed over the pacemaker and then removed and, as noted above, when two consecutive loss-of-capture events result in an increase in the ventricular amplitude. When a threshold search begins, the pacemaker automatically decreases the pulse amplitude in 0.25-V steps until two consecutive loss-of-capture events occur, at which point the primary pacing pulse amplitude is increased by 0.125-V steps until two consecutive capture events are confirmed.

* In some devices with this feature the voltage increment may occur at 0.3-V steps.

Several manufacturers have autocapture on the atrial channel as well.

Once again, capture management or automatic threshold adjustment algorithms vary between manufacturers. They may be responsible for some unusual electrocardiographic findings. It is important to understand the nuances and programming options for each such algorithm used (Fig. 8.23).

Because output adjustment is critical for both extremes, i.e., for maintaining an adequate safety margin and for prolonging battery longevity, automatic regulation of output has become a fairly standard feature and is used with increasing frequency.

**Sensitivity programmability**

All pulse generators sense and filter the intracardiac electrogram delivered through the electrodes. For atrial and ventricular sensing (one or both), the R and P waves must be of significant amplitude (millivolts) and slew rate (dV/dt) for proper sensing to occur. The sensitivity of the pulse generator is the R (or P) wave of the lowest amplitude that the pacemaker recognizes as a ventricular (or atrial) depolarization (Fig. 8.22). The pacemaker definition of whether it is an R or a P wave

![Fig. 8.23 Programmer screen showing options for a capture management algorithm.](image-url)
depends on the channel through which it is sensed. Events sensed through the atrial channel are defined as P waves, those through the ventricular channel as R waves. Nominal ventricular sensitivity is usually in the range of 1.2–2.5 mV, and nominal atrial sensitivity is usually in the range of 0.5–1.2 mV. Although the amplitude of the intracardiac R or P waves may be adequate at the time of implantation, it may change for a variety of reasons, including metabolic and drug effects, myocardial damage and lead dislodgment. Each focus of intrinsic activity, be it atrial or ventricular, is not equally sensed, and some foci (conducted or ectopic) may not reach an adequate level of amplitude or slew rate to be sensed. Some extrasystoles may be sensed, whereas conducted beats may not be sensed, and vice versa.

Sensitivity programming can be accomplished by an increase in the sensitivity of the amplifier, i.e., by decreasing the amplitude of the signal required to trigger the sensing circuit but maintaining the same frequency spectrum. (The terminology is confusing because the amplifier is made more sensitive as the number decreases, i.e., 1.25 mV is more sensitive than 2.5 mV.)

The sensing threshold should be determined during routine pacemaker follow-up and can be accomplished in multiple ways (Fig. 8.24). Manual sensing thresholds can be obtained by programming the pacemaker to progressively less sensitive values until there is failure to sense (Fig. 8.25). (Sensing thresholds could also be determined by altering sensing values during programming to a triggered pacing mode, i.e., AAT or VVT.)

Automatic sensitivity adjustment is available in many pacemakers. Autosensing adjusts sensitivity on the basis of amplitude of the intrinsic waveform.26,27 (This term or similar terms for automatic sensitivity adjustment may have different implications for ICDs. However, the method of dynamically altering sensitivity after paced or sensed events, available in ICDs for generations of devices, is now being used in pacemakers.) The purpose of automatic sensitivity is to prevent or minimize episodes of both oversensing and undersensing. Although not as critical clinically as automatic output management, automatic sensing has merit and is used with increasing frequency. As noted early in this chapter, determination of sensing threshold via the programmer is often available (Fig 8.26A,B).

The differential diagnosis for sensing abnormalities and approach to correcting such problems are discussed in Chapter 10, “Troubleshooting.”

![Fig. 8.24 R-wave amplitude test. It is displaying the Ventricular Sense Amplitude electrogram. The top tracing is the surface ECG. The test results are shown in the upper right and the various values and settings for the actual ECG and EGM recording are in the upper left. (From the Collection of and with the permission of Paul A. Levine, MD, FHRS.)](image-url)
Polarity programmability

Polarity programmability is available on most pacemakers with bipolar configuration. It allows programming from unipolar to bipolar functions. (In some dual-chamber pacemakers, the polarities of the atrial and ventricular channels are independently programmable; in others, they are not.) This feature is helpful in patients who have myopotential or electromagnetic inhibition in the unipolar mode, but not in the bipolar mode. Unipolar and bipolar electrograms have distinctly different characteristics, and programming from one polarity to the other may eliminate the sensing of an unwanted electrogram or interfering signal. It is possible but improbable that sensing in one polarity configuration will be superior to that in the other (Fig 8.27).

Polarity programmability may be helpful in a patient with a lead fracture or inner insulation failure by converting from bipolar to unipolar configuration. If the lead is fractured, effective pacing may be restored through the remaining intact pole of the pacing lead (Fig. 8.28). This alternative should usually be considered a temporary measure, because whatever force resulted in one fractured conductor may eventually lead to fracture of the second. If an inner insulation failure is present, programming the pulse generator to unipolar mode reduces the applied current at the programmed voltage, thus preventing unnecessary battery depletion. In an increasing number of pulse generators, a change from bipolar to unipolar pacing configuration may be triggered automatically when a sudden change in impedance is detected during bipolar pacing if this programmable option is activated (Fig. 8.29).

Refractory and blanking periods

Refractory and blanking periods are described in detail in Chapter 7. In brief, a refractory period can be defined as an interval during which a given sensing circuit does not respond to sensed events. This is in contrast to a blanking period, which is an interval during which a given sensing circuit is disabled.

Single-chamber pacemakers

Refractory period programming may be necessary in a variety of clinical circumstances. In the VVI mode, the ventricular refractory period (VRP) is the interval during which the ventricular sensing circuit does not respond to sensed events. The first portion of the VRP is usually a ventricular blanking period during which ventricular sensing is disabled after paced, sensed, and refractory sensed ventricular events. Depending on the pacemaker, the ventricular blanking period may be fixed, programmable, or dynamic, i.e., vary on the basis of strength and duration of the ventricular event. Refractory period programming in single-chamber pacing is not commonly required, but can be advantageous in some situations. Lengthening of the VRP may help prevent sensing of afterpotential depolarizations, T waves, or premature ventricular contractions. If the refractory period is so long that some ventricular electrograms are not sensed, the subsequent pacemaker stimulus could potentially fall on the T wave of the unsensed beat. In this circumstance, shortening of the VRP would be appropriate.

If a single-chamber pacemaker is used for atrial pacing, a longer refractory period, the atrial refractory period (ARP), is desirable to avoid inhibition of atrial
Fig. 8.26 (A) Trial sense test from a patient with a dual-chamber pulse generator. It is being run with the surface ECG, the event markers and the bipolar (Atip-Aring) electrogram being simultaneously displayed. Note the lack of P markers at the temporarily programmed sensitivity of 3.0 mV. Hence, the sensing threshold is the previous more sensitive setting at which there was consistent sensing for the number of cycles programmed for the test (4 to 10) and this value is shown in the data at the upper right. (B) Semiautomatic "Ventricular Sensing test." Under the central box with the new value, i.e., 9 mV, there is a display of the results the last time that this test was performed, at which time it was > 12.5 mV. (From the Collection of and with the permission of Paul A. Levine, MD, FHRS.)
Fig. 8.27 Electrocardiographic tracing from a patient with a dual-chamber pacemaker programmed to VVI mode at 30 ppm. (Top tracing = surface ECG; 2nd tracing = atrial electrogram; 3rd tracing = ventricular electrogram; marker channel at the bottom of the tracing.) Note that 3rd QRS has no marker channel notation and is followed by a ventricular paced (VP) event. Measuring backward from the VP event by 2000 ms (30 ppm) it coincides with the prior ventricular sensed (VS) event. The measured R wave equaled 2 mV. When programmed to unipolar sensing configuration, the R wave measured 6 mV.

Fig. 8.28 Electrocardiographic tracings from a patient with a VVI pacemaker. (A) In the bipolar configuration, there is intermittent failure to capture. Capture is demonstrated only with the first two pacing stimuli. (B) Programmed to the unipolar configuration at the same pulse duration and voltage, the pacemaker demonstrates consistent capture. (From Hayes DL. Programmability. In: Furman S, Hayes DL, Holmes DR Jr, eds. A practice of cardiac pacing, 3rd edn. Mount Kisco, NY: Futura Publishing Co., 1993:635–63. By permission of Mayo Foundation.)
pacing as a result of sensing of the ventricular electro-
gram (far-field R waves) (Fig. 8.30).

**Dual-chamber pacemakers**

Refractory periods in dual-chamber, dual-sensing units are much more complex than in single-chamber units because the events and timing cycles in one channel affect those in the other. With programming to a dual-chamber sensing mode, refractory periods exist for each sensing channel. The refractory period for the ventricular channel behaves the same as that for single-chamber sensing. The operation of the refractory period on the atrial channel is quite different. After an atrial stimulus or a sensed atrial event, the initial portion of the atrio-
ventricular interval (AVI) is the atrial blanking period. During this interval, atrial sensing cannot take place. Depending on the pacemaker, the atrial blanking period is fixed, programmable, or dynamic, i.e., varying in relation to the strength and duration of the atrial event.

A ventricular blanking period is also initiated with a sensed or paced atrial event. The intent of this interval is to avoid sensing the electronic event of one channel in the opposite channel (“crosstalk”). The blanking period is usually programmable. It may be desirable to prolong the blanking period to prevent crosstalk (Fig. 8.31). It may be necessary to shorten the blanking period if ventricular extrasystoles are sensed during this period, because the result could be pacing during the early portion of ventricular repolarization. Shortening the blanking period should diminish the likelihood of the QRS occurring within the blanking period.

The atrial sensing amplifier remains refractory for the remainder of the AVI plus the programmed atrial refractory interval after the ventricular event, the PVARP.

The first portion of the PVARP disable atrial sens-
ing after paced, sensed and refractory sensed ven-
tricular events, i.e., the postventricular atrial blank-
ing period (PVAB). Once again, this interval may be fixed or programmable, depending on the pacemaker (Fig. 8.32).

Sensed events in the non-blanked portion of the TARP are used for mode switching. Excessive atrial blanking may prevent mode switching, particularly at rapid ven-
Minimal PVAB periods are preferred in patients at risk for 2:1 conduction of atrial flutter. Programmable flexibility of the PVARP is especially important because of its role in preventing endless-loop tachycardia (ELT). Because ELT can occur only when the PVARP is shorter than the retrograde (VA) conduction time, this is an especially important interval. Not all patients, however, have in-

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**Fig. 8.32** Programmer screen displaying device diagnosis of far-field sensing with annotated electrogram. Based on this diagnosis the pulse generator also proposes that the postventricular atrial blanking period (PVAB) be lengthened from 150 to 165 ms and the atrial sensitivity reprogrammed from 0.6 to 0.8 mV.

**Fig. 8.33** Two-channel electrocardiographic tracing that begins with atrial pacing followed by a long AR interval and intrinsic ventricular conduction. The 4th ventricular event (*) is premature. This is followed by the initiation of a pacemaker-mediated or endless-loop tachycardia at a rate of approximately 115 ppm.
Fig. 8.34 Programmer screen during programming a PMT termination algorithm. In this particular algorithm if there are eight consecutive ventricular pace to atrial sense (VP–AS) sequences that have a cycle length that is less than the programmed VA criterion, in this example 360 ms, the “suspicion criterion” for PMT are met. The device then modifies either the AV interval (+ or −50 ms) or adds 50 ms to the upper tracking rate for one cardiac cycle. If the VP–AS remains unchanged, the device confirms the rhythm as a PMT. Once this occurs the algorithm extends the TARP for one cycle to a total length of the V–V cycle length +50 ms.

Fig. 8.35 Tracing display: top, marker channel; middle, atrial electrogram; bottom, surface electrocardiogram. In the marker channel the first “P” indicates a sensed native atrial depolarization. All of the other “P” waves are displayed in a black square. This display is an indication for this manufacturer that the P wave is refractory, i.e., it is recognized but will not alter the timing cycle. Each of the refractory “P” waves is occurring in the PVARP. This is an example of functional atrial undersensing.
tact VA conduction, and some have short retrograde conduction times. Programmable options other than a prolonged PVARP can avoid ELT. Algorithms to detect and interrupt ELT have been incorporated into most dual-chamber pacemakers, accomplishing this by automatically altering the PVARP or the AVI, or both (Fig. 8.34) (see Chapter 7).

The pacemaker may have a programmable option of “PVARP extension.” If this feature is enabled, the PVARP is lengthened a defined duration if a premature ventricular contraction (PVC) is sensed. In actuality, the pacemaker cannot differentiate a PVC from any other ventricular beat. The most common mechanism for designating a ventricular depolarization as a PVC is sensing by the pacemaker of two ventricular events without an intervening atrial event. Should this happen, the PVARP is extended in an effort to avoid sensing of the potential retrograde atrial activation that occurs as a result of the PVC. PVARP extension may result in confusing electrocardiographic presentations. If the extended PVARP encompasses the subsequent atrial or ventricular event, the appearance of undersensing results. This is considered “functional undersensing,” because it is a function of the extended PVARP (Fig. 8.35).

**Mode switching**

One of the main disadvantages of DDD pacing is its tendency to track atrial tachyarrhythmias, which are common in patients with pacemakers. Pacing in patients with paroxysmal atrial arrhythmias can be done with “conventional” dual-chamber pacemakers programmed in various modes or responses to cope with the atrial arrhythmias. In DDD pacemakers, any programming options other than the DDD pacing mode have in common some compromise of AV synchrony or maximal achievable paced rate during exercise. Most contemporary pacemakers have algorithms that are designed to detect and respond efficiently to atrial tachyarrhythmias.

Mode switching refers to the ability of the pacemaker to change automatically from one mode to another in response to an inappropriately rapid atrial rhythm (Fig. 8.36). With the early forms of mode switching, when the pacemaker was functioning in the DDDR mode, the algorithm automatically reprogrammed the pacemaker to the VVIR mode if specific criteria were met for what was considered to be a pathological atrial rhythm.

![Fig. 8.36](image_url) (A) Mode-switch episode: top, marker channel; middle, atrial electrogram; bottom, ventricular electrogram. With the onset of a rapid atrial rhythm, many “P” waves fall in refractory, designated by the “shaded” P. The P waves that fall in the refractory period are counted for the purpose of determining whether a programmed atrial tachycardia detection rate has been achieved. Mode switch criteria are fulfilled where the notation “trigger” occurs, and AMS notes “automatic mode switching.” (Continued.)
rhythm. However, any pacing mode that eliminates tracking of the pathological rhythm, i.e., DDI, DDIR, DVI or DVIR, also eliminates the ability to track normal sinus rhythm, which is usually the predominant rhythm. Mode switching avoids this limitation. The non-atrial-sensing mode (VVI, VVIR, DDI, DDIR, etc.) to which the device is automatically programmed is specific to an individual pacemaker (Fig. 8.37). The sensed atrial rate at which mode switching occurs is usually programmable. Mode switching is particularly useful for patients with paroxysmal supraventricular rhythm disturbances. However, it is reasonable to program mode-switch “on” for most patients unless it compromises or “locks out” some other desired programmable feature for the specific patient (Fig. 8.38).

Many pacemakers provide extensive diagnostic data regarding events that meet mode-switch criteria. However, depending on the mode-switching algorithm in use, inappropriate mode switching may occur. With newer, more specific algorithms, inappropriate mode
In Fig. 8.37, a three-channel electrocardiographic tracing is shown with an underlying rhythm of atrial fibrillation and all ventricular activity is paced. An atrial pacing artifact precedes the 4th ventricular event, an example of mode switching to the DDI mode.

In Fig. 8.38, a programmer screen is depicted from which the mode-switch algorithm is programmed in this pacemaker. Multiple programmable criteria for mode switching exist. Turning it "on" requires setting criteria for atrial tachycardia detection rate or "trigger rate," number of cycles evaluated, number of cycles to satisfy criteria and mode switch, number of cycles to return to the originally programmed mode, duration of time to lower the rate detected at the time mode switch occurs, and lower rate limit to be used during mode switching. Additionally, an atrial flutter response can be turned "on."
switching is less frequent, but certainly still occurs. In some pacemakers, the appropriateness of mode switching can be verified by stored electrograms (Figs 8.39 and 8.40). Although there are multiple issues that may impact the appropriateness of mode switching, there are two comments that bear mention. The first is where to set the mode-switching rate. No single rate is correct for all patients, and the selection is simplified if the rate of the recurrent atrial tachyarrhythmia is known. It’s tempting always to set the detection rate significantly below the known tachyarrhythmia rate in an effort to detect all tachyarrhythmias. This, however, may result in inappropriate mode switching, e.g., secondary to sinus tachycardia or atrial extrasystoles. Although no single value can be recommended for programming the detection rate, the caregiver is advised to review the patient’s tachyarrhythmias, the nominally programmed value and adjust appropriately. Also remember that the measured atrial rate is affected by blanking periods, particularly at high ventricular rates.

**Programming rate-adaptive parameters**
Parameters that determine rate adaptation in a sensor-driven pacemaker vary considerably depending on the sensor incorporated (Figs 8.41 and 8.42). Programming rate-adaptive parameters is discussed in Chapter 9, “Rate-adaptive Pacing.”

**Diagnostics—set-up and assessment**
A variety of diagnostics are available on contemporary pacemakers, some of which have already been mentioned. Pacemakers are capable of detecting high atrial rate episodes, mode-switching events, high ventricular rate episodes, episodes which meet sudden bradycardia response criteria, etc.

During programming there will be an option to view “diagnostics.” (This may be given a different name by some manufacturers.) As part of routine programming this field should be selected and the caregiver

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**Fig. 8.39** Programmer screen displaying a mode-switch event with atrial and ventricular electrograms and marker channel. The entry is “time-stamped” and the peak atrial rate detected was 160 bpm.
Fig. 8.40 Time-stamped stored event of mode switching. In the three-channel recording, the top channel is the marker channel; middle, atrial electrogram with an irregularly irregular rhythm consistent with atrial fibrillation; bottom, ventricular electrogram.

Fig. 8.41 Set-up screen for a rate-adaptive sensor, in this case an accelerometer. Programming options include the lower pacing rate and upper sensor-driven rate as well as the ADL, or activity of daily living, rate. In this device the ADL rate indicates the heart rate that would be desirable for the given patient with moderate activities. Rate profile optimization allows the pulse generator to adapt the ADL and exertional rate response levels one time each day. It does this by comparing the “sensor rate profiles” vs. a target rate profile.
should page through each of diagnostics categories to see if any events have been collected.

If programming the device for the first time, the diagnostic preferences need to be selected (Fig. 8.43). Because there is a limitation to the number of events that can be stored in a pulse generator, it is usually not possible to select all of the available diagnostic categories. Categories selected should be based on the patient’s history. For many patients, selection of “high rate atrial episodes” is appropriate because it allows the caregiver to assess the events that trigger mode switching and the electrograms collected help to determine if the mode switching was appropriate (Figs 8.44–8.46).

### Unexpected programming

There may be times when interrogation reveals something other than the expected programmed parameters. In early programmable devices before programming was accomplished exclusively by radiofrequency, faulty transmission of signals between programmer and pacemaker would occasionally result in abnormal programming. With contemporary devices, unexpected programming probably occurs either because someone has reprogrammed the device and failed to document the changes or because the pacemaker has been exposed to electromagnetic interference.

Sources and management of electromagnetic interference are discussed in Chapter 12, “Electromagnetic Interference and Implantable Devices.”

### Programming during routine follow-up

The importance of a systematic approach to programming during routine follow-up and troubleshooting cannot be overstated. Continued improvements in automaticity will undoubtedly further minimize the time required for programming. At present, however, a defined approach to programming should be adopted to be certain that the pacemaker is thoroughly evaluated and to optimize pacemaker function. The following programming sequence for a rate-adaptive

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**Fig. 8.42** Programming a dual-sensor pacemaker. The accelerometer sensor is “on” with a rate response factor “8” on a 1–10 scale. Minute ventilation is currently “off.” Behind the minute ventilation “drop-down” menu and partially obscured is an option for Expert Ease/Lifestyle. **This option, if activated, assists the clinician by optimizing the behavior of the sensor based on the age of the patient. This feature will suggest programmed values for the lower rate limit, maximum sensor rate, and high rate response factor.**
dual-chamber pacemaker (DDDR) is presented as a potential approach. It is not critical that programming be performed in this specific sequence. (In fact, depending on the manufacturer of the pacemaker being programmed, the way the programming screens are designed may lend itself to another sequence that is more efficient.)

1. Interrogate the pacemaker and print out stored data
2. Assess magnet response
3. Determine underlying rhythm
4. Collect measured data
   - Lead impedance(s)
   - Intrinsic P and/or R wave amplitude
   - Battery voltage—predicted longevity.
5. Determine the atrial and/or ventricular stimulation thresholds.
6. Determine sensing thresholds.
   - If AV conduction intact, consider algorithm to promote intrinsic ventricular conduction
8. Determine the appropriate upper rate limit by considering:
   - Physical activity
   - Subjective response to well-being during activity at current programmed values
   - Associated medical conditions.
9. Program AV delay variables, i.e., PAV/SAV, RAAD.
10. Program rate-adaptive parameters
    - Assess exercise, either formally or informally (see Chapter 9, “Rate-adaptive Pacing”).
11. Evaluate “other” parameters
    - In most patients, activate the pacemaker-mediated tachycardia algorithm
    - In most patients, turn on “mode-switching” algorithm
    - In most patients, program PVARP extension “on”.
12. Review “other” features available for the individual pacemaker, and program “on” desirable features (Fig. 8.47).
13. Review programmable telemetry options, and program as desired.

There are some pulse generators that will make programming suggestions based on details of the patient’s history that have been entered in combination with collected data (Fig. 8.48). Additional discussion on man-
Fig. 8.44  (A) A “summary” screen which notes a clinical event of “ventricular tachycardia.” (B) Stored electrogram of the captured event, in this example, ventricular tachycardia.
Fig. 8.45 (A) Stored electogram: top, marker channel; middle, atrial EGM; lower, ventricular – EGM. The atrial EGM suggests a tachycardia initially, but this is not corroborated by the marker channel. However, the subsequent rapid ventricular rate does correlate with the marker channel. (B) The tachy episode trend is displayed. It displays the onset, duration and termination of the tachycardia detected in (A).
Fig. 8.46 A diagnostic screen displaying the 24-h heart rate trend. The device also reports how much time was spent in each pacing mode and the atrial arrhythmia burden.

Fig. 8.47 Programmer screen from which “additional features” can be programmed. As noted in the programming sequence, the clinician should, at some point during the programming sequence, consider any parameters unique to the pulse generator that may benefit the patient.
Management of routine follow-up is present in Chapter 13, "Follow-up."

**Defibrillator programming and algorithms**

Like pacemakers, ICDs incorporate many programmable parameters that enable device function to be tailored to an individual's cardiac disease. Indeed, many of the programmable features discussed in the pacemaker section of this chapter—pacing modes, programmable pacing output, mode switch, pacing rate smoothing algorithms, and others—are currently available in defibrillators. However, in addition to providing sophisticated anti-bradycardia support, ICDs must be able to detect low-amplitude VF electrograms, to differentiate ventricular tachyarrhythmias from supraventricular tachycardias, and to deliver overdrive pacing and high-energy shocks to treat tachyarrhythmias. These demands have resulted in additional programmable features and algorithms that must be understood for optimal use of the devices. This chapter focuses on the operation and programming of these features and is divided into the following sections:

- ICD sensing
- ICD detection and detection enhancements
- Low-energy ventricular therapies (antitachycardia pacing)
- High-energy ventricular therapies (cardioversion and defibrillation)
- Atrial defibrillators
- Optimizing programming
- Programming cardiac resynchronization devices.

**Implantable cardioverter-defibrillator sensing**

*Sensing* is the process by which an ICD determines the timing of each atrial or ventricular electrical event from the electrogram signals. *Detection* occurs after analysis of a sequence of sensed events by the defibrillator to classify the rhythm and determine whether therapy should be delivered. *Oversensing* on the ventricular
channel occurs when non-QRS potentials are greater than a reference threshold voltage and are considered sensed events; analogous oversensing occurs on the atrial channel. Oversensing may arise from intracardiac events (P waves or T waves) or extracardiac events (diaphragmatic signals, electromagnetic interference) (Fig. 8.49) that lead the defibrillator to determine that cardiac events are present when in fact they are not. In contrast, undersensing occurs when the electrical signals of interest, whether QRS complexes or the electrograms of fibrillation, do not reach the threshold voltage to be sensed as an event (Fig. 8.50). Because the number

![Fig. 8.49 Oversensing. A recording from an implantable cardioverter-defibrillator is shown with surface electrocardiogram (top), electrogram (middle), and marker (bottom). Below are shown the markers again, annotated with cycle length and ventricular fibrillation (VF) and ventricular tachycardia (VT) counter values. During the ventricular couplet, the T wave exceeds the voltage threshold for sensing and is oversensed.](image)

![Fig. 8.50 Undersensing. An implantable cardioverter-defibrillator recording is shown, as in the preceding figure. The QRS complexes marked with an asterisk have a right bundle branch block (RBBB) morphology due to intermittent bundle branch block and are not sensed. Note that the marked complexes appear smaller on the electrogram (bottom tracing) and are not sensed (no marker) and that a pacing pulse is inappropriately delivered shortly after the undersensed event (VP).](image)
of sensed events is either too high (oversensing) or too low (undersensing), the actual rhythm may be misinterpreted by the defibrillator’s algorithm, resulting in inappropriate device activity.

Determination of the intracardiac ventricular rate requires appropriate sensing of QRS complexes of relatively large amplitude and avoiding detection of the subsequent midsized T wave (which would result in double counting of a single event) while maintaining adequate sensitivity to detect fibrillatory electrograms of small amplitude. Because the amplitude of VF may be small, ICDs must amplify electrograms 10 times more than bradycardia pacemakers. The need to sense signals of markedly different amplitude has been addressed by a dynamic gain or sensitivity threshold. In many devices, this effectively increases the sensitivity after each sensed or paced event until the next QRS occurs, at which point sensitivity is diminished (Fig. 8.51). Consequently, oversensing of noncardiac signals is most likely to occur during slow heart rates, late in diastole, when sensitivity is greatest. This is particularly true after paced events, after which the attack rate (rate of increase in sensitivity or gain) is often greatest.

Generally, if the measured R wave during normal rhythm at implantation is at least 5 mV, spontaneous VF is adequately sensed with nominal sensitivity settings (near 0.3 mV). In the absence of VF inductions during implant testing, an R wave > 7 mV has been used. During implant testing, it is useful to assess detection at the least sensitive setting (largest numerical value). This determines the safety margin for sensing should a reduction in programmed sensitivity (increase in numerical value) be required in the future (due to oversensing, for example). However, if inter-
VENING changes in medication or clinical status have occurred since implantation, induction of arrhythmia to reassess VF detection is performed before sensitivity is reprogrammed. During testing, assessment of sensing after a failed defibrillation shock is useful if VF sensing is borderline, as it tests the worst case scenario. This is particularly important for older integrated bipolar leads with short spacing between tip and distal coil electrodes.

The manner in which dynamic sensing is applied differs among manufacturers, and the actual maximum and minimum sensing floor for each setting varies, as shown in Fig. 8.52. In the Boston Scientific devices, “sensitivity” is programmed to one of three values: “nominal,” “most,” and “least.” A “fast” automatic gain control rapidly adjusts with each R wave, whereas a “slow” automatic gain control adjusts the overall dynamic range of the gain. Templates with unique attack rates for pacing, normal rate sensing, and tachycardia sensing are applied (Fig. 8.53).

In Medtronic devices, the concept is similar, except that slow automatic gain control does not occur. A sensitivity level is programmed by selection of the smallest signal that can be detected during the time in the cardiac cycle that sensitivity is maximum, nominally 0.3 mV in the ventricle (Fig. 8.54).

St Jude defibrillators offer the greatest level of programmable control over dynamic sensing function, in that the contour of the sensing envelope can be manipulated at multiple levels (Fig. 8.55).

![Fig. 8.52](image-url) Comparison of automatically adjusted sensitivity after sensed ventricular events for three ICD manufacturers. Left panel shows markedly different performance after large (10 mV) R wave. Right panel shows similar performance after small (3 mV) R wave. Nominal sensing threshold ~0.3 mV. After sensed ventricular events, Medtronic ICDs reset the sensing threshold to 8–10x the time programmed sensitivity, up to a maximum of 75% of the sensed R wave. The value of auto-adjusting sensitivity then decays exponentially from the end of the (sense) blanking period with a time constant of 450 ms until it reaches the programmed (maximum) sensitivity. At the nominal sensitivity of 0.3 mV, there is little difference between the sensitivity curves after large and small spontaneous R waves. If the R wave is big, the entire auto-adjusting sensitivity curve can be altered substantially by changing the programmed value of maximum sensitivity. At nominal settings, the St Jude threshold start begins at 62.5% of the measured R wave for values between 3 mV and 6 mV. If the R-wave amplitude is >6 mV or <3 mV, the threshold start is set to 62.5% of these values (3.75 mV and 1.875 mV, respectively). The sensing threshold remains constant for a decay delay period of 60 ms, and then decays linearly with a slope of 3 mV/s. Both the threshold start percent and decay delay are programmable over the range 50–75% and 0–220 ms, respectively (Fig. 8.55). Guidant ICDs set the starting threshold to 75% of the sensed R wave. Sensitivity (“fast” automatic gain control) then decays with a half-time of 200 ms (time constant of 289 ms) to a minimum value that depends on the dynamic range of the sensing amplifier. “Slow” automatic gain control adjusts the maximum value of this dynamic range to 150% of the value of the average R wave. The minimum value of dynamic range is 1/8 of the maximum value. This is equivalent to 3/16 (18.75%) of the amplitude of the average R wave. After a paced ventricular event, all ICDs also adjust sensitivity dynamically starting at the end of the (pace) blanking period, but the threshold starts at a more sensitive setting. (Reproduced with permission from Swerdlow, Friedman. PACE 2005; 28:1322–46, Blackwell Publishing.)
Implantable cardioverter-defibrillator detection

**Single-chamber defibrillators**

Initial detection of a ventricular arrhythmia is based primarily on two rhythm characteristics: ventricular rate and arrhythmia duration (typically 1–3 s). Both parameters are programmable to meet the needs of the individual patient. The rate criterion distinguishes between a tachyarrhythmia and a normal rhythm, and the duration requirement limits detection of nonsustained episodes. Thus, all defibrillators use ventricular rate zones (Fig. 8.56) as the first step in rhythm classification. Different types of therapies and detection enhancements can be applied to tachyarrhythmias of different rates by further division into “slow VT” (ventricular tachycardia), “fast VT,” and “VF” zones (Fig. 8.56). In programming detection and therapy zones, two general principles apply: (i) detection of unstable (fast) VT and VF must be highly sensitive and therapies highly effective. The cost of this sensitivity is inappropriate treatment of rapid supraventricular tachycardias (SVTs); (ii) algorithms for rhythm discrimination and more than one sequence of antitachycardia pacing are programmed for slower (generally more hemodynamically stable) tachycardias to improve detection specificity and therapy tolerability. This improved specificity may come at the cost of some delay in detection and in the application of effective therapy.

Arrhythmia detection begins with each ventricular event. After each sensed or paced event, the time interval to the next sensed ventricular event determines its classification (Fig. 8.57). A sequence of classified ventricular events is accumulated in VT and VF counters until criteria for arrhythmia detection are met. The type of counting used varies between detection zones and among manufacturers. Because of highly variable electrogram amplitude during VF, some signal dropout may occur despite dynamic sensing. Therefore, to enhance sensitivity, initial detection occurs when a certain percentage (usually 70–80%) of sensed events within a continuously rolling detection window fall within the VF zone (Fig. 8.58). In Boston Scientific defibrillators, X of Y counting is also used for detection in VT zones (Fig. 8.59). During ongoing detection with more than one programmed zone, the higher zone (i.e., VF) takes priority over the lower zone (i.e., VT) (Fig. 8.59). Once the X of Y counter is satisfied, a programmable duration for which the arrhythmia must persist is required before therapy delivery. For unstable VTs and VF, this duration is kept short...
The duration in slower VT zones is independently programmable, so that longer intervals (nominally 1 s) can be detected during the time in the cardiac cycle that sensitivity is maximum. This value can range from 0.15 to 1.2 mV and is nominally programmed to 0.3 mV. After a sensed R wave, the sensitivity decreases to 75% of R-wave amplitude (to a maximum of eight times the programmed value) and then decays with a time constant of 450 ms to the programmed maximum sensitivity, as shown. After a paced event, the sensitivity decreases to 1.8 mV (maximum of 4.5 times the programmed value); the decay constant is not changed on the basis of heart rate. Similar dynamic sensing is applied in the atrium in dual-chamber devices. Note the absence of blanking in the atrial channel after a ventricular sensed event. This diminishes atrial undersensing during tachyarrhythmias. However, far-field R waves may be sensed; this is handled by a far-field R-wave oversensing algorithm.

In Medtronic defibrillators, X of Y counting is used for VF detection (Fig. 8.58), but consecutive interval counting is used for VT detection. Consecutive interval counting requires that a programmable number of consecutive intervals shorter (faster) than the VT cycle length be present for VT detection to occur; a single long (slow) interval resets the VT counter to zero (Fig. 8.60). This method increases specificity by avoiding detection of atrial fibrillation (AF) without compromising VT sensitivity, at the expense of nondetection of irregular VTs with a mean cycle length close to the VT cut-off rate. AF with a mean cycle length shorter than the tachycardia detection interval will be appropriately rejected if periodic intervals longer than the cut-off rate reset the tachycardia counter to zero (Fig. 8.60). For patients with known VT, a detection zone 30–40 ms longer than the slowest VT cycle length is typically used, with the VT counter set nominally to 16. In patients with recurrent
Tiered Therapy: Variable number ATP attempts followed by shocks of increasing strength

Brady Sinus VT FVT VF
SVT Discrimination Zone

Shock only; no ATP or during charging

Bradycardia pacing upper rate limit
Slower Rate Longer cycle length

Bradycardia pacing lower rate limit

Faster Rate Shorter cycle length

Fastest SVT rejected

Too fast for ATP before charging (~240 ms)
Expect VF undersensing

~400 ms or 40 ms > slowest VT
Consider SVT specificity

Different ATP or fewer trials (~320 ms)

Fig. 8.56 ICD rate zones. See text for details. ATP, antitachycardia pacing; FVT, fast VT; SVT, supraventricular tachycardia. Some ICDs permit programming of an additional monitor-only zone.

Sinus rhythm

VT
VF

VF zone
VT zone

VF detection interval
Blanking interval

450 ms
850 ms

Markers

VF VT VS

Fig. 8.57 Classification of each ventricular complex on the basis of cycle length. After each sensed or paced event, the time interval to the next sensed ventricular event determines its classification. If another ventricular event is sensed after the blanking period, but within the programmed ventricular fibrillation (VF) detection interval (typically 300–320 ms), the event is classified as a VF complex, regardless of the actual cause of the ventricular depolarization. A delay greater than the programmed VF interval but shorter than the maximum VT cycle length results in a ventricular tachycardia (VT) event. If the time between ventricular depolarizations exceeds the programmed length of the VT detection cycle, the event is sensed without classification as a tachyarrhythmia ("VS," for ventricular sensed event). If no event is sensed and the pacing rate interval is reached, a pacing impulse is generated. This example includes only two tachycardia zones (VF and VT). In most defibrillators, up to three tachycardia detection zones may be programmed to allow progressively more aggressive delivery of therapy for faster VTs. (From Olson WH. Tachyarrhythmia sensing and detection. In: Singer I. ed. Implantable cardioverter-defibrillator. Armonk, NY: Futura Publishing Co., 1994:71–107. By permission of the publisher.)
runs of nonsustained rapid VT/VF in the VF zone, increasing the number of intervals to detect VF from 12/16 to 18/24 decreases ICD detections and shocks.40,41 Since in Medtronic devices different counting methods are used in the VT and VF zones, when a third tachycardia zone (fast VT (FVT) zone) is added, the physician must choose whether to use VT-type consecutive interval or VF-type X of Y interval counting. If the FVT zone is programmed via VF, events in the VF or FVT zone are added to the VF counter and detection follows VF rules. Once detection is met, if all the preceding eight events were in the FVT zone, FVT therapy is delivered; otherwise, VF therapy is delivered. Conversely, if the FVT zone is programmed via VT, events in the FVT zone are added to the VT counter and detection follows VT rules. Once detection is met, if any of the preceding eight events were in the FVT zone, FVT therapy is delivered (Fig. 8.61).

St Jude defibrillators can also use up to three distinct rate-detection zones, called “VT-1,” “VT-2” and “VF” with separate counters and independently programmable criteria for each of the zones. For detection to occur in any zone, a programmable number of intervals must be classified and counted in that zone. In contrast to other devices, the classification of a sensed event depends on both the current interval and the average of the current interval and the previous three intervals (Table 8.3). The sinus counter is reset to zero whenever any interval is classified in a tachycardia zone.

When the sinus rhythm counter reaches a programmable value, sinus rhythm is detected and all counters are reset to zero. To prevent detection of bigeminy with average rates in a VT zone, a bigeminy detection algorithm withholds therapy if bigeminy is present.

Detection zone boundaries
Unanticipated ICD behavior may occur when arrhythmias straddle the rate detection zone boundaries. In Boston Scientific, older Medtronic (prior to Marquis™) and older St Jude (prior to Atlas 2™) devices, programming a VT zone with no therapies for use as a monitoring zone could accelerate therapy. This occurs since sensed events in the VT zone count towards detection in the adjacent faster zone (Fig. 8.62). Newer devices provide an independent monitor-only zone that avoids this limitation. In St Jude ICDs, if VT is detected in the “monitor zone” VT discriminators are disabled for all zones for that episode.
Fig. 8.59 Use of X of Y counting in multiple zones (in a Boston Scientific implantable cardioverter-defibrillator).

(A) For each zone, the detection heart rate and tachycardia duration are programmed independently. Each zone has a detection window composed of the 10 most recent RR intervals. As each new interval is measured, it is defined as either fast—above the programmed rate threshold for the window—or slow. A window is satisfied when 8 of the 10 most recent RR intervals are fast and remains satisfied as long as 6 of 10 intervals in the moving window are fast. (B) Once a detection window is satisfied, a programmable duration timer is started, nominally 2.5 s for ventricular tachycardia (VT) and 1 s for ventricular fibrillation (VF). If after the duration timer expires the last detected interval is in the zone of the timer, detection is met and therapy is delivered (unless a detection enhancement is programmed; see text). When multiple zones are programmed “on,” the higher zone takes priority over the lower zone. Up to three tachycardia zones (VT-1, VT, and VF) with independently programmable criteria and therapies may be used to enhance detection and specificity of therapy. (C) If the VT detection window does not remain satisfied until the end of the VT duration window, VT duration resets to zero, and timing will resume when the window becomes resatisfied. ATP, antitachycardia pacing. (Modified with permission from Boston Scientific ICD reference manual.)
Fig. 8.60 Consecutive interval counting ventricular tachycardia (VT) detection (Medtronic). At the top is the ventricular electrogram with the individual intervals labeled in milliseconds. Beneath the electrograms are the corresponding markers for each sensed event. Next is a graph of the individual intervals, with dashed lines delineating the tachycardia detection interval (TDI) and fibrillation detection interval (FDI). At the bottom, the graph displays how each sensed event affects the VT event counter. The dashed line denotes the programmed number of intervals needed to detect VT (NID). In this figure, the third electrogram occurs at a cycle length of 300 ms, which is less than the programmed TDI of 400 ms; the VT counter increases to 1. Note that the marker channel displays a VT sense. At point “A,” the VT counter is reset to zero by a sensed interval of 600 ms, which is longer than the TDI. At point “B,” VT detection occurs, as the counter reaches the programmed NID of 8. Depending on the type of therapy programmed, antitachycardia pacing or charging of the capacitors would begin at this point. (From Friedman, Stanton. By permission of Futura Publishing Co.)

Fig. 8.61 Fast ventricular tachycardia (FVT) detection via ventricular tachycardia (VT). In this example, the FVT zone overlaps the upper (faster) end of the VT zone. The second complex (interval, 400 ms) is detected as VT (“TS” for tachycardia sense). The next interval (320 ms) is detected as FVT (“TF”). Once the VT counter is satisfied (at the “TFI” marker), the arrhythmia is classified as FVT, since at least one of the previous eight events was in the FVT zone. The first FVT therapy, in this case antitachycardia pacing, is delivered.
Committed and noncommitted shocks
All early defibrillators delivered committed shocks; once a tachyarrhythmia was detected and capacitor charging initiated, a shock was committed to follow, irrespective of arrhythmia termination. Current generation devices can be programmed to deliver noncommitted therapy; after detection and capacitor charging, the pulse generator confirms continuing tachyarrhythmia before delivery of a shock. If the arrhythmia has ended, the capacitors remain charged, but therapy is withheld and the device continues to monitor the rhythm (Fig. 8.63). For a given episode, typically only the first shock can be programmed as noncommitted; for redetected arrhythmia, shocks are committed. The exception is St Jude ICDs, in which all therapies are noncommitted.

Detection enhancements
Inappropriate therapy delivered for non-ventricular arrhythmias affects 8–40% of ICD recipients, has a deleterious effect on quality of life, can be associated with proarrhythmia, and leads to poor tolerance of life-saving ICD therapy. Consequently, detection enhancements (SVT-VT discrimination algorithms) have been developed to improve the specificity of rhythm classification. When programmed “on,” detection enhancements prevent delivery of therapy despite a heart rate in the tachycardia zone if other factors (e.g., a narrow QRS complex) suggest a supraventricular mechanism. Since the overlap in heart rate between ventricular and supraventricular arrhythmias occurs predominantly in the “slower” tachycardia zones, and since the very fast rhythms detected in the VF zone (usually > 185 to 200 bpm) are more likely to be hemodynamically unstable and require immediate therapy, detection enhancements are typically applied only in the VT zones. In clinical trials, approximately 25% of inappropriate therapies are due to SVTs that exceed the rate at which discriminators are applied. Emerging data suggest ap-
Application of SVT-VT discriminators up to rates of 200 bpm may minimize ICD morbidity. The availability and performance of detection enhancements is linked to the detection zones programmed for ventricular arrhythmias. In Boston Scientific ICDs, detection enhancements are programmed “on” for the entirety of one or both VT zones (in older devices, before Vitality™, discriminators were programmable only in the slowest VT zone). In St Jude ICDs, detection enhancements are programmable independently within the two VT zones (i.e., the cut-off rate beyond which discriminators no longer apply need not match a VT or VF boundary), but they cannot overlap with the VF zone. Medtronic ICDs permit independent detection enhancement programming that includes overlap with the VF zone, but their function in dual-chamber ICDs is degraded in the VF zone to prevent misdiagnosing VF as AF.

There are two broad categories of detection enhancements: morphology-based enhancements and interval-based enhancements. Single-chamber defibrillators must use QRS morphology or interval-based rhythm patterns such as rate of onset or regularity to enhance rhythm diagnosis; dual-chamber defibrillators (discussed in the next section) can also compare and analyze atrial and ventricular rates and patterns. A summary of single-chamber detection enhancements and their operation is provided in Table 8.4. The general principles guiding optimal programming of single-chamber detection enhancements are as follows: (i) specificity enhancements are applied to slower (<185–200 bpm), hemodynamically tolerated VT zones, in which a modest delay in therapy is tolerable; (ii) discrimination algorithms that continuously reassess the rhythm during tachycardia (such as “stability” or “morphology”) are preferable and should generally be used in patients with known SVTs or AF; (iii) discrimination algorithms that perform a single classification based on a limited number of ventricular events (such as “onset”) should be used more judiciously or in conjunction with sustained duration timers that deliver therapy irrespective of classification if the tachycardia duration exceeds a programmable time limit. The overall performance of discriminators is strongly influenced by the logic applied when several are programmed “on” simultaneously. Limited data suggest that analysis of ventricular electrogram morphology, alone or in combination with stability, provides the best single-chamber SVT-VT discrimination for the initial detection of VT.

Technical details vary among manufacturers, as do corresponding recommended program values, which are summarized in Table 8.5.

Morphology
This algorithm is based on acquisition of a patient-specific electrogram template during sinus or baseline rhythm. The morphology of QRS complexes during tachycardia are compared with the template, on the assumption that supraventricular rhythms have the same morphology as the sinus rhythm template. Common elements present in all morphology algorithms include: (i) creation of a template by mathematically extracting electrogram features and storing them; (ii) recording of
Table 8.4 Single-chamber detection enhancements

<table>
<thead>
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<th>Detection enhancement</th>
<th>Function</th>
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| Stability [inhibit therapy if ventricular rate is unstable (i.e., variable QRS intervals); or in some devices, accelerate therapy if unstable] | • Main use: differentiate atrial fibrillation (irregular, unstable intervals) from ventricular tachycardia (regular, stable intervals)  
• Strength: continuously assesses ongoing tachycardia, permitting detection even if initial misclassification occurs  
• Limitation: antiarrhythmic drugs may result in irregular intervals during VT leading to misclassification as SVT; atrial flutter with fixed conduction may be regular, leading to misclassification as VT  
• In Boston Scientific devices, can also be used to accelerate therapy (e.g., to avoid antitachycardia pacing in patient with known polymorphic ventricular tachycardia, which is irregular and unstable) |
| Onset (inhibit therapy if gradual onset) | • Main use: differentiate sinus tachycardia (gradual onset) from ventricular tachycardia (sudden onset)  
• Strength: one of limited number of approaches to distinguish sinus tachycardia from VT  
• Limitation: single assessment at arrhythmia onset does not permit “correction” if misclassification occurs due to ectopy (making ST onset appear abrupt) or VT onset below detection rate with gradual acceleration across ST-VT boundary (leading to misclassification of VT as ST) |
| Morphology discrimination (inhibit therapy if intracardiac morphology matches baseline rhythm morphology) | • Main use: differentiate ventricular from supraventricular arrhythmias irrespective of interval timing  
• Strength: continuously assesses ongoing tachycardia, permitting detection even if initial misclassification occurs; permits differentiation of regular flutter from VT  
• Limitation: misclassifies aberrant SVT as VT; not useful immediately following shock due to shock-related distortion of the electrogram |
| Sustained rate duration (override inhibitor if fast rate persistent) | • Main use: limit the length of time inhibitor can withhold therapy during a high-ventricular-rate episode  
• Overrides therapy inhibitors after duration timer expires  
• Strength: prevents VT underdetection; particularly useful with algorithms that assess rhythm at single point in time (such as onset)  
• Limitation: degrades specificity by increasing the risk of shocking SVT  
• Many devices also have timers to limit the total duration of antitachycardia pacing therapies; after the programmed time elapses, the device delivers a shock, even if pacing therapies remain |

Table 8.5 Recommended programming of SVT-VT discriminators in single-chamber ICDs

<table>
<thead>
<tr>
<th>Medtronic</th>
<th>Boston Scientific</th>
<th>St Jude</th>
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<tbody>
<tr>
<td>Stability*</td>
<td>40–50 ms, NID = 16</td>
<td>24–40 ms, duration 2.5 s†</td>
</tr>
<tr>
<td>Onset</td>
<td>84–88%</td>
<td>9%†</td>
</tr>
<tr>
<td>Morphology</td>
<td>3 of 8 electrograms ≥ 70% match</td>
<td>Rhythm ID “on”†</td>
</tr>
</tbody>
</table>

*Less strict values are required for patients taking type I or III antiarrhythmic drugs.  
†In devices with the Rhythm ID algorithm, stability and onset are not available.
electrograms during an unknown tachycardia; (iii) time aligning the template and tachycardia electrograms; (iv) classifying each tachycardia electrogram as a match or nonmatch based on its comparison with the template; (v) classifying the tachycardia as SVT or VT based on the number of electrograms that match the template. Various manufacturer’s morphology algorithms differ in their electrogram source(s), methods of quantitative representation and alignment. Details of their function are shown in Figs 8.64–8.66.

Since morphology algorithms continually reassess a tachycardia, the risk of underdetection of significant arrhythmias while using them without override timers that override therapy inhibition is very small.43 In single-chamber ICDs, morphology algorithms are the only discriminators that distinguish regular SVT [such as atrial flutter or atrioventricular nodal reentrant tachycardia (AVNRT)] from VT.

Morphology algorithms have common failure modes (reviewed in detail in Chapter 10, “Troubleshooting”), some of which can be prevented with appropriate programming steps, summarized here. An inaccurate template may be recorded because the electrogram has evolved (due to lead maturation or the development of bundle branch block) or because the template was recorded during an abnormal rhythm. Programming automatic template updates “on”, and assessing templates at routine follow-up, minimizes the risk of misclassification due to an inaccurate template of the baseline rhythm. If automatic updates are not available, morphology should be disabled until a stable, chronic electrogram is present. Electrogram truncation occurs when the electrogram signal exceeds the dynamic range of the sensing amplifier, so that the maximum or minimum portions of the signal are clipped. Variable truncation may result in mismatch of two signals that are otherwise identical. Truncation is eliminated by programming the amplitude scale so that the electrogram used for morphology analysis occupies 25–75% of the dynamic range. Alignment errors occur when similar electrograms are misaligned, leading to error in the calculated match score. Template and tachycardia electrograms are aligned by matching the morphology electrogram peak (Medtronic), onset (St Jude), or near-field peak (Boston Scientific). Alignment errors may occur due to truncation (Medtronic), or due to rate-related, at times subtle, electrogram changes (St Jude, Boston Scientific, or Medtronic). Avoiding truncation eliminates some alignment errors. In dual-chamber ICDs, recording a template while atrial pacing at higher rates (e.g., 120 ppm) may prevent misalignment due to subtle electrogram changes (i.e., minor aberrancy). Pectoral myopotentials may distort the electrogram used for analysis leading to classification errors. In Medtronic ICDs, a different electrogram source can be selected. In St Jude ICDs, near-field electrograms are used, so that pectoral myopotentials do not affect the morphology algorithm. Boston Scientific ICDs do use the far-field electrogram and lack a programmable adjustment to compensate for pectoral oversensing; limited early data suggest this problem is uncommon.48,49 Rate-related aberrancy results in misclassification of SVT as VT due to tachycardia–template mismatch. In patients with known rate-related aberrancy, acquiring the template during rapid atrial pacing or disabling the morphology algorithm may prevent rhythm misclassification.

Stability
This detection enhancement is used to differentiate AF from VT based on the difference in RR interval patterns between the two rhythms. When programmed “on,” the stability algorithm withholds therapy despite ventricular rates in the tachycardia zone if the cycle length intervals are irregular (Figs 8.67 and 8.68). The rationale for this approach is that VTs have a relatively stable heart rate with little variation in RR intervals, in contrast to the marked variability seen in AF. In one study, for example, the average stability (i.e., RR variability) during VT episodes was 16 ± 15 ms, compared with 49 ± 15 ms during AF.50 The stability enhancement prevents inappropriate detection of AF in up to 95% of AF episodes, with only a minimal decrease in VT sensitivity.50–52 Atrial flutter may be regular and difficult to differentiate from VT in single-chamber devices; similarly, RR intervals tend to regularize when atrial fibrillation is rapidly conducted (above approximately 170 bpm), limiting the utility of interval stability algorithms. Antiarrhythmic medications may also affect the algorithm’s performance. Use of amiodarone or Class IC antiarrhythmic drugs (e.g., flecainide or propafenone) may cause monomorphic VT to become irregular or polymorphic VT to slow, leading to rhythm misclassification.53,54

Onset
This algorithm differentiates VT from sinus tachycar-
Fig. 8.64 Wavelet morphology algorithm (Medtronic). (A) A template is recorded during normal intrinsic rhythm (with rate < 100), top left. The template is automatically updated (after confirmation that complexes are non-ectopic) continuously, as needed. The electrogram source for the analysis is programmable (nominally RV coil to can). A mathematical transform is used to extract the electrogram features (top right figure). During tachycardia, the electrogram is recorded (bottom left figure), features are extracted in real time (bottom right panel), and the tachycardia and template electrograms are mathematically compared to derive a match score. Each complex is classified as SVT if the match score exceeds a programmable threshold value (nominally 70%). If ≥3 of 8 complexes in a rolling window are SVT, the ongoing episode is classified as SVT. (B) The Haar transform is used to extract waveform characteristics and store them. (Reproduced with permission from Swerdlow CD, Brown ML, Lurie K et al; Journal of Cardiovascular Electrophysiology Vol. 13, No. 5, May 2002.)
Fig. 8.65 (A) Morphology discrimination (St Jude). An electrogram template is made during sinus rhythm, unique for each patient. During a tachyarrhythmia, each tachycardia complex ("test complex") is compared with the template. A morphology score is derived from the sum of the differences of aligned test complex and template. The % Match score is a function of the difference between the areas under the aligned complexes (i.e., [area A – area A’] + [area B – area B’] + [area C – area C’]). The % Match score required to consider a supraventricular complex is programmable. (B) Example of morphology discrimination and appropriate therapy for VT with 1:1 VA conduction. Bipolar atrial electrogram (RA), dual-chamber Marker Channel, and rate-sensing (RV) electrogram are shown. Asterisk denotes onset of VT during sinus tachycardia, identified by abrupt acceleration of ventricular rate and change in electrogram morphology without change in atrial rate. Morphology discriminator requires 5 of 8 match scores ≥60% to withhold therapy. During sinus tachycardia, scores exceed 60%. (Five are 100%, scores are labeled in the figure as "Match score.") During VT, most scores are <60%. Check marks above Marker Channel indicate that morphology algorithm classifies beats as supraventricular (labeled in the figure as "Match"). An "X" mark in the "match" row indicates a beat classified as VT morphology. "Trigger" in lower panel indicates detection of VT. "D =" at onset of ATP indicates that atrial rate = ventricular rate. "S" denotes intervals interval the "Sinus" zone longer than the VT detection interval of 400 ms. "T" denotes intervals in VT zone. DDI, mode switch. Time line is in section. Additional observations confirm the diagnosis of VT. The VT cycle length is moderately irregular. Changes in VV interval precede those in AA interval. Ventricular antitachycardia pacing (ATP) at right of lower panel results in transient VA block without acceleration of the atrial rate followed by 1:1 VA conduction. The near simultaneous atrial and ventricular activation during tachycardia is more typical of typical (antegrade-slow, retrograde-fast) AV nodal re-entrant tachycardia than VT, but the shortening of the AV interval at the onset of tachycardia is inconsistent with this diagnosis. Pacing-induced VA block without acceleration of the atrial rate is also unusual in AV nodal re-entry. (Reproduced with permission from Swerdlow, Friedman. PACE 2005; 28:1322–46, Blackwell Publishing)
dia based on the abrupt onset present in most VTs, in contrast to the gradual onset during sinus tachycardia (Fig. 8.69). The onset criterion rejects inappropriate detection of 64–98% of sinus tachycardia with heart rates in the VT zone, but results in underdetection of 0.5–5% of VTs. Unlike stability, which continuously re-evaluates the rhythm diagnosis during tachycardia, onset is determined only once. Moreover, since ectopy preceding VT may mitigate the abruptness of arrhythmia onset, this enhancement is best limited to slow VT zones (heart rate < 140–150), where the risk for overlap with sinus tachycardia is greatest, to avoid underdetection of faster VT. Additionally, when available, sustained rate duration is often used with the onset algorithm. Since many cardiac patients are unable to maintain prolonged sinus tachycardia, the presumption is that sustained regular tachycardia lasting > 1–3 min reflects VT. This approach ensures that VT is treated, but at the cost of arrhythmia specificity. In patients prone to sinus tachycardia and slow VTs, use of β-blockers and digitalis, which improve survival and reduce symptoms, respectively, may lower the risk of sinus tachycardia crossing the VT boundary.

**Dual-chamber detection**

In addition to providing dual-chamber pacing functionality, dual-chamber defibrillators use the information acquired simultaneously from the atrial and the ventricular leads in an effort to enhance arrhythmia diagnosis. Although different manufacturers have...
 adopted very different approaches, the algorithms share many common principles:

- Comparison of atrial and ventricular rates. A ventricular rate that exceeds the atrial rate accurately identifies VT, eliminating the need for additional analysis.
- Identification of the presence of fibrillation in the atrium to validate the significance of RR irregularity.
- Identification of variation in PR intervals and presence of N:1 AV association to distinguish supraventricular rhythms such as sinus tachycardia and atrial flutter from VT.58–60

Overall, dual-chamber enhancements have been shown to diagnose 60–95% of SVT episodes correctly47 without significantly missing VT episodes. Early dual-chamber ICDs were similar or slightly superior to single ICDs in correctly discriminating SVT from VT. More recently, prospective, randomized trials have found that the odds of inappropriate detection of VT as VT were decreased by half with the use of dual-chamber detection enhancements.43 Although expert opinion is divided, use of dual-chamber algorithms to improve SVT-VT rhythm classification is reasonable in patients in whom a VT zone with rates < 200 bpm will be programmed, who are not in chronic atrial fibrillation and who do not have complete or high-grade AV block. This is discussed in more detail in Chapter 4, “Generator and Lead Selection.”

**Comparison of atrial and ventricular rates**

Since the ventricular rate exceeds the atrial rate in 80–90% of VTs in the VT zone of dual-chamber ICDs,47–52 comparing atrial and ventricular rates is a simple and powerful SVT–VT discriminator if the atrial rate can be reliably sensed. Algorithms that compare atrial and ventricular rates as their first step (Boston Scientific Rhythm ID™ and St Jude) only apply single-chamber discriminators to < 10% of VTs, reducing the risk that they will misclassify VT as SVT. Dual-chamber
discrimination depends on accurate atrial sensing to classify arrhythmias reliably; the predominant cause of detection errors in dual-chamber ICDs has been atrial sensing errors. Atrial sensing function is optimized at implantation by lead placement at a site with a P wave of at least 1 mV and with absent or small far-field R waves (<25% of the atrial electrogram amplitude, in our experience). Selecting a lead with an interelectrode spacing of <10 mm minimizes far-field R waves since closely spaced electrodes create a smaller “antenna.” Early observations with a recently introduced lead with 1.1-mm interelectrode spacing support the concept that more closely spaced electrodes improve atrial sensing specificity, and early experimental work suggests totally intramyocardial electrodes may result in highly tissue-specific sensing.

When P waves are not reliably sensed by an ICD, reprogramming sensitivity may result in acceptable function without surgical intervention. In St Jude ICDs, atrial blanking after sensed ventricular event is programmable. Extending the blanking period lowers the risk of far-field R-wave oversensing, but increases the risk of undersensing atrial fibrillation. The atrial sensing threshold start and decay delay are
also programmable, similar to the sensing function in the ventricle (Fig. 8.70). In Boston Scientific Vitality™ and newer ICDs, atrial blanking is programmable to a series of fixed values, or to SMARTSense™, in which after a short blanking period (15 ms), a period of reduced autoadjusting sensitivity is present to minimize the risk of far-field R-wave oversensing, followed by full atrial sensitivity (Fig. 8.70). Figure 8.71 demonstrates far-field R-wave oversensing that is eliminated after prolonging the PVAB period. Older devices used fixed blanking periods following ventricular events, which could lead to underestimation of the atrial rate during atrial flutter and inappropriate therapy (due to the incorrect calculation that V > A rate). Some Medtronic ICDs have optional atrial blanking to reject far-field R waves, but the nominal setting is no atrial blanking after ventricular events. Instead, they analyze the pattern of activity sensed on the atrial lead to determine algorithmically whether some events recorded on the atrial channel reflect far-field R waves (Fig. 8.70). Intermittent sensing of far-field R waves or frequent premature complexes disrupts the pattern, leading to algorithm error and tachycardia misclassification. Decreasing the atrial sensitivity may eliminate intermittent far-field R-wave oversensing at the expense of increasing the risk of undersensing atrial fibrillation. In our experience, the risk of undersensing atrial fibrillation is low with atrial sensitivity as low as 0.45 mV. Far-field R-wave oversensing that occurs only after paced ventricular events does not increase the risk of inappropriate detection of SVT as VT, as discrimination is applied only during sensed tachycardias greater than the pacing upper rate limit. Elimination of far-field R-wave oversensing at implant and during follow-up results in effective dual chamber SVT–VT discrimination.

Given the marked differences in technical approach to dual-chamber detection taken by manufacturers, each manufacturer is considered separately, as are programming considerations. Table 8.6 summarizes recommended programming of SVT-VT discriminators for dual-chamber ICDs.

Enhanced onset and stability (Boston Scientific)

All Boston Scientific dual-chamber defibrillators introduced before Vitality 2™ add two programmable detection enhancements that use the atrial lead to enhance specificity: V Rate > A Rate and AFib Rate Threshold. These two additional features work in conjunction with the ventricular-based detection enhancements, described above (stability and onset) and are thus applied only in VT zones. The V Rate > A Rate feature utilizes the fact that a ventricular rate greater than an atrial rate is pathognomonic for VT. When V Rate > A Rate (ventricular rate greater than atrial rate) is “on,” therapy inhibitors (onset or stability, or both) are bypassed and therapy is immediately delivered to tachycardias with a ventricular rate...
Fig. 8.69 The onset criterion (as implemented by Medtronic). (A) The first 300-ms interval is less than the tachycardia detection interval (TDI) and 81% of the average of the preceding four intervals; thus, onset is satisfied and the ventricular tachycardia (VT) counter incremented. At point "A," an interval greater than the TDI resets the VT counter to zero. Onset is again met with the next interval, and VT counting resumes anew, culminating in detection at point "B." (B) Onset is programmed to 81%. The heart rate increases gradually (as in sinus tachycardia), so that despite cycle lengths that are less than the TDI (last four intervals), VT is not detected, since no interval is 81% of the average of the preceding four intervals. Note that the marker channel continues to show normal sensing (not VT sensing) and that the VT event counter is not incremented. In newer single-chamber Medtronic defibrillators, onset is replaced by other algorithms (electrogram width). FDI, fibrillation detection interval; NID, number of intervals needed to detect ventricular tachycardia. (From Friedman, Stanton. By permission of Futura Publishing Co.)
greater than the atrial rate by 10 bpm (Fig. 8.72). If the ventricular rate is not greater than the atrial rate (which is indicated as false on the episode detail report), therapy continues to be inhibited. In that case, the V Rate > A Rate analysis continues until the ventricular rate exceeds the atrial rate or other enhance-

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**Fig. 8.70** Methods of prevention of far-field R-wave oversensing (FFRWO) on the atrial channel. Top panel: In St Jude ICDs, sensing in the atrium following a sensed event is analogous to ventricular sensing. Threshold Start and Decay Delay are programmable. Middle panel: Boston Scientific ICDs use a short (15 ms) blanking period, followed by variable sensitivity designed to minimize FFRWO and permit sensing of AF. Bottom panel: Most Medtronic ICDs do not blank the atrial channel after sensed events, but instead, when there are two atrial events within a ventricular interval, look for a short-long pattern of A-A intervals to indicate FFRWO. After paced ventricular events, atrial sensitivity is transiently decreased. FFRW: far-field R wave oversensing.

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**Fig. 8.71** Programming to eliminate far-field R-wave oversensing. In the left panel, far-field R waves are sensed on the atrial channel. Note in the boxed intervals the numbers “250” and “609.” The time from the paced A to the oversensed R wave on the atrial channel is 250 ms. The ventricular signal is seen on the atrial electrogram. In the right panel, the ventricular signals are still seen on the atrial electrogram. However, due to reprogramming, they are no longer sensed on the atrial channel, and the AA interval (859 ms) happens to be the same as the VV interval. Note that sensing is sufficiently programmable to avoid sensing far-field R waves that have a larger amplitude than the atrial electrogram itself.
Fig. 8.72 Use of V Rate > A Rate (ventricular rate greater than atrial rate) to bypass therapy inhibitors. From top to bottom are the atrial and ventricular electrograms, the V intervals, the calculated stability, and the atrial and ventricular rates. The 10 most recent PP intervals and RR intervals are used to assess the rate in each chamber. An irregular ventricular tachycardia (VT) is present. The VT has a shorter interval (is faster) than the slow VT (VT-1) cut-off threshold. Thus, VT detection and duration are both satisfied (top box). Because the RR intervals are variable, the stability threshold is above the programmed value (in this case, 24 ms; not shown on graph). This inhibits therapy in a single-chamber device. However, since the ventricular rate is greater than the atrial rate, the stability inhibitor is bypassed and appropriate therapy delivered.

Table 8.6 Recommended programming of SVT-VT discriminators in dual-chamber ICDs

<table>
<thead>
<tr>
<th>Medtronic PR Logic™</th>
<th>Boston Scientific One Button Detection Enhancements™</th>
<th>Boston Scientific Rhythm ID™</th>
<th>St. Jude Rate Branch™</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aflib/Aflutter: “on”</td>
<td>AFib rate threshold 200 beats/min “on”</td>
<td>Rate branch “on”</td>
<td></td>
</tr>
<tr>
<td>Sinus Tach: “on”</td>
<td>Onset 9%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other 1:1 SVTs: “off”</td>
<td>Inhibit if unstable 10 ms</td>
<td></td>
<td>A&gt;V branch: Morphology</td>
</tr>
<tr>
<td>1:1 SVT boundary: 66%†</td>
<td>V rate &gt; A rate “on”</td>
<td></td>
<td>A&gt;V branch morphology combined</td>
</tr>
<tr>
<td></td>
<td>Sustained rate duration 3 min</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note that these are generally useful settings, which may be customized for the individual patients.

† In devices in which this feature is programmable.

*Stability at 80 ms with AV association of 60 ms.
ments indicate that therapy is warranted, at which time treatment is delivered.

The AFib Rate Threshold increases specificity when used with stability by withholding therapy for unstable (irregular) ventricular rhythms only when the atrial lead confirms the presence of AF. If an unstable (irregular) ventricular rhythm is in the tachycardia zone but the atrial lead does not confirm fibrillation, therapy is delivered. If fibrillation is present in the atrium, the ventricular rate is unstable, and AFib Rate Threshold is “on,” therapy is withheld until the atrial rate drops below the AFib Rate Threshold, the ventricular rhythm becomes stable, or the sustained rate duration (SRD) timer expires (if SRD is programmed).

The main limitations of this earlier Boston Scientific algorithm are the inability to detect VT with 1:1 VA conduction and a gradual onset, the inability to reject 1:1 atrial tachycardias with abrupt onset, and inappropriate detection of rapidly conducted atrial fibrillation due to the obligatory postventricular atrial blanking. The risk of inappropriate classification of atrial fibrillation is minimized by programming the shortest possible postventricular atrial blanking (45 ms) and the AFib Rate to the minimum value of 200 bpm; this combination maximizes the possibility of sensing AF. Additionally, programming a highly specific stability value of 10% takes advantage of the fact that VT during AF is highly regular. However, highly regular 2:1 flutter will be classified as VT, and a slightly irregular VT (which may occur in the setting of antiarrhythmic drugs) will be classified as SVT. Detection enhancements are applied following shocks, but not following ATP.

Rhythm ID™ (Boston Scientific)

In dual-chamber ICDs, Rhythm ID™ integrates interval-based and morphology-based detection enhancements. Rhythm ID™ is programmed either “on” or “off,” without additional operator programmable subparameters. As shown in Fig. 8.73, when programmed “on,” the algorithm first compares the ventricular rate and the atrial rate. If the ventricular exceeds the atrial rate (by 10 bpm), VT is declared and therapy is delivered. If the ventricular rate does not exceed the atrial rate, the Vector Timing and Correlation (VTC) figure morphology algorithm is applied to the ventricular electrogram, as described above. If the VTC algorithm finds a match between the baseline template and tachycardia, therapy is withheld. If a mismatch is found, then a screen for rapidly conducted atrial fibrillation (defined as A rate > 200 bpm and V rate unstable > 20 ms) is performed. If atrial fibrillation is absent, therapy is delivered. During redetection, the VTC component of the algorithm is not included (Fig. 8.73B), since electrogram morphology may be distorted by the shock. The benefit of this algorithm is that it requires no custom programming; the limitation is that its errors cannot be corrected by troubleshooting.

PR Logic™ (Medtronic)

Current dual-chamber Medtronic defibrillators combine single-chamber and dual-chamber detection enhancements, some of which may have different zones of operation. The stability and onset detection enhancements are explicitly linked to the VT (or FVT via VT) detection zones, and are not affected by the independently programmable “SVT Limit.” Onset and stability do not integrate atrial information, and function in a manner identical to their single-chamber counterparts. During a tachycardia in the VT zone, if either onset or stability is programmed “on” and indicates SVT, the VT counter is reset to zero, detection is not met, and additional detection enhancements (i.e., PR Logic) are not applied. For this reason, onset is not routinely programmed “on” as sinus tachycardia is also distinguished from VT using PR Logic™ (discussed below). Stability remains useful in patients with atrial fibrillation since it is the only detection enhancement applied during redetection.

After the VT counter reaches the number of intervals needed to detect (NID), the wavelet algorithm and/or PR Logic are applied if programmed “on”. These algorithms are programmed to function in a heart range defined by the “SVT limit,” which can cross VT/VF detection boundaries, as previously noted.

Tachycardias with ventricular rates in a VT or VF zone not inhibited by other detection enhancements and in the SVT zone are further analyzed by the PR Logic™ algorithm. In addition to the SVT limit, the PR Logic algorithm has three programmable parameters, each of which is programmed either “on” or “off”: a fibrillatory flutter, sinus tachycardia, and other 1:1 SVTs. When programmed “on,” positive identification of one of these arrhythmias is required to withhold ventricular therapies. As shown in Fig. 8.74, the algorithm initially seeks to determine whether a dual tachycardia (simul-
Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach

4 Initial Detection

![Diagram of Initial Detection algorithm]

Redetection: Morphology not used

![Diagram of Redetection algorithm]

PR pattern

The number and position of P waves relative to R waves are analyzed for each of the two preceding RR intervals. On the basis of the number of atrial events and the location relative to R waves, one of 19 codes is assigned to the intervals (Fig. 8.75). Any cardiac rhythm generates a string of code letters, and these are compared with sequences known to occur during specific rhythms, such as sinus tachycardia or sinus tachycardia with PVCs. This pattern-matching is continuous, analogous to a word processing spelling checker analyzing a stream of new text by comparing it with known catalogs of text. As noted in Fig. 8.74, pattern analysis is used not alone, but in conjunction with the other elements of the algorithm (rate, regularity, AV dissociation, far-field R wave, and AF evidence) to
make a rhythm classification. In older versions of PR Logic™, a programmable VT–ST boundary modified differentiation of VT with 1:1 VA conduction from SVT (predominantly sinus tachycardia). New ICDs use a nonprogrammable adaptive algorithm for this differentiation.

**Fig. 8.74** Detection process in PR Logic (Medtronic). (A) With each sensed event, ventricular rate detection, as found in single-chamber implantable cardioverter-defibrillators, is applied. If rate detection criteria are met in the ventricular tachycardia (VT) zone, the stability algorithm (identical to that in single-chamber defibrillators) is applied if programmed “on.” If the rhythm is not unstable, the algorithm proceeds to see whether the dual-chamber supraventricular tachycardia (SVT) criteria are programmed “on” and whether the ventricular rate exceeds the SVT limit. The next step screens for dual tachycardia (simultaneous atrial and ventricular tachycardia); if it is present, ventricular therapy is delivered. If dual tachycardia is absent, the presence of an SVT (“AFib/AFFlutter,” “Sinus Tach,” or “Other 1:1” SVT) is assessed by use of the dual-chamber SVT criteria. If an SVT is not positively identified, ventricular therapy is delivered. (B) Dual-chamber SVT criteria. The column headings are the six elements used for arrhythmia classification; the table indicates the elements required for diagnosis of each arrhythmia by PR Logic. AF, atrial fibrillation; AV, atrioventricular; FFRW, far-field R waves; FVT, fast ventricular tachycardia; VF, ventricular fibrillation.

**Fig. 8.75** Couple code syntax analysis in PR Logic. The PR pattern code is one of six elements used by PR Logic for arrhythmia classification. Details in text. (From Olson.68 By permission of Futura Publishing Co.)
Regularity
This element measures the variability in RR cycle length. Importantly, although conceptually similar to the stability inhibitor, the regularity counter is part of PR Logic and is independent of and not related to the stability algorithm.

AV dissociation
The mean of the most recent eight PR intervals is computed, and an individual PR interval is considered dissociated if its absolute difference from the mean is >40 ms. If four of the last eight intervals in a rhythm are dissociated, the rhythm is declared dissociated.

Far-field R wave
To avoid undersensing of P waves, the algorithm does not include cross-chamber blanking in the atrium after sensed ventricular events and has short (30 ms) atrial blanking after a paced ventricular event. Consequently, far-field R waves are not uncommonly sensed on the atrial channel (depending on atrial lead position). This subalgorithm determines whether sensed events in the atrial channel are likely to be due to far-field R waves by the presence of short–long intervals, the result of which is then incorporated into rhythm analysis (Fig. 8.76). As a practical matter, if far-field R-wave oversensing is consistently present or consistently absent, the algorithm functions effectively. Intermittent sensing of far-field R waves may lead to tachycardia misclassification.

AF evidence
An up-down atrial event counter is used to evaluate atrial tachyarrhythmias. When two or more atrial events occur during an RR interval, the counter is augmented by one. If the preceding RR interval had two or more atrial events but the most recent RR interval had zero or one atrial event, the counter is unchanged. If both the present and the preceding RR intervals had zero or one atrial event, the counter total is decreased. A value of six or more on this counter is evidence for AF, since P and R relationships during AF do not have reliable pattern information.

In summary, the PR Logic algorithm uses the six elements described to classify tachyarrhythmias, as shown in the flow diagram and table in Fig. 8.74. Its function is to distinguish ST, AFib/Aflutter and other 1:1 SVTs from VT, and it is not applied during redetection. Due to its dependence on the rates and patterns of AA, VV, AV and VA intervals to classify arrhythmias, it is most susceptible to intermittent far-field R-wave oversensing, which is not effectively algorithmically identified. In the first month postimplant, rejection of 1:1 SVTs is not programmed “on,” since atrial lead dislodgment into the ventricle may result in the inappropriate classification of VT as SVT. Additionally, in the rare patient with atrioventricular nodal reentrant tachycardia or orthodromic tachycardia (both typically 1:1 SVTs), ventricular antitachycardia pacing usually terminates the SVT, so that an intentional decision to deliver therapy may be appropriate.

St Jude Rate Branch™
The first step in this dual-chamber algorithm is comparison of the atrial and ventricular rates to sort the arrhythmia as A = V, A > V or V > A (Fig. 8.77). If the ventricular rate is greater than the atrial rate, the tachycardia is classified as ventricular tachycardia and therapy is delivered. In the other two branches, single-chamber detection enhancements are applied individually or in combination using “ANY” or “ALL” operators. Using “ANY,” the algorithm detects VT if any discriminator in that rate branch classifies the tachycardia as VT, increasing sensitivity at the expense of specificity. Using “ALL,” on the other hand, requires all discriminators to indicate VT for VT classification, increasing specificity at the expense of sensitivity. Since atrial flutter, atrial fibrillation and rapid atrial tachycardia are typically sorted into the A > V branch, morphology and stability are recommended. The “ANY” operator is used to maintain a high sensitivity for VT. Sinus tachycardia, slower atrial tachycardias and ventricular tachycardia with 1:1 VA conduction are sorted into the A = V rate.
branch. In this branch, morphology alone is recommended for SVT–VT discrimination.

**Ventricular therapies**

**Antitachycardia pacing**

Antitachycardia (overdrive) pacing consists of short bursts of pacing impulses at rates 10–20% greater than the tachycardia. It terminates 80–95% of arrhythmia episodes, eliminating the need for shocks. By pacing the ventricle at a rate greater than the tachycardia, the pacing impulses may enter the tachycardia circuit and render it refractory, so that the returning tachycardia wavefront does not find excitable tissue (further discussed in Chapter 1). Overdrive pacing can be delivered as bursts (all pulses in a sequence at the same rate), as ramps (sequential pulses within a sequence delivered progressively faster), or as a combination (Fig. 8.78). Although no ATP scheme has been shown superior to another for VTs < 200 bpm, for faster VTs, burst is more effective and less likely to cause acceleration. In 1–5% of patients, ATP may accelerate the arrhythmia, which may lead to shock. Due to the safety and efficacy of ATP, routine electrophysiological study to test or tailor therapy is not necessary.

In addition to terminating VT, ventricular ATP also reduces shocks for inappropriately detected SVT either by terminating SVT or delaying shock therapy, providing time for the SVT to terminate or slow. Since ATP improves quality of life and prevents appropriate and inappropriate shocks, it should be programmed “on” empirically in most patients, even if its efficacy has not been assessed.

**Defibrillation**

Defibrillation is the mainstay of therapy for VF and rapid VT. Its efficacy in VF termination exceeds 98%. Current-generation defibrillators deliver up to 6–8 shocks per episode to ensure arrhythmia termination. Available maximal shock energies range from 25 to 36 J, enough to defibrillate most patients through an endocardial approach with biphasic waveforms and modern leads. The mean energy required for successful defibrillation in current devices is approximately 10 J. As discussed in Chapter 1, defibrillation threshold or margin testing is typically performed at implant to assess the energy requirements for defibrillation. Programming shock energy at 10 J above the defibrillation threshold results in a first shock success rate of 87–93% during spontaneous VT/VF. To maximize defibrillation success, all subsequent shocks are programmed to maximum output, and the polarity of the last shock in the sequence is reversed when this feature is available.

Some experts recommend programming a low first-shock strength when patient-specific implant testing indicates a low energy requirement for defibrillation (e.g., DFT + 10 J). Potential advantages of a lower first-shock energy include a reduced risk of syncope (due to faster charge time), battery preservation (particularly in the setting of frequent charges for VT storm...
Fig. 8.78. Antitachycardia pacing. Top: in "burst" mode, a series of pacing pulses are given at the same cycle length. Subsequent bursts may have a shorter or an adaptive cycle, but the rate does not change within a burst. Bottom: in "ramp" mode, each pulse within a sequence comes at a progressively shorter interval. Terminology varies among manufacturers, with that of Boston Scientific shown in the figure. (Adapted with permission from Boston Scientific Manual.)
or SVT), and reduction in postshock myocardial depression. Conversely, programming the first shock to maximal output may improve first-shock success, reducing the likelihood of repetitive shocks, and may increase the odds of spontaneous termination due to the slightly longer charge time.

Low-energy cardioversion
Many monomorphic VTs are susceptible to low-energy shocks (1–5 J), even if they fail to respond to overdrive pacing. However, low-energy shocks are not pain-free, and they increase the risk that additional shocks will be necessary. Additionally, the vulnerable zone during ventricular tachycardia may extend from one cardiac cycle to the next R wave, so that a low-energy shock may induce VF. Thus, shocks to treat ventricular tachycardia should be programmed based on the defibrillation testing or at maximum output to prevent VF induction.

Interactions of antibradycardia pacing with tachycardia programming
The bradycardia and tachycardia functions of defibrillators can interact, putting constraints on programming options and device function. These vary among manufacturers, but common interactions exist:
• The maximum pacing rate must be 5–10 bpm slower than the slowest VT detection zone. This may become problematic in patients with slow VTs on anti-arrhythmic medications who also have chronotropic insufficiency. The single exception is in ELA devices, which permit the pacing upper rate limit to exceed the VT detection cut-off.
• Algorithms that promote pacing at more rapid rates impair VT detection. This results from the obligatory blanking and refractory periods following paced events, during which spontaneous ventricular activity is not detected. This phenomenon has been best characterized with the rate smoothing algorithm, but occurs with any high rate pacing.
• Most important interactions lead to programming lock-outs or parameter interaction warnings on the programmer, which should generally be heeded.

Atrial defibrillators: detection and therapies
In contrast to dedicated ventricular defibrillators, which seek to diagnose atrial arrhythmias so that therapies can be withheld, atrial defibrillators diagnose atrial arrhythmias to enable delivery of specific atrial therapies. In addition to standard ventricular therapies (antitachycardia pacing, cardioversion, and defibrillation), these devices include atrial prevention and termination therapies (Fig. 8.79 and 8.80). Atrial and ventricular algorithms are often separate, so that a supraventricular arrhythmia with a rate in the VT zone may result in inhibition of ventricular therapy.

Fig. 8.79 Atrial rate stabilization is designed to prevent atrial fibrillation onset by eliminating pauses after premature atrial complexes (PACs). From top to bottom are shown the surface electrocardiogram, intracardiac electrogram, and markers. A PAC is present and sensed (first arrow, circled “AS” on marker channel). To prevent a long pause, two paced beats at gradually prolonging intervals are delivered (circled “AP” markers) until sinus rhythm returns (“AS” following the last “AP”).
by means of SVT-VT detection enhancements, but may not result in atrial therapy delivery by an independently programmable atrial algorithm. Moreover, since atrial arrhythmias are not immediately life-threatening (in contrast to ventricular arrhythmias), atrial therapies can be restricted to certain times of the day or limited in the number of therapies per day to enhance patient acceptance. There is one important dictum to guide optimal programming of atrial therapies: unlike ventricular arrhythmias, which are treated because they are immediately life-threatening, atrial arrhythmias are treated to improve quality of life and reduce symptoms. For this reason, shocks for atrial arrhythmias are generally avoided.

Atrial pacing modestly reduces the risk of developing atrial fibrillation.87,88 Atrial termination and prevention therapies have been shown to reduce arrhythmia burden in subsets of ICD recipients using device-based arrhythmia detection.89 However, their ability to reduce the risk of clinically meaningful endpoints (symptoms, stroke, heart failure, etc.) has not been demonstrated. Importantly, none of the studies of atrial therapies controlled for the frequency of right ventricular apical (RVA) pacing. Indeed, in the majority of studies pacing modes were utilized that promoted frequent RVA pacing. Upcoming studies will better define the role of atrial therapies. At present, they are not widely used in routine practice, but may have a particularly useful role in patients with atrial flutters not readily amenable to catheter ablation, including some incisional flutters.

When device therapy is used to treat atrial arrhythmias, the following general principles guide optimal programming: (i) atrial pacing therapies should be used liberally. Even if efficacy is less than that of shocks, the main treatment goal is improvement in arrhythmia symptoms and quality of life; (ii) shock therapies are generally avoided. When used, they should be programmed to make the first shock work. In the clinically effective range, pain is more closely correlated with the number of shocks than with the shock strength;90; (iii) patient preference is critical for effective programming. Some patients may prefer to self-administer pacing therapies (ATP or burst, or both) for treating atrial arrhythmias, typically with no or only brief delay after onset of arrhythmia. Atrial defibrillation is offered after detailed discussion and the option of receiving a test shock in the hospital. The specific approach used for shock delivery depends on the degree of symptoms experienced during arrhythmia and patient comfort with activator use.

Optimizing programming

Optimal programming lowers the risk of appropriate and inappropriate shocks, minimizes the development of comorbidities such as congestive heart failure and atrial fibrillation, and increases battery longevity and the likelihood of detection of malfunction before it becomes clinically relevant. Patient specific characteristics such as underlying disease and arrhythmia history influence optimal programming; the authors' recommendations are summarized in Table 8.7.
### Table 8.7 Patient-specific optimized programming

<table>
<thead>
<tr>
<th>Disease state</th>
<th>Arrhythmia characteristic</th>
<th>Programming considerations</th>
<th>Rationale</th>
</tr>
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<tbody>
<tr>
<td>Channelopathies</td>
<td>Rapid polymorphic VT/VF</td>
<td>Single detection zone for HR &gt;200 bpm</td>
<td>• Clinical arrhythmia is rapid (so that a high cut-off rate is unlikely to underdetect significant arrhythmias; young patients can achieve rapid heart rates with exercise, increasing the risk of inappropriate detection of rhythms with HR &lt;200 bpm)</td>
</tr>
<tr>
<td></td>
<td>Frequent, nonsustained episodes</td>
<td>Detection enhancements “off”</td>
<td>• Enhancements are generally not effective in VF zone at rapid rates</td>
</tr>
<tr>
<td></td>
<td>Long QT during sinus rhythm</td>
<td>Prolong detection (to 30 of 40 beats has been used in Medtronic devices in one clinical trial; analogous programming for other manufacturers’ ICDs exist)</td>
<td>• Prevent inappropriate charging and shocks</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Avoid ATP</td>
<td>• Role in polymorphic VT/VF not established; possibility of proarrhythmia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Use ATP for atrial arrhythmias</td>
<td>• Prevent inappropriate shocks</td>
</tr>
<tr>
<td></td>
<td></td>
<td>ATP: use 1–2 sequences for HR &lt;250</td>
<td>• Minimize risk for inappropriate detections by not exposing detection algorithms to slower rates; two zones permits increased ATP use in the lower HR zone</td>
</tr>
<tr>
<td>Primary Prevention (CAD or DCM)</td>
<td>Fast VT/VF is often monomorphic, HR &gt;200</td>
<td>Use 2 detection zones, VT cut-off 180–190</td>
<td>• Minimize risk for inappropriate detections by not exposing detection algorithms to slower rates; two zones permits increased ATP use in the lower HR zone</td>
</tr>
<tr>
<td>Secondary Prevention (CAD, DCM)</td>
<td>Monomorphic VT with heart rates 120–200</td>
<td>Use 3 detection zones</td>
<td>• Minimize risk for inappropriate detections by not exposing detection algorithms to slower rates; two zones permits increased ATP use in the lower HR zone</td>
</tr>
<tr>
<td></td>
<td>Fast VT/VF is often monomorphic, HR &gt;200</td>
<td>Program detection enhancements “on;” use dual-chamber enhancements if available</td>
<td>• Decrease risk of inappropriate shock</td>
</tr>
<tr>
<td></td>
<td></td>
<td>ATP: use 1–2 sequences for HR &lt;250</td>
<td>• Reduce risk of inappropriate and appropriate shock</td>
</tr>
<tr>
<td></td>
<td></td>
<td>ATP: use 3 detection zones</td>
<td>• Permit increased detection enhancements and ATP for slower VT and tiered therapies</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Program detection enhancements “on;” use dual-chamber enhancements if available</td>
<td>• Decrease risk of inappropriate shock</td>
</tr>
<tr>
<td>CHF</td>
<td>Bradycardias VT/VF</td>
<td>Avoid RV pacing in non-CRT systems. Use RV pacing avoidance algorithms if available</td>
<td>• Chronic RV apical pacing desynchronizes the ventricles, increasing the risk of CHF</td>
</tr>
<tr>
<td>PAF</td>
<td>Atrial fibrillation and sinus bradycardia</td>
<td>Promote atrial pacing and minimize RV apical pacing</td>
<td>• Increased atrial pacing modestly reduces atrial fibrillation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Atrial prevention and termination algorithms (if available)</td>
<td>• RV apical pacing increases risk of atrial fibrillation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Atrial termination algorithms (ATP, HFB) reduce arrhythmia burden, but clinical significance is uncertain</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>May be particularly useful in patients with atrial flutter, incisional atrial reentrant circuits</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Avoid use in first month (possible lead dislodgment)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Avoid shocks for atrial arrhythmias</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Avoid inappropriate shocks</td>
<td></td>
</tr>
</tbody>
</table>

**Note:** ATP = Anti-Tachycardia Pacing, CHF = Congestive Heart Failure, CAD = Coronary Artery Disease, DCM = Dilated Cardiomyopathy, VT = Ventricular Tachycardia, VF = Ventricular Fibrillation, PAF = Paroxysmal Atrial Fibrillation, ATP = Anti-Tachycardia Pacing, HFB = High-Frequency Burst.
independent and general programming considerations are listed in Table 8.8.

**Cardiac resynchronization devices**

In order for cardiac resynchronization to occur, a sufficient “dose” of therapy must be delivered. The “dose” is a function of the frequency of resynchronization pacing and of the effectiveness of each resynchronized beat. In order to be effective, the left ventricular lead must capture the left ventricle. Programming outputs to ensure capture is discussed earlier in the chapter. The effectiveness of resynchronization is also modified by the programmed AV delay and the offset between RV and LV timing. The impact of these parameters, and their optimization, is reviewed in Chapter 2, “Hemodynamics of Device Therapy,” and briefly discussed below. Lastly, optimized resynchronized pacing pulses must be delivered frequently (ideally > 90%) in order to have a clinical impact. Events that disrupt ventricular tracking and permit intrinsic ventricular

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**Table 8.8 Patient independent and general programming optimization**

<table>
<thead>
<tr>
<th>Programming</th>
<th>Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensing</td>
<td>Ensure appropriate detection enhancement operation</td>
</tr>
<tr>
<td>• Screen for far-field R-wave oversensing and program to eliminate it if present</td>
<td>Avoid double counting and inappropriate detection</td>
</tr>
<tr>
<td>• Screen for T-wave oversensing and program to eliminate it if present</td>
<td>Minimize the risk of inappropriate detection</td>
</tr>
<tr>
<td>Detection</td>
<td>Prevent unnecessary capacitor charging and shocks</td>
</tr>
<tr>
<td>• Use detection enhancements in all patients with intact AV nodal conduction and a VT zone with HR cut-off &lt;200 bpm</td>
<td>Minimize the risk of inappropriate detection</td>
</tr>
<tr>
<td>• Prolong number of intervals to detect or detection time in patients with frequent self-terminating arrhythmias (up to 30 out of 40 in Medtronic VF zone)</td>
<td>Prevent unnecessary capacitor charging and shocks</td>
</tr>
<tr>
<td>Bradycardia therapy</td>
<td>RV apical pacing promotes atrial fibrillation and congestive heart failure</td>
</tr>
<tr>
<td>• Minimize RV pacing in all non-CRT systems</td>
<td>Reduce the risk of AF</td>
</tr>
<tr>
<td>• Promote atrial pacing in patients with sinus node dysfunction</td>
<td>Minimize the risk of inappropriate and appropriate shocks</td>
</tr>
<tr>
<td>Tachycardia therapy</td>
<td>DFT or ULV based shock output not likely to transform VT to VF</td>
</tr>
<tr>
<td>• Program liberal ATP in VT zones &lt;200 bpm and 1–2 ATP sequences in faster detection zones</td>
<td>Maximum output shocks more likely to be effective and lower risk of repetitive shocks; longer charge time may allow spontaneous termination</td>
</tr>
<tr>
<td>• Program first shock strength for VT or VF based on DFT or ULV testing or to maximum output. Program all subsequent shocks at maximum output</td>
<td></td>
</tr>
<tr>
<td>Device function/ surveillance</td>
<td>Some device “malfunction” can be corrected by specific manufacturer recommended programming. Example would include software errors (such as “latching”) eliminated by turning off certain features</td>
</tr>
<tr>
<td>• Check for device specific “recalls” at routine follow-up and reprogram accordingly</td>
<td></td>
</tr>
<tr>
<td>• Enable patient alerts</td>
<td>These generate audible tones or vibratory alerts, advising the patient to seek care (see Chapter 13, “Follow-up”)</td>
</tr>
<tr>
<td>• Enable remote monitoring</td>
<td>Automated monitoring generates web-based or other forms of physician notification when parameters (e.g., lead impedance) are out of range suggesting incipient malfunction (see Chapter 13, “Follow-up”)</td>
</tr>
</tbody>
</table>
conduction, frequent PVCs, and rapidly conducted SVTs reduce the frequency of resynchronization. CRT devices incorporate algorithms designed to overcome these events in order to promote continuous resynchronization. These are discussed below, and in Chapter 13, "Follow-up".

**Algorithms to promote continuous tracking**

Tracking (ventricular pacing following a sensed atrial event) may not occur if the atrial event occurs during a refractory period or if intrinsic conduction is shorter than the programmed AV delay. Algorithms to address both scenarios exist. Since atrial complexes that occur during the PVARP are not tracked, a premature atrial complex that falls in the PVARP and is intrinsically conducted to the ventricles may initiate a succession of non-resynchronized complexes. Algorithms such as Atrial Tracking Recovery™ (Medtronic) and Tracking Preference™ (Boston Scientific) shorten the PVARP to allow tracking of sequential atrial events that fall into the PVARP (Fig. 8.81). Negative AV Hysteresis algorithms (St Jude), discussed in the pacing section, above, shorten the AV interval following sensed ventricular events to promote ventricular stimulation.

**Algorithms to manage PVCs**

Premature ventricular complexes may have detrimental effects in heart failure patients by direct effects and by inhibiting resynchronization. Frequent PVCs themselves produce ventricular dyssynchrony that can lead to cardiomyopathy. Since they can occur early in the cardiac cycle, PVCs may inhibit ventricular pacing, limiting continuous CRT delivery. With algorithms such as Ventricular Sense Response™ (Medtronic) a sensed ventricular event on either ventricular channel triggers an immediate ventricular pace, to promote resynchronization (Fig. 8.82).

In resynchronization devices, algorithms designed to prevent pacemaker-mediated tachycardia are generally avoided. These lead to an extension of the PVARP following a PVC (post PVC-PVARP extension), which may disrupt tracking of the next sinus beat. With intact intrinsic conduction, a sequence of conducted sinus beats may occur, inhibiting biventricular pacing. This

**Fig. 8.81** Algorithm to promote continuous tracking (Atrial Tracking Recovery™, Medtronic, shown). The initial atrial complexes (labeled "1") occur during the PVARP and are not tracked. After eight AR–VS cycles, the algorithm shortens the PVARP to break the pattern (at "2"). The algorithmic intervention continues until AV tracking at programmed SAV resumes (at "3").
is particularly true if both the atrioventricular conduction interval and the PVARP are long.

**Algorithms to manage atrial fibrillation**

Rapidly conducted atrial fibrillation frequently inhibits biventricular pacing. Algorithms such as Ventricular Rate Regulation™ (Boston Scientific) and Conducted AF Response™ (Medtronic) regularize the ventricular response atrial fibrillation by adjusting the pacing rate to minimize RR interval variability and also increase the frequency of ventricular pacing (Fig. 8.83). The increased frequency of ventricular pacing has a minimal effect on the average heart rate. The latter issue is important, since inappropriately rapid pacing itself may depress ventricular systolic function. Resynchronization in patients with chronic atrial fibrillation typically also requires pharmacological or nonpharmacological rate control.

**Device-based optimization for cardiac resynchronization**

Since echo optimization of AV and VV intervals is labor intensive, and since early evidence suggests that the optimal settings for these intervals varies over time, a device-based algorithm has been developed to identify AV and VV intervals that optimize hemodynam-
ics (QuickOpt™, St Jude Medical). The algorithm uses intracardiac electrogram timing between the RV and LV leads and analysis of the atrial electrogram to derive optimal AV and VV intervals. Early, small, acute studies suggest high correlation between electrogram-determined and echocardiographically-determined optimal parameters.92,93

**Conclusion**

Pacemakers and implantable defibrillators have undergone enormous technical advances since their early days. With the introduction of many new features, a large number of programmable parameters have been introduced to enable the caregiver to tailor device function to an individual’s clinical arrhythmia. With a thorough understanding of device operation function, it can indeed be optimized to provide life-prolonging therapy with minimal morbidity.

**References**


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CHAPTER 8  Programming


Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach


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CHAPTER 9

Rate-adaptive Pacing

David L. Hayes, Samuel J. Asirvatham

When rate-adaptive pacing was first introduced it was rapidly embraced by clinicians. In the USA the vast majority of pacemakers currently implanted have rate-adaptive pacing capability.

In the early single-chamber rate-adaptive (AAIR, VVIR) pacemaker era, investigators were quick to demonstrate hemodynamic advantages of rate-adaptive modes, that is, VVIR vs. VVI and AAIR vs. AAI. Similarly, when dual-chamber rate-adaptive pacing was introduced later in the 1980s, literature emerged demonstrating hemodynamic superiority of DDDR over DDD in the chronotropically incompetent patient. Rate-adaptive pacing is similarly beneficial with cardiac resynchronization therapy (CRT) devices, but with unique issues because of left ventricular simulation (see below). Although there are other benefits of rate-adaptive pacing, correcting the chronotropic response remains the most important.

Indications for rate-adaptive pacing

The indications for rate-adaptive pacing are relatively straightforward (also reviewed in Chapter 2). VVIR pacing is indicated primarily for the patient with chronic atrial fibrillation and a slow ventricular response that requires bradycardia support. AAIR, not widely used, is appropriate for the patient with sinus node dysfunction and intact atrioventricular (AV) node conduction. Even though a significant number of patients require permanent pacing for sinus node dysfunction, many clinicians remain uncomfortable with a system that does not provide ventricular pacing support.

Chronotropic incompetence also remains the primary indication for DDDR pacing. However, DDDR can be considered for any patient requiring dual-chamber pacing because it provides clinical flexibility. For example, should atrial fibrillation develop in a patient with a DDDR pacemaker, the pacemaker can be programmed to VVIR. In addition, if the patient has symptoms with traditional DDD upper rate response, i.e., symptomatic 2:1 AV block, optimal programming of sensor response in a DDDR pacemaker will minimize V–V cycle length variation.2

The indications for rate-adaptive sensors will expand in the future as the sensors become desirable not just for rate-adaptive pacing but also for hemodynamic regulation.

Sensors available for rate-adaptive pacing

In an early effort to classify sensors on the basis of their response to physiological variables, Rossi3 divided sensors into five orders (Table 9.1). Despite having been published several decades ago, this classification remains a very reasonable way to consider sensor capability.

Sensors can also be classified as open- or closed-loop (Fig. 9.1). All commercially available sensors are open-loop sensors to some extent, in that the parameter being sensed requires input externally to optimize sensor response, and the sensor is unable to react appropriately to stimuli that do not affect the specific sensor. Conversely, a closed-loop system ideally does not require external input or manipulation because intrinsic feedback to the sensor self-regulates its response.
The ideal closed-loop sensor should closely mimic the normal sinus node and not require external input for programming. To mirror normal sinus node function, the sensor should have an anticipatory increase in heart rate prior to activity, increase the heart rate in proportion to the intensity of activity, gradually allow heart rate to return to normal after cessation of activity, and respond to non-physical exertion (emotion). These characteristics of the ideal sensor are summarized in Table 9.2.

A variety of sensors appropriate for rate-adaptive pacing have been developed and clinically assessed; they are displayed in Fig. 9.2 as end-points of some physiological response. Only a few of these sensors are clinically available. Others have previously been investigated and subsequently abandoned commercially. Even though some sensors may never have been clinically released as single-sensor rate-adaptive pacing systems, some iteration of the sensor technology may eventually be used as part for hemodynamic autoregulation.

Table 9.1 Classification of sensors by response to physiological variables

<table>
<thead>
<tr>
<th>Order</th>
<th>Description</th>
<th>Potential physiological variable</th>
</tr>
</thead>
<tbody>
<tr>
<td>First</td>
<td>A sensor that directly measures oxygen consumption or energy expenditure</td>
<td>Oxygen uptake</td>
</tr>
<tr>
<td>Second</td>
<td>A sensor with a linear relationship to sensors of the first order</td>
<td>Cardiac output, minute ventilation, atrioventricular oxygen difference</td>
</tr>
<tr>
<td>Third</td>
<td>A sensor with a linear relationship to sensors of the second order</td>
<td>Heart rate, stroke volume, mixed oxygen saturation, respiratory rate, tidal volume</td>
</tr>
<tr>
<td>Fourth</td>
<td>A sensor that relies on changes in sympathetic activity and circulating catecholamines</td>
<td>QT interval, right ventricular dP/dt, pre-ejection interval, ventricular depolarization gradient</td>
</tr>
<tr>
<td>Fifth</td>
<td>A sensor that responds to physiological feedback from metabolic activity or receptor reflexes</td>
<td>Central venous pH, central venous temperature, right atrial pressure, mixed venous lactate and bicarbonate levels</td>
</tr>
</tbody>
</table>

Data from Rossi.3
Three varieties of sensors account for most rate-adaptive pacing systems worldwide. Activity sensing and minute ventilation have been the primary rate-adaptive pacing systems in the USA and have also been widely used throughout the world. Stimulus-T, or QT, sensing pacemakers have been used less extensively but remain in use.

**Activity sensors**

*Piezoelectric crystal (vibration sensor)*

Activity-controlled pacing with vibration detection remains the most widely used form of rate adaptation because it is simple, easy to apply clinically, and rapid in onset of rate response (Fig. 9.3). The piezoelectric crystal was bonded to the inside wall of the pulse generator “can.” As the body moved and generated low-frequency vibrations that would be transmitted to the torso, the piezoelectric crystal would be slightly deformed. With the slight deformation the piezoelectric crystal produces a weak electrical current, which is then used as the basis of the algorithm to adjust the pacing rate.

The generated electrical currents from the piezoelectric crystal are “counted” based on the size of the output and whether it is large enough to cross a specified threshold. The number of outputs counted, i.e., the number that will meet criteria to alter the heart rate, is therefore a function of both the “size” of the signal (in turn dependent on the extent of movement) and the sensitivity to which the sensor threshold is programmed (Fig. 9.4).

**Accelerometer (acceleration sensor)**

The main difference between the piezoelectric crystal sensor and the accelerometer relates to the way in which the sensor is mounted within the pacemaker as well as the way in which the signal is utilized by the pacemaker. As opposed to being bonded to the inside of the pulse generator “can,” the accelerometer is suspended from the hybrid circuitry by a cantilever
beam (Fig. 9.5). Movement that is perpendicular to the plane of the sensor generates an electrical signal that is then used to alter the pacing rate. Instead of counting signals above a certain threshold like a piezoelectric crystal, the accelerometer integrates the voltage that arises from the piezoelement. The pacemaker then differentially “weights” deflections of large amplitude, which allows a more proportional rate response to a given level of exertion to be achieved (Fig. 9.6).

The ability to respond to anterior/posterior motion allows accelerometer-based systems to respond more appropriately to specific activities, such as cycling. For example, the typical cyclist may not generate much vibratory sensation above the trunk level, which could result in a rate-adaptive pacemaker that incorporates a piezoelectric crystal having a limited response during the activity. Another advantage of the accelerometer is improved specificity of sensor response, i.e., it results in fewer inappropriate responses. For example, a vibration sensor will usually result in a greater rate increase when walking down stairs than walking up...
stairs because more vibration is generated walking down a flight of stairs.\(^5,6\)

Rhythmic body motion such as that produced by walking or bicycle riding is typically in the range of 1–8 Hz.\(^7\) Non-exercise-related vibrations that arise from such sources as riding in a car or from non-specific skeletal muscle noise are often > 10 Hz. The accelerometer, which limits analysis of signals to the 1–10 Hz range, should be more specific in its response to activity.

There have been variations of activity sensors, including a gravitational sensor able to discriminate changes in vertical gravitational acceleration and a moving magnetic ball that measures electrical signals, but none has had the clinical success of the accelerometer or the piezoelectric crystal.\(^8\)

Despite the potential advantages of an acceleration sensor, and given that the accelerometer has replaced the piezoelectric crystal as the motion sensor of choice, objective evidence of the superiority of the accelerometer is still relatively thin. In an earlier study that compared the ability of acceleration, vibration, gravitational and a little known other movement-related sensor to mimic sinus rhythm, the accelerometer was superior.\(^9\)

Clearly there are some advantages in specific patients or with specific activities, but when compared in a large group of patients, there was no significant difference in quality of life when patients with accelerometer and piezoelectric crystal rate-adaptive pacemakers were compared.\(^10\)

Although accelerometers overcome some of the difficulties with piezoelectric crystal sensors, some problems remain. With prolonged duration activity, the amount of acceleration remains constant, but the physiological demand for a higher heart rate increases. The extent of acceleration may be similar climbing uphill or moving downhill, but with considerably different requirement for increase in heart rate. Strenuous isometric exercise typically results in an increase in sinus rate, but will not be proportionately increased with accelerometer-based rate-adaptive pacing. In addition, neither vibration nor acceleration sensors will respond to non-movement-based exertion such as emotion or fever.

**Minute-ventilation sensors**

Minute volume (respiratory rate times tidal volume) has an excellent correlation with metabolic demand. There is a near linear relationship between minute ventilation and heart rate below approximately 70% of VO\(_2\)max.\(^11\) At higher workloads, minute ventilation increases more rapidly and this has to be taken into account in minute ventilation pacing algorithms to prevent an inappropriately high pacing rate.

In a rate-adaptive pacing system, measurement of minute volume is accomplished by emission of a small charge of known current (1 mA every 15 ms) from the pacemaker and measurement of the resulting voltage at the lead tip\(^12\) (Fig. 9.7). When both current and voltage are known, transthoracic impedance can be measured between the ring electrode and the pacemaker can. Because transthoracic impedance varies with respiration and its amplitude varies with tidal volume, the impedance measurement can be used to determine respira-

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**Fig. 9.7** The pacemaker sends a current (i) between the ring electrode and the pacemaker can. The sensor detects voltage (V) modulations between the tip electrode and the indifferent electrode on the pacemaker header that occur as a result of changes in transthoracic impedance.
tory rate and tidal volume, which in turn can be used to alter pacing rate.

Although the sensor has performed well clinically and the long-term reliability of the minute-volume sensor has been excellent, the initial rate response, i.e., at the onset of exercise, compared with an acceleration sensor, is slower. Rate response at the onset of exercise was more problematic in the early minute ventilation sensor algorithms, but subsequent changes in the sensor have improved the initial rate response.13 Inappropriate activation of a minute ventilation sensor can occur with coughing, abnormal breathing patterns, e.g., Cheyne Stokes breathing, and at times by upper extremity movement. Inappropriate activation of a minute ventilation sensor may also occur when the patient is connected to some electrocardiogram (ECG) monitors that are also capable of documenting ventilatory frequency.14

**Peak endocardial acceleration (PEA)**

In healthy persons, the autonomic nervous system adjusts cardiac output to meet hemodynamic and metabolic requirements. Even in persons with chronotropic insufficiency, the autonomic nervous system controls the performance of the heart through changes in myocardial contractility.

Peak endocardial acceleration (PEA) utilizes a microaccelerometer inside a hermetically-sealed capsule incorporated in the tip of the pacing lead (Fig. 9.8). The microaccelerometer measures the amplitude of mechanical vibrations that are generated by the myocardium during the isovolumetric contraction phase of the cardiac cycle. The signal obtained is directly related to contractility of the myocardium and peak-to-peak values of the signal are measured, i.e., peak endocardial acceleration (Fig. 9.9). An algorithm calculates the pacing rate based on the variation of the peak endocardial signals against a dynamic reference value.15

Multiple studies have shown the sensor to be stable and reliable. In fact, as the lead becomes more fibrosed at the lead/myocardial interface there is even better transmission of myocardial signals, further improving sensor function. In an earlier study following patients...
for approximately 1 year, the PEA sensor was said to reflect variations in sympathetic activity due to physical as well as mental stress.16,17

Given the ability to detect early changes in contractility, the sensor has been investigated in patients with malignant vasodepressor syncope. Given the potential ability to react with a change in pacing rate prior to any systemic symptoms from a fall in heart rate or blood pressure (Fig. 9.10), PEA has been shown in small studies to have potential advantages in this group of patients.18,19

Although developed and successful for rate-adaptive pacing, the potential hemodynamic uses for this sensor may eclipse the rate-adaptive potential (see below).

### Right ventricular impedance-based sensor

Closed loop stimulation measures impedance from the right ventricular unipolar pacing lead (Fig. 9.11). The impedance measurements correlate with $dP/dt_{max}$, which is a surrogate for ventricular contractility and in turn is a reflection of autonomic activity.20

Similar to the PEA sensor, the impedance-based sensor has been specifically evaluated in patients with syncope secondary to vasovagal stimulation. Because contractility changes are detected early and prior to the patient experiencing cardioinhibition, symptoms are better prevented.21

As a reflection of autonomic activity, this sensor has the potential to respond to non-exertional stimuli. In
a study where the patients were given a mental-stress text, e.g., arithmetic challenge, a significant sensor-driven rate increase occurred.22

Peak endocardial acceleration or right ventricular (RV) impedance sensors have the above-mentioned advantages of enabling specific responses to exertion and non-exertional stimuli. However, their widespread utilization at present is primarily limited by the lack of a dependable “effector” arm. Thus, although the enhanced specificity and characteristics with chronotropic response with these sensors is desirable, their ability to detect the early changes of a pathological neurocardiogenic response is limited in utility because of lack of therapy to increase blood pressure. Although early detection of “vasovagal” syncope and increasing the heart rate may offset some symptoms of this process, because of prominent vasodepressor responses in these patients, feedback therapy beyond chronotropic response and increasing the blood pressure is required. These sensors may also be important in future iterations of resynchronization devices to optimize hemodynamic benefit (see below).

**Stimulus–T or QT, sensing pacemaker**

The interval from the onset of a paced QRS complex to the end of the T wave has been used for rate-adaptive pacing for many years. Autonomic activity and heart rate affect this stimulus–T interval23 (Fig. 9.12). Because of this relationship, measurement of the stimulus–T interval, a reflection of sympathetic activity, can be used for rate adaptation.

By definition, the “stimulus–T” sensor requires that a pacing stimulus be present and the “T” wave must be detectable. The pacemaker uses a unipolar paced QT for detection. Because it may be difficult to detect the end of the “T” wave, the algorithm measures the peak negative slope of the T wave (Fig. 9.12). Difficulty detecting and measuring the “Stimulus–T” interval has the potential to adjust the sensor algorithm inappropriately and result in a pacemaker-mediated tachycardia.24

Since the QT interval is shortened both with increased sympathetic tone as well as simply increasing the heart rate, a potential problem with these sensors is a positive feedback tachycardia. Thus, if an increased sympathetic tone shortens the QT interval resulting in the sensor driving a faster heart rate, the faster heart rate itself will shorten the QT interval further, etc. This potential problem has largely been overcome with algorithms currently used with these sensors. PEA and using the pre-ejection interval (see below) do not have this potential difficulty, since the parameters used by these sensors change only with activity/emotion-driven changes in heart rate rather than increasing the heart rate per se.

Overall the QT-sensing rate-adaptive pacing system has been successful clinically and offers the advantage of being able to provide some degree of rate response to non-exertional stimuli such as pain and emotional stress. It may also have advantages for sensor cross-checking in a dual-sensor system (see below).

**Temperature sensors**

Because central venous temperature increases with exercise, it is reasonable to consider this factor as the basis for a physiological sensor. The increase in temperature can be measured by a thermistor contained within the right ventricular portion of the pacing lead.25 At the onset of exercise, core body temperature decreases as cooler peripheral blood is returned to the central circulation.

Temperature-sensing rate-adaptive pacemakers have been available for many years, but never gained widespread acceptance, in part because a special pacing lead that incorporated a thermistor for temperature measurement was required. Also, because of the relatively slow response of central venous temperature, the sensor...
Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach

was somewhat slow to react and delivered a suboptimal rate response at low workloads.

**Other sensors**
Several other rate-adaptive sensors have been used either investigationally or market approved, but available for a relatively brief period.

The pre-ejection interval, the systolic interval from the onset of electrical ventricular depolarization to the onset of ventricular ejection, has been used as the physiological parameter for rate-adaptive pacing (Fig. 9.13). For ventricular pacing, the pre-ejection interval is the interval between a RV pacing stimulus and the onset of contraction determined by an impedance catheter. The pre-ejection interval shortens as exercise workload increases, and this effect can be used as a signal to increase the pacing rate. An increase in heart rate does not appreciably affect the pre-ejection interval, i.e., no significant positive feedback occurs.

Stroke volume, also measured by an impedance catheter in the RV, has been used for rate-adaptive pacing by incorporation of a pacing algorithm that alters the pacing rate to keep the RV stroke volume relatively constant and within physiological values (Fig. 9.13).

Change in RV pressure, dP/dt, has been used for rate-adaptive pacing. This change is measured by a pressure transducer incorporated in the RV portion of the pacing lead. In clinical investigations and in follow-up, the sensor performed well.

Mixed venous oxygen saturation, measured by hemorelectance oximetry, varies with physical activity and changes rapidly with the onset of exercise (Fig. 9.14). For a rate-adaptive pacing system, the oximeter is incorporated in the RV portion of the pacing lead. In earlier investigations there was concern about long-term reliability of the sensor, but more recent investigations suggest that it may be possible to design an O₂ saturation sensor that will have long-term stability and reliability. Proprietary investigations of this sensor are ongoing, and the sensor has potential advantages for hemodynamic management as well as management of comorbidities that may be present in the heart failure patient.

Paced depolarization integral refers to the vector integral of the paced QRS, or ventricular depolarization gradient (VDG). During fixed-rate ventricular pacing, exercise and the effect of circulating catecholamines decrease the VDG. An increase in pacing rate increases the VDG. In a normal heart, therefore, the VDG should remain relatively unchanged during exercise and other forms of stress, representing a closed-loop rate-adaptive pacing system.

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**Fig. 9.13** Waveform obtained from a rate-adaptive pacemaker that utilized a sensor to measure pre-ejection interval (PEI) and stroke volume (SV). PEI is defined as a right heart analog of the pre-ejection period, which also includes a portion of the ejection time. The SV was calculated as the relative difference between end-diastolic and end-systolic volumes.

**Fig. 9.14** Effect of exercise on right ventricular oxygen saturation measured by a permanently implanted rate-adaptive pacing system recording myocardial oxygen consumption in a canine. (Reproduced with permission from St. Jude Medical.)
Dual-sensor rate-adaptive pacing

The overall performance of market-approved, single-sensor, rate-adaptive systems has been excellent, and newer sensors come closer to functioning in a “closed-loop” fashion. However, the perfect sensor would mimic the response of the normal sinus node at all levels of activity and during emotional stress and would be resistant to non-physiological stimuli.

A multisensor rate-adaptive pacing system could improve specificity by having one sensor verify or cross-check the other33,34 (Fig. 9.15). For example, a dual-sensor pacemaker could be designed so that if the first sensor indicated a rate response to a given stimulus, but the second sensor indicated that a rate increase was inappropriate, no rate increase would occur. Both sensors would have to indicate a rate increase before it would be allowed.

Because some sensors perform in a more physiological manner at low levels of exercise and others perform in a more physiological manner at high levels of exercise, a combination of two or more sensors could better simulate the normal sinus node response (Fig. 9.16).

When more than one sensor is available, the programming must remain relatively simple. Although there should be options for choosing one sensor or both, if both sensors are used, the pacemaker must be capable of blending or mixing their responses (Fig. 9.17). At this time, dual-sensor combinations include: accelerometer and minute ventilation, accelerometer and stimulus–T sensing, and impedance monitoring with accelerometer-based motion sensing. Dual-sensor technology is not an option in implantable cardioverter-defibrillators or CRT systems.

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**Fig. 9.15** Diagram demonstrating the logic involved in sensor cross-checking. Sensors must be in agreement on the appropriateness of a rate increase for any change in the paced rate to occur. (Modified and reproduced with permission from Insignia Ultra Tech. Manual, p. 637.)

**Fig. 9.16** Schematic representation of time-dependent interaction of two sensors. The diagram demonstrates sensor-indicated rates from rest to peak exercise for a minute ventilation sensor (MV) and an acceleration sensor (XL). The diagram depicts the potential superior outcome of blending the response of the two sensors.
There are relatively few investigations directly comparing single- and dual-sensor systems. The Sensor and Quality of Life (SQL) study assessed the effect of single-sensor, minute ventilation or accelerometer, vs. dual-sensor programming on quality of life. They demonstrated that pacemaker implantation definitively improved quality of life in this group of patients with sinus node dysfunction or AV block. However, neither single- nor dual-sensor-driven pacing provided additional quality of life improvement for the first 8 months after device implantation.35

In the DUSISLOG study (Dual Sensor vs. Single Sensor comparison using patient activity LOGbook), patients were randomized to a single sensor, either minute ventilation or motion sensor and then reprogrammed after 3 months to the dual-sensor combination of minute ventilation and motion sensing. The investigators concluded that a single sensor achieved satisfactory rate adaptation for most patients. Dual-sensor programming provided additional clinical benefits in selected patients, specifically patients who were said to have “advanced atrial chronotropic disease.”36

There is no question that certain patients will benefit from dual-sensor technology, and when the sensors are carefully optimized they will benefit from the combination of sensors. It is also clear that sensor cross-checking prevents inappropriate false-positive rate response if complementary sensors are used.

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**Fig. 9.17** Example from a patient in whom rate response was suboptimal with a single sensor. The pacemaker was capable of minute ventilation (MV) and acceleration sensing. The sequences were obtained with the patient completing a variety of tasks including: slow walk; brisk walk; walking to and riding up in an elevator; walking down stairs; walking up stairs. In each plot the dark line represents the actual heart rate achieved and the lighter line represents what the rate-adaptive sensors would have done had the patient been dependent on a sensor-indicated rate. (A) Pacemaker programmed so that minute ventilation is the primary sensor, i.e., acceleration programmed in such a way that it is effectively inactive. Note that the sensor-indicated rate is below the patient’s actual rate at the onset of exercise, i.e., the MV sensor is relatively sluggish. Also note that the MV rate is lower than the patient’s actual rate when coming down stairs and walking to and riding on an elevator. (B) Sensors programmed so that acceleration sensing is relatively “sensitive” and MV is programmed so that it is contributing little if anything to rate response. Acceleration sensing provides a rate similar to the patient’s own rate during slow walk, but is suboptimal during brisk walk and walking down and up the stairs. Also note that the acceleration indicated rate is almost the same with walking up and down the stairs. (C) Example with both sensors programmed to complementary thresholds yields a rate response that very closely mimics the patient’s own rate.
Sensor applications for hemodynamic management

Considering the rapidly increasing use of CRT devices for the management of congestive heart failure (CHF) and the prevalence of chronotropic incompetence in the heart failure population, it was predictable that sensors implanted for rate-adaptive purposes would be investigated for their potential to aid in hemodynamic management in patients that required implantable devices.
In a relatively early study, the PEA sensor was assessed for its ability to reflect hemodynamic changes in different pacing modes in a group of patients with biventricular pacing. In a group of 13 patients, the PEA signals indicated improved hemodynamics with biventricular and left ventricular pacing over right ventricular pacing.38

Automatic determination of the AV interval by the PEA sensor in patients with AV block has been shown in several studies to be comparable to AV interval optimized by the accepted technique of Doppler echocardiography.39,40

Monitoring and interpretation of activity and minute ventilation profiles have both been used to predict when patients with CRT devices may be developing early cardiac decompensation.41,42

It is hoped that data from one or more sensors could be incorporated into a system that can "learn" and manage hemodynamics in a CRT system. Such artificial intelligence capable of managing hemodynamics in an implantable device has only been used in simulation to date43,44 (Fig. 9.18).

**Programming**

As sensor technology has matured, programming has become simpler. Contemporary rate-adaptive pacemakers, both single- and dual-sensor systems, have an increasing degree of automaticity which assists in programming and optimization of sensor function.45 However, one basic tenet persists: every patient with a pacemaker programmed to a rate-adaptive pacing mode MUST be functionally assessed, in some way, to determine that the functional response to the sensor is appropriate. Despite autocalibration or autoprogramming of the sensor, there must be a clinical determination that the sensor response is appropriate, or perhaps more importantly, that the sensor response is not inappropriate. At a minimum, sensor histograms should be assessed to determine if the patient’s rate profile seems appropriate (Fig. 9.19). In many patients it is helpful to assess their rate response with exercise, either informal or formal.

Our approach is relatively simple. If the patient, regardless of age, performs at a very high functional aerobic capacity, a standard stress test that pushes the patient to a high level of exertion is reasonable to be certain that the sensor responds appropriately (Fig. 9.20). If a treadmill test is performed, the chronotropic assessment exercise protocol46 should be considered (Fig. 9.21). It allows for a gradual increase in speed and grade and thus mimics more levels of exercise likely to occur during activities of daily living. Also, if formal treadmill exercise is performed and especially if a motion sensor is being evaluated, the patient should be encouraged to

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*Fig. 9.18* Simulation results obtained with an adaptive cardiac resynchronization therapy prototype during which heart rate increases with exercise, dashed line. In the non-adaptive mode, gray line, neither the AV nor VV intervals are automatically adjusted during exercise. In the simulated adaptive mode, black line, both AV and VV intervals are adjusted during exercise, resulting in a 30% increase in cardiac output.44 Reproduced with permission from Blackwell Publishing.

*Fig. 9.19* Schematic of a rate profile demonstrating distribution of rates at rest and low level exertion, activities of daily living (ADL) and higher levels of exertion. (Reproduced with permission from Medtronic, *Adapta Technical Manual*, p. 27.)
Fig. 9.20 Electrocardiographic tracings taken from a patient undergoing formal exercise assessment to observe and optimize sensor response. (A) 1:1 P-synchronous pacing at a rate of 175. (B) Pseudo-Wenckebach which correlated with the immediate onset of fatigue and inability to continue exercise.

Fig. 9.21 Comparison of three exercise protocols: Bruce, Naughton, and CAEP (chronotropic assessment exercise protocol). METS, metabolic equivalents.
walk on the treadmill without holding on, i.e., allowing the arms to swing naturally at the sides. Gripping the treadmill tightly may blunt the sensor’s response.

Many pacemaker recipients do not routinely reach high levels of exertion, and it may be more important to be certain that rate response is appropriate during an exertional range that corresponds to their activities of daily living. For these patients, casual exercise assessment is performed. If the patient is to be dismissed from the hospital in a rate-adaptive pacing mode, the assessment is performed in the hospital the morning after implantation. The rate response is reassessed in the outpatient clinic at approximately 3 months and subsequently depending on the sensor histogram profile. Whether monitored by hospital telemetry or a strip-chart recorder in the pacemaker clinic, the patient is asked to walk at a casual pace in the corridor for approximately 2 min. The rate is assessed during the walk or immediately afterward. Any patient capable of walking at a faster pace is asked to repeat the walk at a brisk pace. We use the values shown in Fig. 9.22 as a guideline for appropriate rates during casual and brisk walks.

Reassessment of sensor response should be considered if the patient has complaints of exertional fatigue or sudden changes in heart rate (Fig. 9.23). Rate histograms can be invaluable in determining whether rate response is appropriate. For example, Fig. 9.23 displays a histogram in which the rates remain in the lowermost “bins,” suggesting that the sensor is not programmed aggressively enough. Conversely, a histogram in which there is a significant amount of time at faster sensor-driven rates may suggest that the sensor is programmed too aggressively, especially if there are associated symptoms (Fig. 9.24).

If rate response is inadequate, whether determined by histograms or by exercise, determine whether the sensor needs to be programmed more sensitively and determine that the sensor is definitely programmed correctly. Terminology for sensor settings is not uniform among manufacturers; be certain that in an attempt to obtain more rate response, the sensor is not inadvertently being made even less sensitive.

**Programmable parameters**

There are elements of programming rate-adaptive parameters that are similar between devices. To program a specific sensor from a specific manufacturer optimally, you should consult the technical manual and contact the manufacturer if you have questions. The reader is
Fig. 9.23 Sensor histogram in a patient where rate profile reflects inadequate chronotropic response. The base rate is programmed to 60 ppm and 96% of the heart rates captured in this histogram fall into the lowest rate bin of 60–75 ppm and only 4% fall into the next rate bin. (Courtesy of and copyrighted by St Jude Medical.)

Fig. 9.24 Sensor histogram in a patient in whom the rate profile is too aggressive and the patient’s rate is primarily in the upper bins. (Courtesy of and copyrighted by St Jude Medical.)
For most activity sensors it will be necessary to program a “threshold” for the sensor (Fig. 9.25). This can be defined as a programmable value for rate-adaptive pacemakers that determines, in part, the level of activity necessary to activate the sensor. Programming the sensor threshold is not consistent across manufacturers (Fig. 9.26).

In addition, for most sensors it is necessary to program a rate-response value (Fig. 9.27). Although the terminology for the rate response factor will vary between manufacturers, the higher the rate response factor the steeper the sensor-indicated rate response (Fig. 9.28).

To define the rate at which the sensor-indicated rate increases and decreases, it is necessary to program a sensor acceleration or reaction time and deceleration or recovery time (Fig. 9.29). These are programmable options for most sensors and determine how rapidly the pacing rate changes in response to increasing or decreasing exertion or response to non-exertional stimuli if these are being sensed.

Many rate-adaptive devices take “lifestyle” into consideration. That is, categorizing the patient by being very sedentary, moderately sedentary, minimally active, moderately active, very active, etc., assists the auto-rate adaptive programming in setting the appropriate ranges.

It must be stressed that regardless of the sophistication of programmable options, the patient’s sensor response must be assessed in some manner.

**Rate-adaptive pacing with cardiac resynchronization devices**

Patients with CHF and who have had CRT devices im-
Fig. 9.27 Diagrammatic representation of the effect of various rate response values. In this example, and terminology may vary between manufacturers, the greater the rate response value the steeper the sensor-indicated rate response. (Reproduced with permission from Boston Scientific Ultra Insignia, p.6.24.)

Fig. 9.28 Schematic depiction of the clinical effect of programming different rate response values. If too aggressive, the patient will achieve faster rates more quickly and perhaps faster than desirable. If not programmed aggressively enough, an appropriately fast rate response will not be achieved. (Reproduced from SJM p 8.5 courtesy of and copyrighted by St Jude Medical.)

Fig. 9.29 Various example of programming the (A) reaction or acceleration times and (B) the recovery or deceleration times of a rate-adaptive sensor. (Reproduced from SJM p 8.5 courtesy of and copyrighted by St Jude Medical.)
planted have specific requirements ideally aided with rate-adaptive pacing. Because of the underlying cardiomyopathy or from the use of negatively chronotropic drugs (β-blockers), chronotropic competence is often severely limited in patients with CHF. In some of these patients, however, even normal (predicted) increases in heart rate with activity may result in functional compromise (provocation of ischemia, etc.). Thus, careful evaluation and programming of rate-adaptive sensing is required in these patients.

V-V timing
Programming and offset between stimulation of the right and left ventricle is an option available in most presently implanted CRT devices. Because of increased capture latency and decreased intraventricular conduction time in the left ventricle compared with the right, often the left ventricular stimulus needs to be delivered earlier than the right ventricular stimulus to obtain optimal ventricular synchrony. Programming this optimization is typically done at rest (implant or follow-up), but both capture latency and intraventricular conduction time change significantly with exercise. Future CRT devices may allow dynamic interventricular optimization using input from the rate-adaptive sensor.

Rate-adaptive atrioventricular timing
With exercise, patients with intact atrioventricular nodal conduction will have accelerated atrioventricular conduction. The premise for CRT benefits is consistent biventricular stimulation with the avoidance of intrinsic conduction or fusion. Rate-adaptive sensor-based AV shortening with exercise may be more effective in promoting biventricular pacing than solely rate-based AV shortening in these patients with abnormal sinus rate increases with exercise. Interatrial conduction varies significantly in patients with CHF as a result of activity. In patients with standard dual-chamber pacemakers, this variation in interatrial conduction is of no significant physiological consequence. With biventricular devices, however, optimization of the atrioventricular interval is greatly dependent on intra-atrial conduction. Optimal left atrial to left ventricular contraction intervals may be quite different from right atrial to right ventricular conduction intervals, and optimal AV intervals with CRT devices need to factor in these differences. In present practice, the detailed programming of these intervals is of little value, since intra-atrial conduction varies so significantly with exercise. Future CRT devices utilizing rate-adaptive sensors may allow differential, dynamic automated programming of AV intervals with exertion.

As with devices for neurocardiogenic syncope, improvements in rate-adaptive sensors (presently utilized and PEA, RV impedance sensors, etc.) are limited in their effectiveness with CRT devices because of limitations in the “effector” arm. That is, even when a need for improved synchrony along with chronotropic response is sensed, changing the pacing vector, site of left ventricular stimulation (multielectrode lead), or differential varying of RA-RV and LA-LV timing is not presently available. When such options to optimize CRT devices dynamically are available, presently available improvements in rate-adaptive sensing will be critical.

References
CHAPTER 9 Rate-adaptive Pacing


36 Padeletti L, Pieragnoli P, Di Biase L et al. Is a dual-sensor
pacemaker appropriate in patients with sino-atrial disease? Results from the DUSISLOG study. PACE 2006; 29:34–40.
CHAPTER 10

Troubleshooting

Paul A. Friedman, Charles D. Swerdlow, David L. Hayes

Pacemaker troubleshooting

Although the increasing sophistication of pacemakers continues to make troubleshooting a more complex endeavor, pacemaker diagnostics and automaticity have become increasingly more sophisticated as well. Knowledge of the device capabilities and the clinical situation of the patient is key to successful troubleshooting. Whether a patient has a clinical episode that suggests system malfunction or is having routine follow-up, evaluation should be performed in an orderly fashion so that potential malfunction is not overlooked. Some instances of device “malfunction” are not malfunctions at all, but rather are the result of an inappropriately programmed device functioning as programmed or unrecognized appropriate function, i.e., pseudomalfunction. In some instances, early component failure is intermittent, and even meticulous evaluation of the system may not initially reveal a problem.

Successful troubleshooting requires a systematic approach (Table 10.1). It should initially be non-invasive and involve careful evaluation of: any symptoms; the pacing indication; available electrocardiographic tracings; the function and radiographic appearance of the lead system; and stored information obtained by telemetry from the pulse generator. Non-invasive diagnosis and correction of any malfunction are always preferable to operative management. Before invasive troubleshooting, one should take advantage of every possible source of assistance, including careful review of the technical manual and contacting the manufacturer for help. If non-invasive evaluation is unrewarding, operative assessment may be necessary.

In order to adopt a systematic approach to troubleshooting, an understanding of the common problems encountered in a device clinic and their causes is necessary.

Table 10.1 Troubleshooting steps

<table>
<thead>
<tr>
<th>Clinical assessment</th>
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<tbody>
<tr>
<td>Indication for pacing</td>
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<tr>
<td>Focused history and physical examination</td>
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<tr>
<td>Review of operative report</td>
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<tr>
<td>Electrocardiography</td>
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<tr>
<td>Rate</td>
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<tr>
<td>Pacing and sensing</td>
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<tr>
<td>QRS axis</td>
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<td>Magnet response</td>
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<tr>
<td>Chest radiograph</td>
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<tr>
<td>Device type and location</td>
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<tr>
<td>Proper contact between lead pins and setscrews</td>
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<tr>
<td>Lead integrity</td>
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<tr>
<td>Lead position</td>
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<tr>
<td>Pacemaker interrogation</td>
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<tr>
<td>Sensing threshold(s)</td>
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<tr>
<td>Pacing threshold(s)</td>
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<tr>
<td>Lead impedance</td>
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<tr>
<td>Battery status</td>
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<tr>
<td>Special features</td>
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<tr>
<td>Histograms</td>
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<tr>
<td>Trend data</td>
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<tr>
<td>Counters</td>
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<tr>
<td>Programming</td>
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<tr>
<td>Review technical manual for other “clues” to perceived malfunction</td>
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<tr>
<td>Contact the manufacturer for assistance</td>
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<tr>
<td>Operative assessment</td>
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<tr>
<td>Appearance of pocket</td>
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<tr>
<td>Assessment of lead connection(s)</td>
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<tr>
<td>Visual inspection of lead(s)</td>
</tr>
<tr>
<td>Electrical assessment of lead(s)</td>
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<tr>
<td>Patency of venous system</td>
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</table>
The most common problems encountered in pacemaker management include:
• Failure to sense
• Failure to capture
• Failure to output
• Change in magnet rate
• Recurrent pre-implant symptoms
• Palpitations/tachyarrhythmias
• Hemodynamic compromise
• Device advisory or recall.

**Clinical assessment**
Knowing the patient (Table 10.2) and taking a careful history are very important in evaluating any pacing system, especially if malfunction is suspected. Clinical assessment should include the following information: the original indication for pacing, whether or not the patient is pacemaker-dependent, activity immediately preceding the clinical event, symptoms experienced by the patient during the event, observations made by witnesses and duration of the event. Symptoms of pacing system malfunction may be subtle and include fatigue, weakness, confusion, neck pulsations or activity intolerance. Some types of pacing system malfunctions may occur totally without symptoms. For example, intermittent failure to capture in the non-pacemaker-dependent patient or undersensing may not be associated with any symptoms and may be discovered only at the time of routine evaluation. During routine follow-up, the patient should be asked about symptoms potentially related to pacemaker complications, such as recurrence of pre-implant symptoms, syncope, near-syncope, palpitations and a slow, fast or irregular pulse. It is important to obtain information from the operative report if possible, including device model, lead models, acute intraoperative pacing and sensing thresholds and impedance values and any difficulty encountered during implantation.

Most devices store information on lead models, acute threshold data, clinical information (e.g., medication regimen), name of institution where the device was implanted and name of the implanting physician in their memory, for retrieval upon interrogation. All manufacturers have toll-free numbers that may be used to obtain implant information (generally kept by the manufacturer of the pulse generator), such as device model and lead models (Table 10.3). The manufacturers can also provide technical information about device and lead performance and assist with electrocardiographic interpretation and troubleshooting.

It is also very useful (but sometimes difficult) to obtain the chest radiograph taken immediately after implantation for comparison with current radiographs (see Chapter 11, Pacemaker and ICD Radiography).

**Identifying the pulse generator**
The first step in troubleshooting is pacemaker interrogation. However, interrogation requires knowing

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**Table 10.2 Knowing the patient and the pacing system**

<table>
<thead>
<tr>
<th>Know the patient</th>
<th>Know the pacemaker</th>
</tr>
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<tbody>
<tr>
<td>Cardiac diagnoses</td>
<td>Manufacturer</td>
</tr>
<tr>
<td>Noncardiac diagnoses</td>
<td>Model number</td>
</tr>
<tr>
<td>Exposure to electromagnetic interference, e.g., workplace, hobbies, medical procedures</td>
<td>Serial number</td>
</tr>
<tr>
<td>Any physical trauma since prior evaluation</td>
<td>Medical advisory or recall that may apply</td>
</tr>
<tr>
<td>Any reprogramming at other institutions since prior evaluation</td>
<td>Previously programmed values</td>
</tr>
<tr>
<td></td>
<td>Previous battery status</td>
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<tr>
<td></td>
<td>Device idiosyncracies</td>
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</tbody>
</table>

**Know the lead or leads**

<table>
<thead>
<tr>
<th>Manufacturer</th>
<th>Model number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serial number</td>
<td>Medical advisory or recall that may apply</td>
</tr>
<tr>
<td>Connector type</td>
<td>Polarity</td>
</tr>
<tr>
<td>Insulation material</td>
<td>Fixation mechanism</td>
</tr>
<tr>
<td>Normal radiographic appearance</td>
<td></td>
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</tbody>
</table>

Modified from Love and Hayes. By permission of WB Saunders Co.

**Table 10.3 Toll-free, 24-h telephone numbers of manufacturers**

<table>
<thead>
<tr>
<th>Manufacturer</th>
<th>Telephone number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biotronik</td>
<td>1–800–547–0394</td>
</tr>
<tr>
<td>Boston Scientific</td>
<td>1–800–CARDIAC</td>
</tr>
<tr>
<td>Sorin/ELA</td>
<td>1–800–352–6466</td>
</tr>
<tr>
<td>Medtronic</td>
<td>1–800–328–2518</td>
</tr>
<tr>
<td>St Jude Medical</td>
<td>1–800–722–3774</td>
</tr>
</tbody>
</table>

*As of September 2007.
the pulse generator manufacturer, because pulse generators only communicate with programmers made by the same manufacturer. Methods to identify the manufacturer include review of the pacing system identification card that all patients should carry, review of medical records that identify the pacemaker manufacturer or radiographic identification.

With a high-quality posteroanterior chest X-ray, it may be possible to see a radiographic code that will identify the manufacturer (Chapter 11, Fig. 11.3). Alternatively, interrogation with available programmers can be attempted. No adverse consequences should occur from attempting to interrogate the device with a non-compatible programmer. In the event that interrogation by multiple programmers is unsuccessful or not possible, i.e., programmers from multiple manufacturers not available, calls can be made to each of the device manufacturers. When calling the manufacturer, ask for the area that can assist with “patient registration.” You will need to provide the patient’s name and possibly the date of birth. If the manufacturer’s products were implanted and appropriately registered, the information should be on record. If the appropriate programmer is not immediately available, the company should be contacted at the number given in Table 10.3 and assistance requested.

Electrocardiographic interpretation and troubleshooting is made significantly easier once the pulse generator is identified, programmer acquired and interrogation completed.

**Electrocardiographic interpretation**

If presented with a paced electrocardiogram and no other information, it is reasonable to approach interpretation with several specific questions:

- Is pacing occurring in the atrium, the ventricle or both?
- Is sensing occurring in the atrium, the ventricle or both?
- Based on the first two questions, is it possible to identify the pacing mode or at least narrow the possible options?
- What are the lower and (if applicable) upper rate limits?
- What other measurable intervals are present? For example, what is the atrioventricular (AV) interval; more specifically, what are the AV, PV or AR intervals?
- Is there any evidence that the programmed rate, if identifiable, has been violated?
- On the electrocardiographic tracing available is there evidence of normal sensing and capture?

Only after extracting as much information regarding what is believed to be “normal” operation should attention be turned to any possible abnormality.

**Lead integrity**

The lead system is the most vulnerable component of the pacing system and the most frequent site of system failure other than expected battery failure (depletion). However, lead technology continues to improve, and many manufacturers now have lead performance data indicating some models with 10-year survival rates (i.e., no evidence of lead failure) > 97%.

Insight regarding etiology of transvenous lead failure, presenting signs of failure and time distribution of failure can be gained from data from a multicenter registry of recognized lead and pulse generator problems. It should be emphasized that this registry includes only leads and pulse generators that have been removed from service; it is not a prospective registry of all implants. The causes of transvenous lead failure in the registry are shown in Fig. 10.1. As seen in Fig. 10.1, insulation defects are the most common cause of failure, followed by conductor and fixation failure, although there is variability depending on lead construction. When the distribution of lead failure by number of years in service is analyzed (Fig. 10.2), the median time to failure for all leads was 7.2 ± 5.2 years, with a "failure" peak in the first year followed by a second peak at approximately year “9.” The clinical signs of lead failure reported by the registry are listed in Table 10.4, and the adverse clinical events (most commonly syncope) and their relationship to the mechanism of failure are shown in Fig. 10.3.

If there is concern or evidence of lead failure, it may be helpful to look at product performance data available from the manufacturer. Most manufacturers make product performance information available on a regular basis. Other sources include the Food and Drug Administration (FDA) website, specifically the MAUDE database (http://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfMAUDE/search.cfm) and another registry, www.pacerandicdregistry.com. This registry includes a limited number of centers that report data, and only pulse generator and lead problems are reported.

The chest radiograph is a valuable component in lead evaluation (see Chapter 11). The lead should be inspected in its entirety, from the contact of the pin with the set-screw to the position of the lead within its cardiac chamber. Unfortunately, deterioration of the lead insulation is not visible on the chest radiograph, and fractures of...
the coil are not always obvious. A frequent site of lead damage if a subclavian implant approach has been utilized is the region between the first rib and the clavicle; this site should be carefully inspected for coil fracture ("subclavian crush syndrome"). The risk of subclavian crush becomes higher if multiple leads are in place. If an older chest radiograph is available for comparison, the presence of gross lead dislodgment can be determined. Abandoned leads in contact with the electrode of an active lead may cause an artifact, which can be interpreted by the pacemaker as a cardiac event and cause inappropriate inhibition. Although findings on the chest radiograph suggestive of lead fracture, dislodgment or poor connection between the lead pin and generator setscrew are helpful in identifying lead problems, absence of such findings does not exclude lead failure. (For detailed information on the radiographic appearance of pacing systems, see Chapter 11.)
Lead evaluation should also include the telemetered lead impedance. For currently available leads, an impedance > 2000Ω indicates a conductor fracture or loose setscrew, and a low impedance (< 200Ω) indicates an insulation defect. (Note: expected impedance is a function of lead design, so that for some leads values > 1500Ω are abnormal. A call to the manufacturer is helpful in determining an acceptable impedance “range” for a given lead model.) Most contemporary pacemakers periodically measure and store impedance values, and create plots or tables summarizing periodic values, facilitating detection of changes in lead function (Fig. 10.4). Even if

![Figure 10.3](image-url) Fig. 10.3 Major adverse clinical events and their relationship to the causes of transvenous lead failure. (Reproduced with permission from Hauser RG, Hayes DL, Kallinen LM et al. Clinical experience with pacemaker pulse generators and transvenous leads: an 8-year prospective multicenter study. Heart Rhythm 2007; 4:154–60, Elsevier)

![Figure 10.4](image-url) Fig. 10.4 Telemetry print-out noting atrial and ventricular lead impedances but also a “trend” of impedance values for both leads. In this example the trend displays information for approximately 11 months and impedance values are stable for both leads.
a measured value is within the “normal” range for that particular lead, a notable change in impedance from previous values should raise suspicion. Many pulse generators have the ability to respond to a sudden change in impedance by automatically reprogramming the pacemaker from bipolar to unipolar pacing and sensing configuration (Fig. 10.5).

Pulse generators
Multicenter registry data provide insight into pulse generator failure mechanisms and associated adverse clinical events. The single most common reason for pulse generator removal from service was expected battery depletion, accounting for 92% of removals (Fig. 10.6). Other reasons included medical advisory or recall, electronic failure, connector failure and unknown cause of failure. The impact of rate responsiveness on observed battery longevity is shown in Fig. 10.7. Figure 10.8 demonstrates major adverse clinical events and their relationship to pulse generator removal.

Clinical troubleshooting
The number of programmable features available in pacemakers continues to increase. Although these options permit individualizing optimal pacing therapy for patients, they can make troubleshooting a complex endeavor. A detailed understanding of the correct function of these devices is necessary to provide comprehensive evaluation; technical manuals and expert representatives from the manufacturer provide important assistance.

Pacing and sensing threshold evaluation
An initial step in troubleshooting is to determine the patient’s native rhythm. This may require turning down the rate of the pacemaker or programming it to a non-tracking ventricular mode or both. The presence, type and time to appearance of a spontaneous rhythm after the pacing rate is lowered should be noted. If the patient is found to have no underlying rhythm (usually defined as absence of a native rhythm with the pacemaker programmed to 30 beats per minute), care should be taken to ensure that the patient does not become asystolic for

![Fig. 10.5] A programmer “screen” with a warning that the lead monitor identified a ventricular lead warning. Such a warning may be a sudden change in lead impedance resulting in automatic switch from bipolar to unipolar pacing configuration.
Fig. 10.6 Reasons for removing a pulse generator from service, i.e., removing or replacing the pulse generator; from a prospective registry of pacemaker and ICD problems. (Reproduced with permission from Hauser RG, Hayes DL, Kallinen LM et al. Clinical experience with pacemaker pulse generators and transvenous leads: an 8-year prospective multicenter study. Heart Rhythm 2007; 4:154–60, Elsevier)

Fig. 10.7 Observed pulse generator battery longevity assessed by those with vs. those without the capability of rate-responsiveness. The percentages along the abscissa represent the proportion of pulse generators that failed in ≤3 years, the definition of premature battery failure in this registry. (Reproduced with permission from Hauser RG, Hayes DL, Kallinen LM et al. Clinical experience with pacemaker pulse generators and transvenous leads: an 8-year prospective multicenter study. Heart Rhythm 2007; 4:154–60, Elsevier)

Fig. 10.8 Major adverse clinical events and their relationship to the causes of pulse generator failure. (Reproduced with permission from Hauser RG, Hayes DL, Kallinen LM et al. Clinical experience with pacemaker pulse generators and transvenous leads: an 8-year prospective multicenter study. Heart Rhythm 2007; 4:154–60, Elsevier)
any significant length of time during the troubleshooting session.

Pacing thresholds should then be evaluated. Most devices allow automated evaluation of pacing thresholds; the output is incrementally decreased until loss of capture occurs, and termination of the test results in immediate pacing at pretest values (Fig. 10.9). Pacing thresholds can always be obtained manually. In pacemaker-dependent patients, testing is safest using a “temporary pacing” or “manual threshold” feature in which

![Chart A](image1)

**Fig. 10.9** Two examples of autothreshold determination by the programmer. Autothreshold determination allows ventricular thresholds to be determined in the pacemaker-dependent patient, with minimal risk of clinically significant asystole. (A) Ventricular capture is maintained to the lowest value of 0.25 V. (B) Ventricular capture is lost at 1.75 V.
CHAPTER 10 Troubleshooting

the pacemaker reverts to the permanently programmed output when contact with the programmer screen is interrupted. The operator should be familiar with the programmer emergency pacing feature should it become necessary to restore nominal pacing outputs quickly. After determination of the pacing threshold(s), the chronically programmed output parameters, i.e., voltage amplitude and pulse width, should be reassessed to be certain that the patient has an adequate safety margin. There are several ways to program output parameters to ensure an adequate safety margin. These are described in detail in Chapter 8, Programming. During the evaluation of pacing thresholds, the presence or absence of ventriculoatrial conduction should be noted, and the patient should be questioned about symptoms of pacemaker syndrome, especially if it is suspected on the basis of the clinical findings.

Threshold values should be compared with those at implantation if possible. The development of exit block, some medications, severe electrolyte or metabolic abnormalities, lead dislodgment and occurrence of new myocardial infarction may affect pacing thresholds (Table 10.5).

Many devices now offer automated periodic assessment of pacing threshold in the ventricle, with adjustment of the pacing output to maintain capture with a safety margin that minimizes battery depletion\(^1\) (Fig. 10.10). In these devices, the output on the ventricular channel may be different at the time of interrogation from that initially programmed because of self-adjustment of the device. Additionally, electrocardiographic monitoring during a periodic threshold check may give the appearance of device malfunction due to variations in pacing outputs during the self-diagnostic test.

The sensing threshold in each chamber should be assessed. Some newer devices automatically measure the native atrial and ventricular electrograms, whereas in older devices the value must be manually measured.

**Table 10.5 Effect of drugs on pacing thresholds**

<table>
<thead>
<tr>
<th>Increase in threshold</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bretylium</td>
</tr>
<tr>
<td>Encainide*</td>
</tr>
<tr>
<td>Flecainide</td>
</tr>
<tr>
<td>Moricizine*</td>
</tr>
<tr>
<td>Procainamide†</td>
</tr>
<tr>
<td>Propafenone</td>
</tr>
<tr>
<td>Sotalol</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Decrease in threshold</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atropine</td>
</tr>
<tr>
<td>Epinephrine</td>
</tr>
<tr>
<td>Isoproterenol</td>
</tr>
<tr>
<td>Corticosteroids</td>
</tr>
</tbody>
</table>

*Off market in the USA.
†At supratherapeutic levels.

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**Fig. 10.10** Electrocardiographic tracing demonstrating proprietary Autocapture threshold determination. In this example, capture is lost at 0.25 V and the output is automatically increased to 0.38 V, which fails to capture, and the device again increases output, to 0.5 V, and confirms capture for two consecutive beats. If one is not familiar with this specific pacemaker function, it may suggest malfunction.
The measured sensitivity should be compared with the programmed value to ensure that the chronically programmed value is adequate.

**Assessing the pacing rate**

An understanding of basic pacemaker timing cycles is mandatory (explained in detail in Chapter 7). Many abnormal-appearing electrocardiograms actually represent normal device function when it is understood how the device is programmed.

To know whether the pacing rate is appropriate, it is first necessary to determine the pacing mode and programmed lower and (if applicable) upper rate limits. Under certain circumstances, the rate may be outside programmed values. The upper rate limit may be overridden by the spontaneous sinus rhythm, atrial or ventricular tachyarrhythmias, or, in rare instances, runaway pacemaker (see below).

Several optional features allow intrinsic rates below the programmed lower rate limit to occur without pacing under specific circumstances. Some devices have a nocturnal or sleep function. During sleep time, the pacemaker allows programming of the lower rate limit to a rate generally 10 or 15 ppm less than that during wake time, replicating the natural circadian sleep–wake cycle and helping to conserve battery life. If it is not known that a "sleep rate" is programmed "on," confusion may arise when the patient’s paced rate decreases at the programmed time.

Hysteresis allows the intrinsic heart rate to decrease to a rate below the lower rate limit before pacing begins at the programmed lower rate (Fig. 10.11). However, if the intrinsic rate slows below the hysteresis rate, then pacing will commence at the lower rate limit (which by definition is faster than the hysteresis escape rate). Thus, hysteresis permits intrinsic rhythms slower than the programmed lower rate limit, but will not result in pacing at rates below the lower rate limit, distinguishing it from "sleep rate" function. Although useful in patients whose intrinsic rate approximates that of the programmed lower rate limit to promote native conduction, hysteresis has been a source of confusion.

**Fig. 10.11** A portion of a 12-lead ECG from a patient with a VVI pacemaker programmed to a pacing rate of 60 ppm, i.e., VV interval of 1000 ms. However, an interval of approximately 1240 ms is explained by a programmed hysteresis rate of 40 ppm, i.e., 1500 ms. This means that if an intrinsic QRS complex is present, the pacemaker will wait for 1500 ms to "time-out" before delivering the first paced artifact. Once pacing has occurred, pacing will continue at the programmed lower rate, i.e., intervals of 1000 ms, unless intrinsic ventricular depolarization occurs and again initiates the hysteresis interval.
for many years. Unless it is realized that hysteresis is programmed “on” and the mechanism of this feature is understood, electrocardiographic tracings are often misinterpreted as “oversensing,” since the cycle length is intermittently longer than the recognized programmed lower rate limit.

Multiple parameters affecting the AV intervals are programmable. Independently programmable paced and sensed AV intervals allow for more consistent mechanical AV activation in patients with interatrial and intra-atrial conduction delay, but can cause confusion when electrocardiographic strips are interpreted. This effect may also cause minor variations in the paced lower and upper rates. Rate-adaptive AV delay is also available in most contemporary dual-chamber pacemakers. Because the rate-adaptive AV interval affects the total atrial refractory period and therefore the achievable upper rate limit, confusion may arise (see Chapter 7).

Another classic source of confusion during assessment of pacing rates and alterations in cycle length is rate smoothing. Rate smoothing avoids abrupt changes in pacing rate, such as those that can occur during a sudden transition to pseudo-Wenckebach or 2:1 upper rate behavior, and may eliminate symptoms associated with sensed dysrhythmic events. Rate smoothing controls sudden changes in pacing rate by monitoring the interval between ventricular events (both paced and sensed) and storing the most recent RR interval in memory (Fig. 10.12). On the basis of this RR interval and the programmed rate-smoothing percentage, the pulse generator sets up two rate-control windows for the next cycle—one for the atrium and one for the ventricle. For example, if the monitored VV interval is 800 ms and 6% rate smoothing is programmed, the algorithm allows the upcoming ventricular rate of the cycle to increase or decrease a maximum of 6%, or ±48 ms (752–848 ms). Rate stabilization algorithms function in an analogous manner to prevent long pauses, and similarly can result in “unexpected” pacing on the surface ECG.

Other algorithms also result in “unexpected” atrial pacing or intrinsic conduction. For example, St Jude’s AF Suppression™ algorithm maintains atrial pacing at a rate slightly faster than the intrinsic rate, and periodically extends the cycle length to reassess the intrinsic rate. Once intrinsic conduction is seen, the rate is again increased to maintain atrial pacing. Thus, surface electrocardiography reveals predominantly paced atrial tachyarrhythmia, there is gradual shortening of the VV cycle length. The cycle length is regulated by rate smoothing and is allowed to change by the programmed smoothing factor; in this example, the smoothing factor is 9%. AS, atrial sensing; V-A, interval from ventricular sensed or paced event to atrial paced event; VP, ventricular pacing.

Fig. 10.12 Rate smoothing can confound electrocardiographic interpretation if one is not familiar with it or aware that it is programmed “on.” In this tracing, normal sinus rhythm is replaced by an atrial tachyarrhythmia. Rather than an abrupt increase in the paced ventricular rate in response to the atrial
rhythms with spontaneously slow, up to two conduct-
ed intrinsic beats, and then more rapid atrial pacing. Medtronic’s atrial pacing preference algorithm may lead to analogous electrocardiographic results.

Most manufacturers have some variation of rate drop response or sudden brady response, designed primarily for patients with neurocardiogenic syncope. This type of feature is triggered when the native heart rate decreases through a programmed rate-drop window, after which the device paces at a faster programmed rate (generally 90–100 ppm) until spontaneous rhythm is detected. Other devices offer some type of search hysteresis, in which the device paces for a programmed number of cycles at an accelerated rate (usually 90–100 pulses/min) when the native rate drops below the lower rate limit. Search hysteresis can lead to misinterpretation of the electrocardiogram if the feature is not well understood or if the health-care professional interpreting the electrocardiogram is unaware that the feature is programmed “on” (Fig. 10.13).

Rate-adaptive sensors can also cause confusion. Some sensors are based on physiologic stimuli, such as thoracic impedance or QT interval, and therefore may drive the rate even when the patient is not physically active (as with activity-based sensors). The sensor-driven rates may therefore cause confusion because the paced rates may seem inappropriate for a given activity (Fig. 10.14). Patients may experience symptoms of fatigue or effort intolerance if the sensor is not programmed aggressively enough and symptoms of palpitations or tachycardia if it is programmed too aggressively (see Chapter 9).

**Diagnostic features**

Most contemporary devices offer sophisticated diagnostic options that can help troubleshoot potential causes of clinical events and aid in optimal programming and detection of potential problems before symptoms develop. Such diagnostic features include the number of mode-switching events, number of high-rate atrial events, number of ventricular high-

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**Fig. 10.13** Sudden onset of pacing at a rate of 100 ppm. This occurs as a result of a sudden bradycardia response, i.e., a pacemaker feature that will pace at a faster rate for a programmed period of time when there is a drop in heart rate that meets preset criteria of the algorithm. The purpose of this feature is to better support the patient’s blood pressure when neurocardiogenic responses result in cardioinhibition, which is often accompanied by vasodepression.
rate episodes, number of ectopic events, percentage of time paced and sensed in all chambers, electrograms and trending of such values as lead impedance.

Pacemakers provide diagnostic interpretation channels. An electrocardiographic recorder from the programmer is applied to the patient, and telemetry is established with the pacemaker. The marker channel feature offers real-time, simultaneous electrocardiographic signals and markers denoting paced and sensed events as well as refractory events occurring in each channel. This feature is especially helpful in attempting to determine whether the device is undersensing or oversensing, or if "functional" sensing abnormalities exist (Fig. 10.15).

In non-pacemaker-dependent patients who have experienced a clinical event suggesting possible pacing system failure which is not identified by a thorough

![Fig. 10.15 Programmer-derived tracing with: top, surface ECG; middle, marker channel; bottom, ventricular electrogram. On the surface ECG there is a VV interval that is longer than any other interval present on the tracing. On the marker channel the pause correlates with multiple ventricular sensed events (VS) and ventricular events that occur in a refractory period (VR). This represents oversensing that was caused by a conductor coil fracture. Although oversensing would be suspected based on the surface tracing alone, the marker channels confirm oversensing and provide diagnostic information as to the mechanism. The sharp, saturated electrograms are consistent with "make-break" contact noise from a fracture site.]

![Fig. 10.14 Inappropriate sensor response is determined from the "Sensor Indicated Rate Histogram." During a casual walk of 1 min 26 s, the patient's heart rate varied from the lowest rate bin to a point near the maximum sensor rate of 110. Sixty percent of the counts were in the 106–108 ppm rate bin. The patient complained of dyspnea during the casual walk. With reprogramming to less aggressive rate-adaptive parameters, the walk was well tolerated.]

<table>
<thead>
<tr>
<th>Bin Number</th>
<th>Range (ppm)</th>
<th>Time</th>
<th>Sample Counts</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>45–53</td>
<td>00:08:14s</td>
<td>9</td>
</tr>
<tr>
<td>2</td>
<td>54–95</td>
<td>00:08:06s</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>96–97</td>
<td>00:08:06s</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>98–99</td>
<td>00:08:17s</td>
<td>11</td>
</tr>
<tr>
<td>5</td>
<td>100–102</td>
<td>00:08:06s</td>
<td>0</td>
</tr>
<tr>
<td>6</td>
<td>102–104</td>
<td>00:08:06s</td>
<td>1</td>
</tr>
<tr>
<td>7</td>
<td>104–106</td>
<td>00:08:06s</td>
<td>0</td>
</tr>
<tr>
<td>8</td>
<td>106–108</td>
<td>00:08:05s</td>
<td>32</td>
</tr>
<tr>
<td>9</td>
<td>108–110</td>
<td>00:08:05s</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td></td>
<td>53</td>
</tr>
</tbody>
</table>
noninvasive evaluation, Holter monitoring or an event recorder may be considered (Fig. 10.16).

One of the most important pieces of information that can be obtained from the pulse generator is the battery voltage. Battery depletion, although expected, is still the most common cause of pacemaker failure. Some devices provide a telemetered numerical battery voltage, which should generally be above 2.4 V in lithium-based batteries, and others give a “gas-gauge” representation of battery status. Another way to determine battery voltage is to assess the magnet rate. All pacemakers have a characteristic magnet rate that changes predictably as the battery voltage decreases. Unfortunately, each manufacturer uses a different magnet rate, necessitating knowledge of or access to that information to ascertain battery status. Assessment of the magnet rate is a key component in transtelephonic pacemaker monitoring. As the battery approaches depletion, most devices reset to a “backup” mode. Backup mode is usually fixed-rate ventricular pacing at maximal output. Most devices also lose telemetry function and programmability when the battery nears imminent failure.

**Unexpected device failure**

Contemporary pacemakers and implantable cardioverter-defibrillators (ICDs) have achieved an extraordinary level of reliability. However, rare random component failures occur. If a component malfunction is suspected, the manufacturer should be asked whether similar problems have been reported. Physicians should report adverse device events via the FDA’s MedWatch program using a simple online form (https://www.accessdata.fda.gov/scripts/medwatch/). Other sources of information also exist. Although there is no comprehensive, mandatory database of pulse generator and lead usage and function at present in the USA, a voluntary database designed to document hardware, lead, pacemaker and ICD failures is accessible online. Because only failures are reported, the database does not provide the incidence with which specific failures occur, but it may alert the caregiver to a potential problem or allow a search to see if others have reported a similar problem. This site, www.pacerandicdregistry.com, can be searched by model, manufacturer or type of failure. This site is proving to be an excellent resource. Practitioners are encouraged to register their own device failures.

**Operative evaluation of pacing systems**

Sometimes the status of a pacing system is impossible to determine non-invasively. If intermittent system failure is strongly suspected clinically and comprehensive non-invasive evaluation has been unrevealing, operative assessment may be required (Table 10.5). Invasive troubleshooting should begin with pulse generator manipulation in the open pocket while

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**Fig. 10.16** Three-channel tracing from an ambulatory monitor that was obtained because the patient had recurrent symptoms after pacemaker implantation. The tracing demonstrates failure to capture and the pacemaker malfunction correlated with symptoms. The patient was found to have developed excessively high pacing thresholds on epicardial pacing leads.
the electrocardiogram is observed for abnormalities in pacing or sensing. After delivery of the generator from the pocket, the connection between the pins and the setscrews should be inspected. The lead or leads should then be disconnected from the pulse generator, and thresholds, current, and impedance should be directly measured by the pacing system analyzer before any further manipulation of the leads. Normally, only minor variation exists between the values obtained by telemetry and those obtained by direct measurement; gross differences suggest lead abnormalities. One of the most important measurements obtained at the time of intraoperative troubleshooting is lead impedance. After the electrical integrity of the lead is assessed, the leads should be carefully dissected away from any fibrous tissue and visually inspected. Any obvious breaks in the insulation or fractures in the coil should be noted, especially in the area under any anchoring or purse-string sutures. Blood in the lead is de facto evidence of a breach in the insulation. Unfortunately, only that portion of the lead that is extravascular can be visually inspected. We believe that any breach of lead integrity warrants lead replacement rather than repair, with rare exceptions. 

One is sometimes left, however, with a clinical situation strongly suggestive of pacing system malfunction and no obvious point of failure. In that case, careful consideration could be given to empirically placing a new lead, especially the ventricular lead in a pacemaker-dependent patient. However, empiric replacement of any portion of the pacing system should be considered only as a last resort.

**Focused troubleshooting**

Now that the broader troubleshooting issues have been discussed, we focus on specific, presenting clinical problems. Most problems can be categorized by electrocardiographic abnormalities:

- Failure to capture
- Failure to output
- Undersensing
- Alteration of pacing rate.

Or by patient symptoms:

- Syncope or near-syncope
- Palpitations
- Fatigue.

Each category is discussed after a differential diagnosis has been provided. Some problems occur more commonly early after implantation (i.e., within the first few weeks post implant), some much later and others completely independent of time. An attempt is made to classify each abnormality by whether it is most likely to occur early, late or at any time after implantation.

**Failure to capture**

The different reasons for failure to capture and the most likely time of appearance for the abnormality are as follows:

- Lead dislodgment Early
- Damage at the electrode–myocardial interface At any time
- Exit block (Fig. 10.16) After the first 4–6 weeks
- Perforation Acute (usually manifest within 48 h)
- Lead fracture Usually late
- Lead insulation failure Usually late
- Loose setscrew (Fig. 10.17) Usually early
- Battery failure (Fig. 10.18) Usually late
- Circuit failure At any time
- Air in pocket (unipolar) Acute
- Pseudomalfunction At any time
- Metabolic or drug effect At any time

A clinical approach to assessment of the most common causes of "failure to capture" is detailed in Table 10.6.

Lead dislodgment usually occurs within the first few weeks after implantation. It may be microdislodgment or macrodislodgment. Macrodislodgment implies that the problem is radiographically evident. Microdislodgment implies that the clinical situation is consistent with dislodgment, but that there is no radiographic evidence that the lead has moved (see Chapter 6, Implant-related Complications).

With lead dislodgment, failure to capture may be intermittent or persistent. It is often, but not always, accompanied by sensing abnormalities (Fig. 10.19). If macrodislodgment is confirmed, the lead should be repositioned. The diagnosis of microdislodgment should be entertained and the lead repositioned only if other causes of failure to capture have been excluded.
In Fig. 10.20, atrial lead dislodgment leads to cross-stimulation. “Cross-stimulation” is defined as stimulation of a cardiac chamber different from the one to which the stimulus is directed. This may result from atrial lead dislodgment into the ventricle or from atrial lead stimulation near the tricuspid valve or in the coronary sinus. Cross-stimulation as a reversal of lead connection could also occur, but is uncommonly reported (Fig. 10.21).

Elevated thresholds may be due to a variety of causes, including lead dislodgment, perforation, loss of lead integrity (e.g., fracture or insulation defect), damage at the electrode–myocardium interface and metabolic, electrolyte or drug changes (Fig. 10.16). Although the etiology of the elevated pacing threshold must be determined and managed, as a temporary measure an attempt should be made to re-establish capture by increasing output parameters.

Damage at the electrode–myocardial interface may occur from several causes. Myocardial infarction, an infiltrative cardiomyopathic process, or localized damage secondary to cardioversion or defibrillation could damage the myocardium at the site of the electrode. With an infiltrative process, the alteration in pacing or sensing threshold may be permanent. Following myocardial infarction, cardioversion or defibrillation, the changes may be transient or permanent.

Altered pacing-sensing thresholds after cardioversion-defibrillation occur if the electrical current is transmitted through the lead and results in a circumscribed burn at the electrode–myocardial interface (Fig. 10.22). The threshold alteration is usually transient, minutes or hours. The risk may be minimized by maximizing the distance between the cardioversion pads and the implanted device.

Exit block, by definition, is manifested as increased thresholds. Exit block is defined as chronically elevated thresholds, presumably due to excessive fibrosis or some other problem at the electrode–myocardial interface. True exit block is uncommon, and the cause is not well understood. Steroid-eluting leads generally, but not always, prevent exit block. The diagnostic problem is trying to differentiate exit block from microdislodg-
Fig. 10.18  (A) Electrocardiographic tracing from a patient with a ventricular pacemaker and recurrent near-syncope. The patient had not had her pacemaker checked in several years. The electrocardiogram reveals intermittent failure to capture, and troubleshooting results were consistent with nearly total battery depletion. (B) The patient was admitted to the hospital, and a subsequent tracing demonstrated complete failure to capture and ventricular rhythm disturbances secondary to the bradycardia. At the time of pulse generator replacement, lead function was normal.
If microdislodgment is the presumed diagnosis, the lead is repositioned and thresholds improve and are maintained long-term, microdislodgment is confirmed as the correct diagnosis. If exit block is the real problem, the thresholds will rise again. If this occurs with steroid-eluting leads, the only option is to program the pacing output to levels that allow consistent pacing and maintain an adequate safety margin. Most contemporary pacemakers can be programmed to maximum outputs of approximately 7.5 V and 1.5 ms. If capture cannot be maintained at these levels, therapeutic options are limited. Repositioning the ventricular lead or placing a new lead in an alternative ventricular site may be successful. Alternatively, coronary sinus pacing may be considered, although the patient should be told that the long-term outcome is less predictable. Epicardial pacing could also be considered. Even though epicardial thresholds are characteristically higher than endocardial thresholds, that may not be the case in the patient with exit block.

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**Table 10.6 Failure to capture**

<table>
<thead>
<tr>
<th>Determine pacing threshold</th>
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<tbody>
<tr>
<td>• Able to obtain consistent capture at higher output—yes or no</td>
</tr>
</tbody>
</table>

**Check impedance**

<p>| |</p>
<table>
<thead>
<tr>
<th></th>
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</thead>
<tbody>
<tr>
<td>• If low—recheck in unipolar configuration</td>
</tr>
<tr>
<td>• If impedance normal in unipolar configuration suggests loss of integrity of the outer insulation</td>
</tr>
<tr>
<td>• If high—recheck in unipolar configuration</td>
</tr>
<tr>
<td>• If impedance normal in unipolar configuration suggests defect in the outer conductor coil</td>
</tr>
<tr>
<td>• Obtain chest X-ray and inspect carefully for conductor coil fracture and connection at the connector block</td>
</tr>
<tr>
<td>• If normal—obtain chest X-ray to look for dislodgment</td>
</tr>
<tr>
<td>• Gross dislodgment—reposition</td>
</tr>
<tr>
<td>• No definite evidence of dislodgment, consider other causes for a rise in threshold</td>
</tr>
<tr>
<td>• Damage at the electrode/myocardial interface</td>
</tr>
<tr>
<td>• Drugs that may raise pacing thresholds</td>
</tr>
<tr>
<td>• Exit block (assumes lead is not acutely placed)</td>
</tr>
</tbody>
</table>

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**Fig. 10.19** Tracing from a patient with the pulse generator programmed to VVI at a rate of 30 ppm. The surface tracing, top, reveals a single paced beat and corresponds to a ventricular pace (VP) marker. (The second tracing from the top represents the atrial electrogram and the bottom tracing represents the ventricular electrogram.) It is important to take advantage of all of the information offered by the pacemaker, but it is just as important to take advantage of information “not provided” by the pulse generator. In this example, all of the ventricular events are labeled on the marker channel with the exception of the third ventricular event. This is because the event was not sensed and therefore not labeled as a VS event. The interval from the paced event backward to the second event equals approximately 2000 ms or 30 ppm.
Fig. 10.20 (A) Electrocardiographic tracing shortly after the implantation of a dual-chamber pacemaker. The tracing confirms atrial and ventricular capture. (B) Electrocardiographic tracing from the same patient at a 4-week follow-up examination. The atrial pacing artifact results in ventricular stimulation, and the ventricular artifact occurs at the same atrioventricular interval but falls after the ventricular depolarization. Chest radiography confirmed that the atrial lead had dislodged into the ventricular lead. Because the intrinsic deflection of the ventricular depolarization was consistently falling within the blanking period, the atrioventricular interval was not disturbed.
on a transvenous lead. Again, long-term outcome cannot be predicted and only long-term observation will determine success.

The use of systemic steroids to treat high outputs may be contemplated. Large doses of systemic steroids usually result in a decrement in pacing thresholds. However, when administration is discontinued, the thresholds generally increase again. Long-term use of steroids is obviously not desirable because of the systemic side effects.

Perforation may cause elevated thresholds (see Chapter 6, “Implant-related Complications”).

Metabolic and drug alterations may also affect thresholds and result in failure to capture.13–22 Drugs that may affect pacing thresholds are listed in Table 10.5.

Two comments should be made about drug therapy. Class IC antiarrhythmic agents are the most likely drugs to affect pacing thresholds. If a pacemaker-dependent patient is placed on a Class IC agent, administration should be done cautiously and the patient’s course followed carefully. This class of drugs may also affect sensing thresholds13–15,23 (Fig. 10.23).

A common misconception is that amiodarone will frequently result in an increase in pacing thresholds.

**Fig. 10.21** Telemetry from a dual-chamber pacemaker; top channel is surface ECG; second channel atrial electrogram; third channel ventricular electrogram and bottom channel is of “markers.” The pacemaker is programmed to the VVI mode during this tracing, but there is consistent atrial pacing and the ventricular EGM channel actually displays the atrial electrogram. The ventricular lead had been connected to the atrial port of the dual-chamber pacemaker and the atrial lead connected to the ventricular port.

**Fig. 10.22** Single-channel electrocardiographic tracing from a patient with a dual-chamber pacemaker programmed to the VVI pacing mode. Immediately after cardioversion, ventricular failure to capture occurs with the first two pacing artifacts and capture with the third. This is compatible with cardioversion-related damage at the electrode–myocardial interface.
Fig. 10.23 Series of electrocardiographic tracings from a patient who received a dual-chamber pacemaker for tachycardia-bradycardia syndrome. The top tracing, obtained on day 1 post implant, reveals dual-chamber pacing with atrial capture and ventricular fusion or pseudofusion. (Without an intrinsic ventricular event, it is impossible to state with certainty whether any degree of fusion exists.) The second tracing, obtained on day 5 post implant, demonstrates a flat line that suggests an artifact. In fact, a monitoring electrode had fallen off, resulting in 21 s of artifactual recording. The third tracing, obtained on day 6 post implant, reveals intermittent ventricular failure to sense and capture. The underlying rhythm appears to be atrial fibrillation. The bottom tracing, obtained on day 7 post implant, suggests intermittent failure to capture and sense in both chambers. Administration of propafenone had been started for treatment of tachyarrhythmias. The drug resulted in significant elevation of both pacing and sensing thresholds.
Amiodarone may increase defibrillation thresholds. Amiodarone can raise pacing thresholds secondary to drug-induced hypothyroidism. In the euthyroid patient, amiodarone rarely if ever causes elevated pacing thresholds.

Most severe metabolic disturbances can affect pacing and sensing thresholds. Hyperkalemia is the most commonly encountered metabolic disturbance to do so. The most common clinical occurrence is in the patient with a pacemaker who is undergoing dialysis. Although programming output or sensing variables may help in the short term, the definitive treatment is to lower the potassium levels and avoid subsequent episodes of hyperkalemia.

Older studies have documented sensing and pacing threshold variations with such everyday activities as sleeping and eating. Although well documented, the threshold variations are minimal. In addition, with the low achievable pacing thresholds and outstanding sensing thresholds achievable with contemporary pacing systems as well as a broad range of programming options, the issue is rarely clinically significant.

Loose setscrew may cause failure to capture. Although this is usually detected in the early postimplantation period, a setscrew that has not been effectively tightened may not work loose for months. The clinical presentation of a loose setscrew depends on the degree of contact between the connector pin and the header. If the header and pin are completely disconnected, complete failure to output occurs because of the circuit interruption. If failure of contact is intermittent, failure to output is also intermittent (Fig. 10.17). Although this is the most common presentation, minimal contact between pin and header may allow transmission of a pacing artifact, but inadequate energy is transmitted to result in capture. Intermittent contact may also lead to oversensing with consequent inhibition of pacing.

Conductor coil fracture may produce failure to capture, failure to output and sensing abnormalities (Fig. 10.24). Although this could theoretically happen...
at any time, it is most often a “late” or chronic complication. In complete fracture, the electrocardiographic findings are persistent. In “make-or-break” fracture, the electrocardiographic abnormalities are intermittent. If the fracture is complete, i.e., the circuit is interrupted, failure to output occurs. If the break is incomplete, i.e., only a portion of the output pulse is transmitted to the heart, failure to capture is seen. Often, both manifestations are present. Escaping current at the “break” may also cause sensing abnormalities.

Although programming a bipolar lead to a unipolar pacing and sensing configuration may restore normal pacing, this “fix” should be considered temporary (Fig. 10.25). Whatever mechanism fractured the outer coil of a coaxial lead could eventually affect the inner coil also.

Insulation break also can produce a variety of clinical manifestations and is most often a “late” complication. Insulation defects may involve the outer insulation or, in a bipolar lead, the insulation between conductors. Sensing abnormalities are the most common electrocardiographic manifestation. These can be diagnosed as a reduction in bipolar lead impedance with normal unipolar impedance in leads at risk for this failure mode.

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Fig. 10.25 (A) Programmer-derived surface electrocardiogram, ventricular bipolar intracardiac electrogram, and markers demonstrating VVI pacing at 70 ppm with failure to capture. Telemetered ventricular impedance was >9999 Ω. (B) Reprogrammed to the unipolar pacing configuration at the same output settings, ventricular capture is now consistent and the lead impedance is within normal range.
Failure to capture and failure to output, intermittent or persistent, may also be seen (Figs 10.26 and 10.27).

As with conductor coil fracture, programming a bipolar lead to unipolar pacing and sensing configuration may restore normal pacing and sensing. If the insulation defect is in a ventricular lead in a pacemaker-dependent patient, even if programming to a unipolar configuration restores normal function, this should be considered a temporary solution and the lead should be replaced.

Battery depletion is an expected late occurrence. Fortunately, battery depletion is almost always a predictable phenomenon, one that is readily detected as part of a regular follow-up program. Some pulse generators have had battery depletion patterns that were unpredictable or earlier than projected. If a pulse generator is known to have unpredictable or sudden battery failure, the device should be prophylactically replaced, following guidelines provided by the manufacturer.

Air in the pocket is a complication that is rarely seen but may result in a pacing failure if the pacemaker is functioning in a unipolar pacing configuration. Since a pacemaker in the unipolar pacing configuration must make tissue contact to “complete the circuit,” air can insulate the pacemaker and prevent tissue contact. Historically, this was of greater potential when one side of the pacemaker was coated and the coated side was placed next to the underlying muscle. This meant that contact had to be maintained with the anterior portion of the pulse generator. Now that the pulse generator is no longer coated, any surface of the device programmed to the unipolar configuration that is in contact with tissue should maintain normal function. Therefore, even if there was air or fluid anterior to the pulse generator that prevented tissue contact, it would still be maintained on the posterior surface.

Circuit or component failure is rare. Contemporary pacemakers have achieved an extraordinary level of reliability. If a circuit or component failure occurs, the clinical manifestation is usually not predictable and almost any electrocardiographic abnormality is possible.

Pseudomalfunctions
Several pseudomalfunctions may suggest failure to capture. Functional failure to capture occurs if a pacing artifact is delivered when the ventricle is functionally refractory. For example, during magnet application and asynchronous pacing, a pacing artifact that occurs early after an intrinsic event will not capture the myocardium (Fig. 10.28).

Isoelectric atrial or ventricular depolarization may suggest failure to capture; that is, a pacing artifact is recorded but there is minimal, if any, electrocardiographic evidence of depolarization (Fig. 10.29). Confirmation can be obtained by a multichannel recording (Fig. 10.30).

Electrical artifact, i.e., non-pacemaker output artifact, may be misinterpreted as being pacemaker artifacts and therefore may suggest failure to capture. Electrical artifact can present in many ways. If there is very regular and very rapid artifact present, one should be suspicious of 60-cycle interference. The first question to ask is whether the patient is symptomatic. If not, then before reacting to the electrocardiographic finding, look for and disable any possible source of electrical interference (Fig. 10.31).

Failure to pace (no output)
- Battery failure
- Circuit failure
- Lead fracture
- Insulation failure
- Oversensing
- Loose setscrew
- Crosstalk
- Unipolar lead with pulse generator programmed to bipolar configuration
- Pseudomalfunction
- Ventricular pacing avoidance algorithm
- Small bipolar pacing artifacts
- Sleep function (reduction in rate under specific circumstances)
- Isoelectric intrinsic rhythm.

A clinical approach to assessment of the most common causes of “failure to output” is detailed in Table 10.7. Battery failure (Fig. 10.18), circuit or component failure, lead fracture, insulation defect and loose setscrew have all been discussed as potential causes of failure to output.

The most common cause of failure to output is oversensing. Oversensing implies that something is sensed other than an intrinsic atrial or ventricular depolarization, and therefore the timing cycle is reset and the pacing output inhibited (Fig. 10.32). As previously noted, oversensing may be a manifestation of lead failure, either conductor coil fracture or insulation defect (Fig. 10.27).

Crosstalk occurs when an atrial pacing output is sensed on the ventricular sensing channel, inhibiting ventricular output (Figs 10.33, 10.34 and 10.35). Mechanisms that contribute to crosstalk include high
Fig. 10.26 Electrocardiographic tracing obtained from a patient with an older ventricular polyurethane pacing lead on advisory. There is intermittent ventricular failure to capture. The finding is relatively subtle. The second and fourth ventricular events are paced. The other ventricular events are intrinsic. The ventricular pacing artifact precedes the intrinsic ventricular events, but fails to capture. Interrogation revealed a lead impedance of <250Ω.

Fig. 10.27 Electrocardiographic tracing obtained from a patient with a pacing lead on advisory due to an unacceptable incidence of insulation failure. In this example, there are two pauses. The intervals are not exact multiples of the paced VV cycle (the distance from the second to third ventricular event). Without marker channel or intracardiac electrograms, it is not possible to say whether this is an example of oversensing or whether there is an undetected paced ventricular artifact with failure to capture. However, with the lead advisory, the possibility of a defective lead is difficult to ignore.
Fig. 10.28 Magnet application in a patient with a VVI pacemaker. When asynchronous pacing artifacts occur early after an intrinsic event, there is no capture. This is considered functional failure to capture because it is a function of the refractory state of the myocardium due to the immediately preceding intrinsic depolarization.

Fig. 10.29 Transtelephonic tracing with magnet application. The first magnet beat is labeled “1.” There is failure to capture on the “3rd” magnet evoked pacing artifact. On the beat that follows, 4, it is difficult to separate a paced QRS from the pacing artifact. Given the accepted variation of pacemaker artifact size when a digital recording system is used, it would be possible to assume that the artifact was a larger variation of the artifact seen in beat 3 and that there was again failure to capture. However, the artifact labeled “4” is followed by a definite “T” wave. If there is repolarization there had to be depolarization. Therefore, capture was normal with the artifact labeled “4.”

Fig. 10.30 Three-channel electrocardiographic tracing from a patient with P-synchronous pacing. If the top tracing is viewed in isolation, it is difficult to tell which chamber is paced and if there is capture. However, a clue in the top recording is that there appears to be ventricular repolarization, i.e., a T wave; therefore, there must have been depolarization, even if the QRS is difficult to identify.
Table 10.7 Failure to pace (output)

Assess multichannel electrocardiogram to determine whether pacing artifacts may be visible in one lead but not another—if artifacts are indeed present, no further evaluation needed

Reprogram the pulse generator to high output settings and to a pacing rate that is unequivocally faster than the patient’s underlying rhythm

Obtain telemetry with markers

- If telemetry verifies that the pacemaker is delivering an output at the programmed rate, the pacing circuit has been interrupted and should be carefully evaluated for circuit interruption:
  - Perform pocket manipulation while monitoring the patient; ability to see intermittent output would suggest a loose connection in the connector block
  - Check lead impedance; if there is circuit interruption the lead impedance will be very high
  - Carefully inspect connector block radiographically
  - Carefully inspect the entire length of the lead radiographically

Invasive troubleshooting will probably be necessary at this point

- If no output is seen on telemetry, is there telemetric evidence of other sensed events that may be inhibiting output? If there is evidence of sensed events that do not correlate with any intrinsic cardiac activity:
  - Reassess the lead carefully looking for an intermittent loss of integrity, either insulation or conductor coil, that could be the source of leaking current sensed as activity and inhibiting output
  - Look for any sources of electromagnetic interference. Remove any potential sources from the environment and recheck telemetry
atrial output, ventricular sensitivity programmed to a very sensitive value, and positioning of atrial and ventricular leads in close proximity.

In an effort to prevent crosstalk, pacemakers incorporate a ventricular blanking period (see Chapter 7, Timing Cycles). The blanking period is the initial
Fig. 10.34 Three-channel tracing from an ambulatory monitor. The tracing begins with AV sequential pacing with a short AV interval, approximately 100 ms, which probably represents ventricular safety pacing, although this cannot be proven without more diagnostic information and programming information. This is followed by an intrinsic atrial depolarization with ventricular tracking. The next event is an atrial paced event without a subsequent ventricular event, i.e., ventricular output inhibition. This is again followed by two cycles of AV sequential pacing, again with a relatively short AV interval. The paced AA interval is consistent between the final two intervals on the tracing. The ventricular failure to output could represent some event that was oversensed on the ventricular sensing channel. However, the consistency of the AA interval and the lack of any artifact on any of the three channels would suggest that oversensing of external noise or an isoelectric event is less likely and crosstalk more likely. If the other AV intervals reflect safety pacing then it is possible that whatever is being sensed as crosstalk during these cycles is instead being sensed beyond the crosstalk sensing window and inhibiting output.

Fig. 10.35 Single-lead tracing from a patient with a dual-chamber pacemaker programmed to the DDI mode and a rate of 86 ppm, AVI 165 ms and a ventricular blanking period of 13 ms. The tracing demonstrates a ventricular rate of approximately 104 ppm, RR interval = 575 ms. If the programmed rate is supposed to be 697 ms (86 ppm), the VA interval should be 697 ms – 165 ms [programmed AVI] = 532 ms. However, the effective rate is at a cycle length of 575 ms. 575 ms – the calculated VA interval of 532 ms = AVI of 43 ms. This means that there was sensing of ventricular activity at 43 ms after the atrial output which terminated the AVI and initiated the VA interval. This occurred because there was consistent sensing of the atrial output on the ventricular sensing channel, i.e., crosstalk, but the tracing appears relatively normal because the patient has consistent, albeit prolonged, AV conduction. The abnormality is only clear once the programmed parameters are known.
portion of the AV interval. During this period, decay of the atrial pacing output is maximal and the atrial output has the greatest potential for being sensed on the ventricular channel. If something is sensed immediately after the blanking period, it is not possible to distinguish between crosstalk and an intrinsic event. As a safety measure, dual-chamber pacemakers deliver a ventricular pacing artifact at a foreshortened AV interval if something is sensed in the interval after the blanking period. This portion of the AV interval has been dubbed the “crosstalk sensing window.” If something is sensed in the “crosstalk sensing window,” ventricular safety pacing results in effective ventricular capture at a short AV interval, usually 100–110 ms, and prevents ventricular asystole. If an intrinsic ventricular event is sensed in the crosstalk sensing window, e.g., a premature ventricular contraction, the ventricular safety pacing artifact is delivered within the intrinsic event or shortly after and not during ventricular repolarization (T wave). The ventricle would be re-

Fig. 10.36 (A) Electrocardiographic tracing from a pediatric patient with a dual-chamber pacemaker after surgical correction of a congenital cardiac anomaly. The atrial lead was programmed to excessively high outputs. Every other ventricular event is paced at an abbreviated atrioventricular interval consistent with ventricular safety pacing. We were unable to explain the bigeminal occurrence of the safety pacing. (B) Tracing obtained after reprogramming of the atrial output to values that allowed for an adequate safety margin but no longer resulted in ventricular safety pacing.
fractory and the pacing artifact would not depolarize the ventricle, i.e., functional failure to capture.

When ventricular events are sensed on the atrial sensing channel and reset the timing cycle, the most appropriate description for this abnormality is far-field sensing (Fig. 10.37). The longer intervals are equal to the AR or AV interval plus the programmed AA interval; for example, if the AR interval is 200 ms and the programmed lower rate limit is 60 ppm, or 1000 ms, the interval lengthened as a result of far-field sensing is 200 plus 1000, or 1200 ms. This effect can usually be eliminated by lengthening the atrial refractory period so that the intrinsic ventricular event is ignored or by programming the atrial channel to a less sensitive value (Fig. 10.38). The former may reduce the pacemaker’s upper rate limit, whereas the latter may result in failure to mode switch during atrial fibrillation.

Oversensing can be caused by many things, which can be classified as:

- Biological sources of interference, e.g., retrograde P wave, T-wave myopotentials
- Paced ventricular afterdepolarization
- Non-biological sources of electromagnetic interference (see Chapter 12).

If myopotential inhibition is suspected, a series of maneuvers should be performed in an effort to document the cause (Fig. 10.39). In our pacemaker clinic, a series of isometric maneuvers is accomplished while the electrocardiogram is monitored. These include:

- Hands clasped, pulling against each other
- Palms of hands together, pushing against each other
- Reaching with right arm across left shoulder
- Reaching with left arm across right shoulder
- Pocket manipulation (although not specifically to bring out myopotential inhibition, but instead to assess for integrity of the lead or leads and integrity of the lead-connector block connection, this procedure is done in concert with the other maneuvers).

If maneuvers induce myopotential inhibition, the programmed sensitivity is made less sensitive and the maneuvers are repeated in an effort to find a sensitivity value at which significant myopotential inhibition no

![Fig. 10.37 Electrocardiographic example obtained from a patient with an AAI pacemaker. The programmed AA cycle length is 95 ppm, 630 ms. However, some AR intervals are >630 ms. This occurs because there is far-field sensing; that is, the ventricular event is sensed on the atrial sensing channel and resets the timing cycle. This is verified on the simultaneous marker channel. Three ventricular events—first, third, and fourth—are sensed as atrial events. AP, atrial paced event; AR, atrial event occurring in the refractory period; AS, atrial sensed event.](image-url)
Fig. 10.38 Refractory. (A) Programmer-derived tracing that demonstrates sensor-driven atrial pacing (P-Sr) and intermittent sensed (S) events that follow the sensor-driven atrial event. The pacemaker was programmed AAIR at a rate of 75ppm; atrial refractory period of 280ms. (B) The arrhythmia logbook had recorded very frequent atrial tachycardias. Although this represents far-field sensing, the far-field events fall within the programmed ARP and therefore do not alter the pacing rate. However, for purposes of tachycardia detection they are counted and the double counting results in the arrhythmia logbook recording this as a tachycardia.
longer occurs but sensing of intracardiac events is still intact (Fig. 10.40).

When an interval longer than the programmed lower rate is observed, the point of sensing can be determined by measuring backward from the pacing artifact that terminates the longer interval (Fig. 10.41). For example, if a VVI pacemaker is programmed to 60 ppm, 1000 ms, and an interval of 1500 ms is observed, measuring back 1000 ms from the pacing artifact that ends the 1500-ms interval marks the point of sensing, i.e., the point at which the timing cycle is reset.

An incompatible lead-header combination is a clinical possibility, but is rarely seen. Lead design and compatibility are discussed in Chapter 4. Any incompatibility of the lead and header should be readily apparent at the time they are connected. To be certain that an adequate connection has been established at the time of implantation if pacing output is not observed, a magnet should be applied to confirm output and capture and/or device interrogation should be performed and thresholds documented via the programmer.

With international standard (IS)-1 lead-header designs, unipolar and bipolar leads are of the same dimensions. If a unipolar IS-1 lead is connected to a bipolar pacemaker that is programmed to a bipolar configuration, no pacing will occur. Most contemporary pacemakers detect the incompatibility and prevent the programming combination, allowing the pacemaker to be programmed only to a unipolar configuration.

Pseudomalfunctions may also suggest failure to output. Small bipolar pacing artifacts may not be visible on the electrocardiogram and raise the question of failure to output or of oversensing.

Digital recording systems may also give the appearance of failure to output. Digital recording systems, the type of electrocardiographic recording system used by most hospitals and offices today, artificially create the pacing artifact (Fig. 10.42). As a function of the system, pacing artifacts may not always be seen. A clue to this abnormality is that the ventricular depolarizations with and without pacing artifacts are of the same morphology. In Fig. 10.43 this pseudomalfunction is associated with true failure to capture in a patient with exit block. In this situation, another clue is that all the pauses are a multiple of the programmed lower rate.

Ventricular avoidance pacing algorithms are designed to allow the patient’s intrinsic ventricular conduction to dominate and minimize ventricular pacing. Multiple algorithms are available to achieve this outcome (see Chapter 8, “Programming”). With some algorithms the ventricular output may be inhibited in an attempt...
Fig. 10.40 Series of electrocardiographic tracings obtained during provocative maneuvers to induce myopotential inhibition in a patient with a pacemaker programmed to the VVI mode. There is significant inhibition at programming to 1 mV (A) sensitivity. Inhibition decreases at 2.0 mV (B) and is absent at 4.0 mV (C).

Fig. 10.41 Electrocardiographic tracing from a patient with a VVI pacemaker. The programmed rate of the pacemaker is 70 ppm, 857 ms, but there are longer intervals. Measuring 857 ms backward from the ventricular pacing artifact that ends the longer intervals determines that the point of sensing is either a retrograde P wave or a T wave. Without electrograms or a diagnostic interpretation channel, one cannot be certain which event was oversensed. (From Hayes DL, Zipes DP. Cardiac pacemakers and cardioverter-defibrillators. In Braunwald E, Zipes DP, Libby P, eds. Heart disease: a textbook of cardiovascular medicine, 6th edn. Philadelphia: WB Saunders Co. By permission of the publisher.)
Fig. 10.42 Part of a 12-lead ECG from a patient with a dual-chamber pacemaker. The paced QRS morphology is the same for all beats in each of the three channels depicted. However, there is no discernible pacing artifact preceding the third QRS complex. This complex is indeed paced, the patient is completely pacemaker dependent, but due to the digital recording system, the pacemaker artifacts are not always reliably seen. This may be a source of confusion when interpreting the paced electrocardiogram.

Fig. 10.43 Electrocardiographic tracing from a patient with a pacemaker programmed to the VVIR pacing mode. The lower rate of the pacemaker is programmed to 60 ppm. The tracing was obtained urgently in the emergency department and demonstrates intermittent failure to capture and a long pause without evidence of pacing artifacts, a suggestion of oversensing (pacemaker telemetry was not available at the time.) Careful inspection of the second ventricular depolarization reveals the lack of a pacing artifact. This patient was pacemaker-dependent, and all ventricular activity was paced. Pacing artifacts occur during the interval that appears to be a pause without any pacing activity. This finding was proven by intracardiac electrograms. It can also be suspected from the surface electrocardiogram, because the pause is an even multiple of the programmed lower rate interval. If this were oversensing, a pause that was an exact multiple of the pacing rate would be unlikely. The inability to detect pacing artifacts is not uncommon with digital recording systems.
to allow intrinsic conduction to occur. This gives the appearance of intermittent failure to output and may be misinterpreted as pacemaker malfunction if the programmed settings are not known (Figs 10.44, 10.45).

Although not a pseudomalfuction, a common error is operator misdiagnosis of oversensing because a near-isoelectric P or R wave is overlooked on a surface ECG lead. If an event is truly isoelectric, an intracardiac electrogram or a diagnostic interpretation channel is necessary to make the diagnosis. Figures 10.46, 10.47 and 10.48 show examples of near-isoelectric events. A basic tenet to remember in troubleshooting is that even without a recognizable depolarization, a subsequent repolarization, i.e., a T wave, affirms a preceding depolarization.

**Undersensing**
- Change in intrinsic complex, e.g., bundle branch block, ventricular fibrillation, ventricular tachycardia, atrial fibrillation
- Myocardial infarction
- Lead dislodgment or poor positioning (Figs 10.49, 10.50 and 10.51)
- Lead insulation failure
- Magnet application
- Battery depletion
- Pulse generator component failure or header abnormality (Fig. 10.52)
- Metabolic or drug effect
- Functional undersensing.

A clinical approach to assessment of the most common causes of “undersensing” is detailed in Table 10.8.

Any event that results in an intrinsic complex that differs from the intrinsic complex that was present and measured at the time of pacemaker implantation may cause undersensing. For example, a premature ventricular contraction, which may actually appear larger than the normally conducted ventricular event on the surface electrocardiogram, may not be sensed (Fig. 10.53). Making the ventricular sensing channel more sensitive may allow normal sensing of premature ventricular contractions. However, if the undersensing is only intermittent, the premature ventricular contractions occur rarely, and the normally conducted beats are appropriately sensed, it may not be necessary to take any additional action.

Undersensing in either chamber is theoretically troublesome because of the potential for competitive pacing. Specifically, the concern is one of pacing in a vulnerable portion of the cardiac cycle, i.e., during repo-

*Fig. 10.44 Portion of a 12-lead ECG where there is a single episode of ventricular failure to output after a paced atrial event. This is an example of Managed Ventricular Pacing (MVP), a ventricular pacing avoidance algorithm. The pacemaker performs periodic one-cycle checks for AV conduction and the opportunity to resume AAIR or AAI therapy.*
Fig. 10.45 Three-channel recording from an ambulatory monitor. The tracing begins with atrial pacing and intrinsic ventricular conduction with a narrow-complex QRS. This is followed by atrial pacing and capture and no subsequent ventricular pacing output. The next event seen is an atrial pacing artifact that occurs immediately before a wide-complex QRS escape. (The atrial artifact gives the appearance of a pseudo-pseudofusion event.) The initial wide-complex event is followed by another atrial pacing artifact and yet another which is immediately followed by another wide-QRS escape complex. The last two RR cycles occur at a shorter cycle length and there is presumed AV nodal conduction from the atrial paced artifacts, but with a continued wide QRS morphology. This is an example of managed ventricular pacing (MVP), one of the ventricular pacing avoidance algorithms. With this algorithm, if two of the four most recent non-refractory A-A intervals are missing a ventricular event, the pacemaker will identify this as a persistent loss of AV conduction and switch to the DDDR or DDD mode. However, in this example, there are only single A-A intervals missing a ventricular event, so the switch to DDD mode does not occur. This is normal function.

Fig. 10.46 Three-channel recording from a patient with a DDD pacemaker. The top tracing would suggest oversensing with failure to output unless the other ECG leads were available for comparison. The tracing also demonstrates varying degrees of fusion beats. (Tracing courtesy of Dr Seymour Furman.)
Pacing on a T wave could initiate ventricular fibrillation or ventricular tachycardia, and pacing during atrial repolarization could initiate atrial fibrillation. Both have been documented, but both are uncommon. This conclusion is supported by the fact that although magnet application results in competitive pacing, as a routine part of transtelephonic follow-up it is rarely identified as inducing a tachyarrhythmia.

As noted above, any event that damages the myocardium at the electrode–myocardial interface could alter sensing thresholds. Undersensing may be a manifestation of conductor coil fracture and insulation break. In fact, sensing abnormalities are the most common electrocardiographic manifestation of loss of insulation integrity.

Also noted previously, metabolic disturbances and drugs can affect sensing thresholds. Class IC antiarrhythmic agents are the most likely to alter sensing thresholds, and hyperkalemia is the most common metabolic disturbance to result in undersensing.

If the pacemaker battery reaches a very low voltage level, undersensing may occur.
Fig. 10.49 Tracing from an ambulatory monitor shows intermittent atrial failure to sense. This is best seen at the fifth ventricular complex. A P wave precedes the intrinsic ventricular event, but this is not sensed, and an atrial pacing artifact occurs immediately before the intrinsic ventricular complex. The ventricular complex is sensed in the crosstalk sensing window. As a result, the ventricular pacing artifact is delivered early after the intrinsic ventricular event, i.e., ventricular safety pacing.

Fig. 10.50 Telemetry with surface electrocardiogram, top, atrial electrogram, middle, and ventricular electrogram, bottom. Markers are present on the ventricular channel. There are six ventricular events but only three with “marker” annotation. The first ventricular event is not sensed and not annotated. The second ventricular event is labeled as a paced ventricular (V) event; the third is an intrinsic ventricular event (R); the fourth is not sensed and is followed approximately 360 ms later by a paced ventricular event (V); and the final event is not sensed.
Fig. 10.51 Two-channel rhythm strip from a patient with a pacemaker programmed to the VVI mode with AutoCapture. There is intermittent ventricular undersensing. The (*) note ventricular events that are not sensed and followed by a pacing artifact. Because there is failure to capture, albeit functional failure to capture, the AutoCapture mechanism follows this with a second pacing artifact delivered at higher output. On one occasion (**) the second output is distant enough from the intrinsic ventricular depolarization that the myocardium is no longer refractory and paced depolarization occurs. On the others (arrowhead) there is functional failure to capture on the second output because the myocardium is still refractory.

Fig. 10.52 (A) Electrocardiographic tracing from a patient with a dual-chamber pacemaker and intermittent atrial failure to sense. Despite reprogramming, the intermittent atrial undersensing persisted. (B) Side view of pulse generator and (C) top view. At the time of pulse generator replacement, inspection revealed an area of corrosion at the site of one of the atrial grommets. (D) After pulse generator replacement, no further atrial undersensing was noted. Normal P-wave tracking is noted in this tracing.
As noted below, the first programming step used to correct undersensing is often to make the sensing channel more sensitive, i.e., a smaller number, although uncommon exceptions occur.

Functional undersensing occurs when an intrinsic event falls within a blanking period or refractory period and is not sensed as a function of the pacemaker programming. This can cause confusion in electrocardiographic interpretation unless electrograms or a marker channel is available. Figure 10.54 demonstrates functional undersensing as a result of extension of the postventricular atrial refractory period. Because the postventricular atrial refractory period is extended in consecutive cycles, the P waves are consecutively within this period and therefore not tracked. The appearance is of true undersensing, but function is normal.

When undersensing occurs (Fig. 10.55), the approach should be as follows:

- Determine the cause of the undersensing.
- Assess the amplitude of the undersensed event by telemetered electrograms, if available. Compare the measured signal amplitude with the programmed sensitivity.
- If possible, correct whatever change has resulted in undersensing.
- If the cause of the undersensing cannot be corrected or if immediate resolution is required, reprogram the sensitivity to a more sensitive value.

**Table 10.8** Failure to sense

- Check sensing threshold; automatic sensing threshold or manual
- If possible, reprogram sensitivity to a value more sensitive than the measured value
- If unable to program to a sensitivity value that will allow consistent sensing and/or if there is concern that the sensitivity value necessary is so sensitive that oversensing may be a concern, recheck in the unipolar sensing configuration
- If unipolar sensing thresholds are improved, be suspicious of a loss of lead integrity, especially an insulation defect
- Check impedance value
- Depending on whether the sensing abnormality is early or late after implant, work through the differential diagnoses

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**Fig. 10.53** Electrocardiographic tracing in which a single premature ventricular contraction is not sensed.
Alteration in programmed pacing rate

- Circuit failure
- Battery failure
- Magnet application
- Hysteresis
- Crosstalk
- Undocumented reprogramming
- Oversensing
- Runaway pacemaker
- Malfunction of electrocardiographic recording equipment; alteration in paper speed.

Undocumented reprogramming is probably the most common cause of an alteration in the programmed pacing rate; that is, another healthcare professional has reprogrammed the pacing rate and failed to document the change. To avoid this, a system should be in place whereby any reprogramming requires that the pacemaker be interrogated, the programmed values stored and the programming change recorded in the patient’s medical record.

Hysteresis, crosstalk, far-field sensing and oversensing alter pacing rate, as previously described. If hys-
Hysteresis is the cause of the altered rate, obviously no action need be taken, since the hysteresis is presumably desirable. If oversensing of any type is causing the altered pacing rate, steps should be taken to correct or remove the source that is being oversensed (see above).

Circuit or component failure could alter the programmed rate in an unpredictable fashion. Runaway pacemaker is a manifestation of a component failure or a software-based programming error that results in pacing at dangerously fast rates, in excess of 1000 ppm. It is most likely to occur if a pacemaker is in the field of therapeutic radiation.\textsuperscript{35,36} Therapeutic radiation can cause failure of the complementary oxide semiconductor. The failure is unpredictable by both time of exposure and total radiation. Runaway pacemaker constitutes an emergency. If the patient is hemodynamically compromised, the pacemaker must be urgently disconnected. If time allows, the pacing lead can be properly released from the pacemaker. If hemodynamic failure does not allow the extra time required to properly release the lead, the lead should be transected and temporary pacing should be available if necessary.

Confusion may arise if there is malfunction of the electrocardiographic recording equipment; for example, the paper sticks or the speed is not constant. This defect can give the appearance that the pacing rate is different from the programmed rate or erratic. The clinical approach to an alteration in pacing rate depends entirely on the cause.

\textbf{New symptoms after pacemaker implantation}
As initially discussed, some of the most frequently encountered new complaints after pacemaker placement are recurrent syncope or presyncope, fatigue, and palpitations. Several pacemaker-specific causes should be considered in the paced patient complaining of these symptoms.
- Pacemaker syndrome
- Failure to capture
- Reversion to backup mode
- Inappropriate programmed rate or sensor
- Symptomatic upper rate response
- Primary myocardial abnormality or ventricular rhythm disturbance.

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{Fig_10.55}
\caption{Electrocardiographic tracing demonstrating VVI pacing with intermittent ventricular failure to sense.}
\end{figure}
Pacemaker syndrome should be suspected if the paced patient complains of general malaise and fatigue. This diagnosis is discussed in Chapter 2, “Hemodynamics of Device Therapy.” Symptoms may include:

- General malaise and fatigue
- Chest discomfort
- Cough
- Symptomatic cannon A waves
- Presyncope or syncope
- Confusion
- Dyspnea on exertion.

As part of the troubleshooting process, blood pressure readings should be obtained with and without pacing if the patient is not pacemaker-dependent. Blood pressure differences may be even more dramatic if paced and non-paced pressures are checked in the supine and upright positions. It must be remembered that pacemaker syndrome can occur with any pacing mode if AV synchrony is uncoupled.

Failure to capture or output may result in relative bradycardia and symptoms compatible with low cardiac output. Failure to restore rate response adequately, that is, persistent chronotropic incompetence, may also be accompanied by symptoms compatible with low cardiac output.

Exposure of the pacemaker to electromagnetic interference could potentially cause reversion to a backup mode. Because most back-up pacing modes are non-rate-responsive and often single-chamber, patients may experience symptoms of pacemaker syndrome or general fatigue, effort intolerance from lack of chronotropic support and loss of AV synchrony.

Palpitations are also frequently reported by paced patients. These may be due to:

- Intrinsic tachyarrhythmia
- Ventricular tracking of an atrial tachyarrhythmia
- Pacemaker-mediated tachycardia
- Excessive rate response from a sensor-driven pacemaker
- Search hysteresis.

Before mode switching was available, ventricular tracking of atrial tachyarrhythmias was a frequent source of palpitations. Without mode switching, treatment often required programming the pacemaker to a non-tracking mode.

Pacemaker-mediated tachycardia (PMT) may occur when AV synchrony is uncoupled and a ventricular event results in retrograde atrial activation. The retrograde atrial event is sensed on the atrial sensing channel of the pacemaker and initiates the AVI. When the AVI interval times out, a ventricular pacing artifact is delivered. If retrograde conduction is sustained, the patient will have a sudden increase in their paced rate, usually to a rate at or near the programmed maximum tracking rate. The tachyarrhythmia will persist until retrograde atrial activation ends, the retrograde atrial event falls in the postventricular refractory period and is not sensed or a PMT termination algorithm is invoked (see Chapter 8, Programming). PMT is most commonly initiated by a premature ventricular event or atrial failure to capture.

A rate-adaptive pacemaker that is programmed too aggressively and search hysteresis may also cause symptoms (see above). Occasionally, patients who are paced after being chronotropically incompetent for a long period of time may have poor tolerance of the new rate response. Although paced rates may be appropriate, patients may have a sensation of relative tachycardia and complain of palpitations. This may require reprogramming the pacemaker to a less aggressive rate response and allowing the patient to adjust slowly to faster rates. The rate response can then be reprogrammed as tolerated.

As discussed in Chapter 8, an inappropriately programmed rate-adaptive sensor may result in suboptimal cardiac output, which can be corrected by optimization of sensor settings.

Patients who have abrupt 2:1 AV block at the upper rate limit may have symptoms from the sudden change in ventricular rate. Although this is much less common at present given the fact that the majority of patients are programmed to a rate-adaptive mode, 2:1 AV block upper rate response may lead to symptoms, and caregivers need to understand the phenomenon. A sudden decrease in heart rate obviously affects cardiac output (cardiac output = heart rate × stroke volume). If this upper rate behavior is recognized as the cause of the patient’s symptoms, reprogramming the pacemaker may alleviate the problem. Programming changes may include:

- Shortening the total atrial refractory period to allow a higher achievable upper rate limit
- Shortening the postventricular atrial refractory period
- Programming rate-adaptive AV delay “on”
- Programming “on” or optimizing sensor-driven pacing, or both
- Programming “on” another feature, such as rate smoothing or fallback, to minimize sudden changes in cycle length.
Primary cardiac abnormalities may also result in suboptimal cardiac output. Development of a primary cardiomyopathic process, left ventricular dysfunction due to ischemic disease or tachyarrhythmias may be manifested by symptoms compatible with low cardiac output.

Conclusion

The number and versatility of current pulse generators and lead systems available challenge the caregiver to understand all the various devices and their features when assessing a system for potential malfunction. A thorough understanding of pacemaker timing cycles and electrocardiography (Chapter 7), pacemaker radiography (Chapter 11), and programming (Chapter 8) is critical for successful troubleshooting. An understanding of the clinical situation, a systematic approach to troubleshooting, assessment of pacing system integrity and judicious use of manufacturers’ technical support teams can help distinguish between malfunction and apparent malfunction.

Implantable defibrillator troubleshooting

ICDs reduce the risk of death from lethal tachyarhythmias in high-risk individuals. As with pacemakers, ensuring continuous therapeutic efficacy requires periodic assessment of device function to detect battery deplition, mechanical system disruption, and suboptimal programming before any of these conditions becomes manifest. Preventive programming to minimize morbidity such as heart failure and shock delivery is covered in Chapter 7; routine follow-up, including device-enabled remote health status monitoring, is discussed in Chapter 13. At times, however, a patient comes to medical attention because of frequent (possibly inappropriate) device discharges or delayed, ineffective, or absent therapy in the face of a ventricular tachyarrhythmia. In these circumstances, a systematic approach must be followed to determine the cause of the problem and offer an appropriate solution.

This section of the troubleshooting chapter describes a stepwise approach to evaluation in patients with suspected device malfunction and reviews the diagnostic tools available and their interpretation. It then reviews the two most common problems seen: excessive device therapy and insufficient or inadequate therapy. Thus, this section is divided into three subsections:

- Diagnostic tools and patient approach (patient history; system radiography; telemetered device status, including battery voltage and charge time; pacing parameter assessment; real-time telemetry and maneuvers; stored electrogram analysis; and surface electrocardiography)
- Differential diagnosis and management of frequent or recurrent ICD shocks
- Differential diagnosis and management of delayed, absent or ineffective therapy for documented ventricular tachyarrhythmias.

The evaluation of medical complications associated with device implantation—infecion, pericarditis, pneumothorax, and so on—is covered in Chapter 5. The assessment of apparently inappropriate pacing function is covered in the preceding section.

Diagnostic tools and patient approach

Advances in ICD technology have greatly enhanced the ability to determine the cause of suspected system malfunction. Table 10.9 summarizes our systematic approach for identifying the cause of suspected defibrillator malfunction. The interpretation of each of the diagnostic steps is discussed in this section; patient management is discussed in the subsequent sections.

Patient history and physical examination

The patient’s history can provide important information about whether shocks are inappropriate (due to non-ventricular arrhythmias, system malfunction or oversensing) or appropriate (triggered by a sustained, hemodynamically significant ventricular tachyarrhythmia). Defibrillators perform periodic self-diagnostic tests and generate an audible tone or vibrate if an abnormality (such as low battery voltage or abnormal lead impedance) is found; therefore, device interrogation should be performed in patients reporting audible device tones or vibrations (Figs 10.56 and 10.57). Syncope preceding defibrillator discharge occurs in only 10–20% of patients, but is highly indicative of therapy for a ventricular tachyarrhythmia. Risk factors for syncope include low ejection fraction, chronic atrial fibrillation, and ventricular rates above 180 bpm. Severe symptoms are present in the minority of episodes; when they occur before shocks,
they strongly suggest appropriate therapy. Pacemaker-dependent patients are an exception to the rule: interruption of pacing caused by oversensing (e.g., lead fracture noise or diaphragmatic myopotentials (Fig. 10.58)) may present as bradycardic syncope followed by a shock. Importantly, absence of symptoms
Fig. 10.56 Conductor failure identified by lead alert. Top panel shows marked, abrupt increases in all measured impedance values triggering a lead alert on July 28, 2 days after the patient was seen in clinic. The patient did not hear the alert tones, probably because the ICD was implanted submuscularly. Middle panel shows episode summaries from inappropriately detected episodes of VF during intense stationary bicycle exercise in “spinning” class (August 1). Bottom panel shows the corresponding stored electrogram from the true-bipolar sensing lead. Signals are probably caused by mechanical motion at the fracture site. Saturation of rate sensing electrogram is characteristic of conductor failure; but persistence of oversensing throughout the cardiac cycle is not and is probably caused by intense exercise.

Fig. 10.57 Plot of high-voltage impedance of the SVC coil. The abrupt increase in impedance on 06/10/07 indicates lead malfunction. Since the SVC coil is used for shock delivery but not sensing, this type of malfunction in isolation does not cause inappropriate shocks, but can result in ineffective shock delivery.
Fig. 10.58: Oversensing of diaphragmatic myopotentials causes inhibition of bradycardiac pacing and delivery of an inappropriate shock. High-frequency myopotentials have greater amplitude on integrated bipolar sensing electrogram (RV tip-coil) than high-voltage electrogram (shock). VP-S, paced intervals; VS, intervals in sinus zone; VF, intervals in VF zone; Epsd, episode start/end. Repositioning the lead eliminated the problem.
does not indicate absence of ventricular arrhythmias. In a study with electrocardiographic documentation of the rhythm leading to shock, > 60% of ventricular tachycardia (VT) episodes and > 90% of supraventricular tachycardia (SVT) episodes were associated with minimal or no antecedent symptoms. For the
asymptomatic patient who receives shocks, additional information is required to determine the appropriateness of therapy.

The temporal pattern of shock delivery may offer information about whether shocks are appropriate. Clusters of shocks occurring within seconds or minutes in rapid succession are a strong indicator of inappropriate therapies. Intuitively, this makes sense; the success rate for two shocks for termination of VT or ventricular fibrillation (VF) exceeds 90% in a patient with an acceptable defibrillation threshold at implantation. Additional shocks in a patient who is clearly free of fatal tachyarrhythmias suggest that the therapy is probably inappropriately triggered by a rapidly conducting supraventricular rhythm not terminated by the first shock (particularly sinus tachycardia), by device malfunction (such as lead fracture noise), or by electromagnetic noise mimicking VT or VF and resulting in inappropriate detection. Indeed, inappropriate therapies have been associated with clusters of 4.0 ± 2.0 shocks per episode, compared with 1.6 ± 0.9 shocks per appropriate episode. Knowledge of a patient’s arrhythmia history may also be useful—persons with known atrial fibrillation or with previously documented SVT may be at increased risk of having one of these arrhythmias as the cause of shocks, although this has not been consistently seen in prospective studies.

Conversely, therapy for SVT does not occur in patients with complete atrioventricular block who are pacemaker-dependent. Termination of a tachyarrhythmia by a single shock is not diagnostic of VT.

Patient activity at the time of device discharge may also be helpful. Stretching or deep breathing may unmask a latent conductor defect, with “make-break” noise that can lead to inappropriate VT detection. Similarly, deep breathing or sitting up may lead to myo-
potential oversensing with similar results. Multiple shocks during vigorous exercise suggests inappropriate therapy for sinus tachycardia if the integrity of electrodes is verified.

History and physical examination (along with radiography) can usually determine the location of the pulse generator and whether the patient has more than one device. There is a significantly increased risk of lead failure with the use of abdominal ICDs compared with pectoral ICDs. Therefore, lead malfunction should be particularly considered in patients with frequent shocks and abdominal implantable defibrillators. Patients with separate pacemakers and defibrillators are at increased risk for device–device interactions, which could lead to over-detection or underdetection (discussed further, below).

Radiography
In our experience, radiographic manifestations of lead failure were present in 43% of patients found to have lead malfunction. Defibrillators should be examined radiographically when failure is suspected and after signif-

Fig. 10.61 Subclavian crush. (A) Note the sharp bend on the lead at the site where it passes between the subclavian vein and the first rib (arrow). (B) Lead extracted from a patient with subclavian crush. Note the disrupted insulation and the disarray of the fillers (the circumferentially coiled conductor) at the site of the crush.
A number of abnormalities may be found on system radiographs. Macrodislodgment is diagnosed when the lead tip is in a clearly different location (often pulled back) than that on a previous film. Coronary sinus lead dislodgment is best seen in the lateral film (Fig. 10.59). Right ventricular (RV) apical leads dislodge towards the tricuspid valve (Fig. 10.60). Since the lead appearance may shift between radiographs because of differences in systole and diastole or degree of inspiration, only gross changes can be diagnosed. Subclavian crush occurs when a lead is compressed in the narrowly confined space between the first rib and the clavicle; this is a common site of fracture, and this region should be carefully examined on all device radiographs (Figs 10.61 and 10.62). Radiography may also detect conductor defects, a loose pin connection in the header, or abandoned or incompletely removed leads, which may give rise to contact noise. Patients with Twiddler’s syndrome often unconsciously manipulate their systems, frequently rotating the pulse generator in the pocket. This can lead to twisted, dislodged, or fractured leads, and the resultant torsion is radiographically visible (Figs 10.63 and 10.64). The left ventricular (LV) electrode of cardiac resynchronization ICDs, most commonly placed in a tributary of the coronary sinus, dislodges proximally into the coronary sinus or the right atrium, although exceptions occur (Fig. 10.65). When lead malfunction is suspected and system radiography is unrevealing, fluoroscopy may be diagnostic. In patients with recent device implantation, radiography detects surgical complications such as pneumothorax and retained sponge. System radiography is covered extensively in Chapter 11 “Pacemaker and ICD Radiography,” and briefly in Chapter 13 “Follow-up.”
Fig. 10.63 Twiddler’s syndrome. The left panel shows a patient with Twiddler’s syndrome. Repeated twisting of the device (in this case, a pacemaker) resulted in coiling of the lead in a manner atypical for implanter management of extra lead in the pocket (long arrow). This twisting of the lead applies torsion, and can result in lead fracture, insulation breakdown, or dislodgment. A loop is also seen in the distal lead, suggesting that torque has accumulated along the lead's length (short arrow). The right panel shows a more typical appearing implant—note the absence of twisted lead in the pocket.

Fig. 10.64 Twiddler’s syndrome in a patient with a resynchronization ICD (CRT-D). This patient has a history of Twiddler’s syndrome and previous device revisions. Note that in this example the twisting was performed in a manner such that the leads looped in the pocket under the can in an orderly manner. This resulted in retraction (rather than twisting, as shown in Fig. 10.63) of the leads, so that the CS lead was pulled back to the subclavian vein (“LV lead” in figure). Note the tension on the atrial lead as well. The right ventricular lead has appropriate slack. The effect of Twiddling depends on the amount of twisting or spinning applied by the patient, the amount of redundant lead in the pocket, and the effectiveness of fixation to the pectoralis, among other factors.
Fig. 10.65 Dislodgment of an EASYTRAK® 3 lead from the coronary sinus into the right ventricle. The black arrow indicates the dislodged lead in the posteroanterior (PA, left panel) and lateral (right panel) chest X-ray. Note the anterior position of the lead in the lateral chest film, consistent with a ventricular position. Note, however, that in the PA film the dislodged lead remains proximal to the right ventricular apex (as seen by location of the RV lead tip). Coronary sinus leads most commonly dislodge into the right atrium.

Fig. 10.66 ICD leads and electrograms. The left panel shows an ICD system including left-pectoral active can and RV lead. Right panel shows telemetered electrograms. The dual-coil lead uses true-bipolar sensing between tip and ring electrodes. Right panel shows telemetered high-voltage (shock), far-field (FF-VEGM) and sensing, near-field (NF-VEGM) electrograms with annotated markers. Arrows on marker channel denote timing of R waves sensed from true-bipolar electrogram. ICDs measure all timing intervals from this electrogram and display them on the marker channel, which also indicates the ICD's classification of each atrial and ventricular event by letter symbols. In this figure, VS indicates sensed ventricular events in the sinus rate zone and numbers indicate RR intervals. The stored near-field, rate-sensing electrogram is a wide-band (unfiltered) signal in Medtronic ICDs, but filtered in Boston Scientific and St Jude ICDs.
Telemetered data

Device telemetry reveals battery status and the most recent capacitor charge time. If the device battery reaches end of life, function may become unreliable. Additionally, a depleted battery or excessive capacitor charge time may lead to absent, delayed or ineffective therapy. Telemetry also provides the programmed parameters, including programmed pacing rate, VT and VF detection zones and detection enhancements, all of which are important to determine whether device function is appropriate for a given situation.

Battery depletion

ICDs use batteries composed of lithium anodes and silver vanadium oxide (Ag$_2$V$_4$O$_{11}$) cathodes in which reduction at the cathode occurs in multiple steps. Unloaded cell voltage falls from about 3.2 to 3.1 V at about 30% battery depletion. In most ICD batteries, voltage then falls rapidly to about 2.6 V, where it reaches a plateau until the battery is 80–90% depleted. With the exception of Medtronic ICDs, the 2.6-V plateau is nominal performance for most ICD batteries. End of service indicators correspond either to an unequivocal reduction in unloaded voltage about 0.1 V below this plateau or an excessive increase in charge time. Battery assessment is discussed in greater detail in Chapter 13.

Premature battery depletion

The most common cause is excessive external power drain due to pacing or capacitor charging. Pacing problems include unnecessary ventricular pacing, high pacing outputs, and lead insulation failures. Asymptomatic premature battery depletion related to capacitor charging may stem from repeated aborted shocks due to repetitive non-sustained VT or oversensing due to lead-connector problems. Repeated shocks due to VT storm may also rapidly deplete the battery. In hospitalized patients with...
Fig. 10.68 Provocative maneuvers during real-time telemetry to diagnose system malfunction. (A) Cough during real-time telemetry. From top to bottom are the surface electrocardiogram (ECG), far-field ventricular electrogram, and marker channel. During the cough, electrical noise is seen on the far-field electrogram (recorded between the coil and the device can) while the surface ECG displays continuing normal sinus rhythm, confirming that the electrical noise is non-cardiac. The marker channel shows appropriate QRS sensing (one sensed event for each QRS), indicating that the fracture involves the shocking coil but spares the conductors to the tip or ring used for sensing. (B) Oversensing of diaphragmatic myopotentials during deep inspiration. The patient has marked underlying sinus bradycardia and high-grade atrioventricular block. From top to bottom are shown the surface ECG, atrial electrogram, ventricular electrogram, and markers. With deep inspiration, diaphragmatic myopotentials are sensed as ventricular tachycardia (VT) and ventricular fibrillation (VF) events (beginning at marker VT-1 436). During VT/VF detection, pacing is suspended, resulting in a pause. This patient had received a shock during deep breathing exercises. Reprogramming from “nominal” to “least” sensitivity eliminated the problem. Assessment of adequate ventricular fibrillation detection should be performed when sensitivity is diminished.
VT storm, reducing shock output to a safe level based on the required shock strength may prolong ICD generator service significantly. Some diagnostic features have high power consumption. For example, in Medtronic ICDs storing electrograms prior to each VT/VF episode requires continuous amplification of the unfiltered electrogram, substantially reducing longevity. ICD component failure is a rare cause of battery depletion.

Pacing parameters

**Impedance**

Abnormal pacing parameters may indicate the presence of lead conductor fracture, insulation breach, microdislodgment or macrodislodgment, ineffective antitachycardia pacing or impaired sensing (Table 10.9). As in pacemakers, significant elevation of the pacing impedance suggests a discontinuity in the lead or its connection to the pulse generator, and low impedance suggests an insulation defect or short within the lead. Pace-sense lead discontinuities in ICDs, unlike those in pacemakers, may not only lead to pacing failure (which may remain asymptomatic in a patient who is not pacemaker-dependent), but also result in ineffective termination of VT (if antitachycardia pacing is attempted) or in inappropriate shocks if “make-break” noise is detected by the pulse generator as VT/VF. Impedance values are avail-

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**Fig. 10.69** Using multiple real-time electrogram channels to diagnose the source of malfunction in a patient who received a shock while shaking his cardiologist’s hand the day after a CRT-D (resynchronization ICD) implant. In all four panels, the top tracing is the surface ECG (which contains artifact due to hand-shaking during recording in effort to reproduce malfunction); the second row depicts the marker channels, which show device interpretation of events; the bottom row shows the device electrogram (RV tip to RV ring from the true-bipolar lead is top left; RV tip to RV distal coil bottom left; can to RV coil top right; RV tip to LV tip bottom right). Note that in all of the panels there are more “VS” markers than surface QRS complexes, consistent with oversensing of noise. “VS” is seen (as opposed to “TS” or “FS”) since detection is turned off for troubleshooting. The noise is seen in all of the electrograms (arrows) except the can to RV coil electrogram (top right). The can to RV coil tracing is the only one in which the RV tip is not part of the circuit. This suggests the problem lay in the conductor or connection to the RV tip. At re-operation, a loose setscrew was found in the RV-tip port of the header.
**Fig. 10.70** Use of real-time telemetry to assess baseline electrogram morphology during sinus rhythm. (A) Stored electrograms from an episode of wide-complex tachycardia. From top to bottom are the atrial electrograms, far-field (can to right ventricular (RV) coil) electrogram, and marker channels. Atrial electrograms show continuing atrial flutter, whereas ventricular electrograms show a regular wide-complex tachycardia, which could represent conducted atrial flutter or concomitant ventricular tachycardia. (B) Real-time recordings during sinus rhythm showing (top to bottom) surface electrocardiogram marker channels, and far-field (can to RV coil) electrogram. Note the similarity between the far-field electrogram during sinus rhythm and the electrogram morphology during the episode in (A), indicating that the tachycardia was rapidly conducted atrial flutter. Also incidentally noted in (B) is far-field R-wave oversensing (sensing of the QRS on the atrial channel), seen as an “AS” marker immediately following the “VS” marker. The PR Logic detection algorithm can take far-field R-wave oversensing into consideration if it is consistently present.
able non-invasively by device telemetry. Normal pacing impedance is a function of lead design, but for any lead (except Y-adapted lead combinations) impedance $< 200\Omega$ indicates an insulation defect and impedance $> 2000\Omega$ indicates conductor failure. A loose set screw or faulty adapter can also cause abnormally high imped-

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Fig. 10.71 Method for analysis of stored electrograms in dual-chamber ICDs (upper panel) and single-chamber ICDs (lower panel). Asterisks denote weaker criteria. ATP, antiarrhythmic pacing; AFib, atrial fibrillation; AFlu, atrial flutter. See text for details. (Reproduced with permission from Sverdlov C, Friedman P. Advanced ICD troubleshooting: Part I. PACE 2005; 28:1322–46, Blackwell Publishing.)
Fig. 10.72 Use of stored episode data for diagnosis of arrhythmia. All tracings are from the same patient. From top to bottom are atrial, rate (near-field) and shock (far-field) electrograms. (A) Episode of atrial tachycardia. Beginning with the fifth atrial complex (cycle length, 430), the atrial rate accelerates and is conducted rapidly to the ventricles. The changes in AA intervals precede the changes in VV intervals, as expected in atrial tachycardia (AA 430 leads to VV 430, AA 342 leads to VV 348, AA 330 leads to VV 336 and so on). Also, the far-field morphology is unchanged, consistent with supraventricular tachycardia. The atrial tachycardia is detected as ventricular fibrillation (VF), leading to a shock. (B) After the shock, nine atrial tachycardia complexes are conducted with a wide QRS (most likely a result of the shock) and are followed by resumption of sinus rhythm. Despite the wide QRS complex, the fact that the AA intervals “drive” the VV intervals confirms a supraventricular rhythm. (Continued.)
Fig. 10.72 (Continued.) (C) Subsequent development of ventricular tachycardia (VT). The tachycardia begins with a ventricular complex (with cycle length 285). The ventricular rate is greater than the atrial rate, and the morphology of both near-field and far-field electrograms is different from the sinus morphology (as seen in the first four ventricular complexes). (D) After a successful shock (the first four complexes are sinus tachycardia with aberrant QRS, as seen in B), VT recurs. The tachycardia begins with a ventricular complex (VF 281), has a ventricular rate exceeding the atrial rate, and has a morphology that is different from the aberrantly conducted sinus tachycardia, all consistent with the diagnosis of VT. This example demonstrates how the initiation of VT during an aberrant supraventricular rhythm can be diagnosed.
The magnitude of impedance change caused by lead fracture is likely to be model specific; early reports indicate that the Medtronic Fidelis lead failure occurs with impedance in the 1400–2000 $\Omega$ range.52,53 Abrupt changes in the trend of daily, measured lead impedance can be useful in diagnosing lead fracture. The electrograms in these two panels are essentially identical, indicating that treated arrhythmia is SVT. The electrogram during tachycardia is clipped at the maximum amplitude of +8 mV. The lower panels are “flashback interval” plots of the RR-interval cycle lengths prior to rate-only detection of VF, which occurs at the right side of each panel. The interval number prior to detection is plotted on the abscissa, and the corresponding interval is plotted on the ordinate. The lower left panel shows 2000 RR intervals prior to detection. A tachycardia is present throughout. Shortly after the 400th interval, the rhythm accelerates gradually in a manner typical of sinus tachycardia and decreases below the programmed VF detection interval of 340 ms. The lower right panel shows this gradual acceleration on an expanded scale during the last 100 intervals prior to detection. Cycle-length measurements are truncated to the nearest 10 ms. (B) Stored electrogram during therapy of the tachycardia detected in (A). The first VF shock (VF Rx 1) results in widening of the electrogram without change in the cycle length of 330 ms. This is probably due to shock-induced right bundle branch block, which was documented in this patient at electrophysiological testing. The tracing is discontinuous at the end of the first line and continuous thereafter. Shocks 2, 3 and 4 resulted in no change in rate or electrogram morphology. On the second line, the fifth VF shock (VF Rx 5) induces VT with cycle length 280 ms, despite appropriate synchronization to the nadir of the R wave. The sixth VF shock (VF Rx 6) accelerates the VT to cycle length 210 ms. This rhythm terminates spontaneously 21 s later, and sinus tachycardia with a wide electrogram resumes (asterisk). No additional shocks were delivered during these 21 s, because the maximum number of therapies per zone is six in this ICD. The patient reported that during exertion he experienced multiple shocks followed by syncope. The programmed shock strength was 24 J for the first shock and 34 J for subsequent shocks. (Reprinted with permission from Swedlow C. Optimal programming of sensing and detection in single chamber ventricular ICDs. Card Electrophysiol Rev 2001; 5:85–90, Springer.) (Continued.)
tone or vibratory stimulus if impedance measurements are abnormal. Figure 10.57 depicts a trend plot of shock-lead impedance with an abrupt impedance rise consistent with fracture; the plot was retrieved via remote monitoring (discussed in Chapter 13) and the lead revised before the failure became clinically manifest.

High-voltage lead impedance as measured by weak pulses correlates well with values measured during high-energy shocks. In Boston Scientific ICDs, values recorded by the painless lead impedance are comparable to those values recorded during delivery of a high-voltage shock. In older Medtronic devices, the pulse is delivered between the lead tip and the distal coil, resulting in normal low-voltage impedance values of 11–20 Ω, significantly lower than the impedance of a high-energy shock. If a proximal coil is present, its integrity is not assessed. Medtronic Marquis® and newer systems report independent proximal and distal coil impedances that closely approximate those seen during high-voltage shocks. When a lead defect is suspected with normal lead impedances, a full output shock is necessary to confirm lead integrity. Partial insulation defects are not always identified by low-energy pulses that result in insufficient current to activate the shorted high-output protection feature of modern ICDs (see below).

R wave (P wave) and pacing threshold
The amplitude of the R wave during sinus rhythm should be at least 5–7 mV for assurance that the lower amplitude electrograms that occur during VF are appropriately sensed. A number of factors may result in electrogram diminution, including medications, myocardial infarction, failed shock, lead tip fibrosis, microdislodgment and macrodislodgment. If the R-wave peak-to-peak voltage is < 5 mV, electrophysiologic testing to confirm adequate detection is warranted, particularly if arrhythmia detection has been delayed or absent. The introduction of automatic capture assessment in conjunction with remote monitoring systems...
that permit review of device measurements and trend plots between clinic visits may further facilitate early diagnosis of diminished R-wave amplitude and deteriorating sensing function.

The same factors that affect the R-wave amplitude may also increase the pacing threshold. Increases in pacing threshold due to some drugs (particularly Vaughan-Williams class I agents, such as flecainide) may be rate-dependent. Therefore, threshold increases may not be detected during bradycardia pacing, but may manifest as ineffective antitachycardia pacing. In patients with infrequent pacing, an elevated pacing threshold may be
acceptable as long as appropriate sensing function is confirmed, lead malfunction is excluded, and, if programmed, the ability to capture during antitachycardia pacing (for which outputs are independently programmable in many devices) is confirmed.

Real-time telemetry

Implantable defibrillators telemeter and store electrograms between various implanted electrodes. Typically, one or more source electrograms are programmable (Fig. 10.66). Far-field electrograms are recorded between widely spaced electrodes including at least one shock electrode (such as the right ventricular coil and the pulse generator can), resulting in a large “antenna.” They integrate electrical signals from a large volume of myocardium, and thus reflect QRS morphology. The far-field electrogram more closely resembles the surface electrocardiogram than the near-field (pace-sense) electrogram, may demonstrate P waves when they are present and is used by some morphology detection enhancements for rhythm diagnosis. Recording the far-field electrogram, particularly with the patient in various positions, is useful for comparison with stored electrograms to determine whether a tachyarrhythmia was supraventricular or ventricular in origin. In contrast, near-field electrograms are recorded between the lead tip electrode and the adjacent ring or coil. Since they have a “smaller antenna,” they provide less morphological information, but are used for heart rate determination by the device due to their characteristic sharp (high slew) morphology. Since near-field electrograms are used by the defibrillator to determine ventricular rate, they are helpful for diagnosing oversensing. Importantly, the morphology of electrograms associated with ventricular and supraventricular arrhythmias may appear very similar when examined with near-field electrograms (Fig. 10.67), and 5–10% of VT near-field electrograms mimic the electrogram recorded during normal sinus rhythm.61

Fig. 10.75 Underdetection of VT and undersensing of VF. The lower panel is a “flashback interval” plot of RR-interval cycle lengths prior to detection of VF, which occurs at the right side of each panel. The interval number prior to detection is plotted on the abscissa, and the corresponding interval is plotted on the ordinate. Horizontal lines indicate the VT detection interval (TDI) of 400 ms and VF detection interval (FDI) of 320 ms. Shortly after the 500th interval preceding detection, regular tachycardia begins abruptly. The constant cycle length indicates reliable sensing. This VT is not detected despite reliable sensing because the cycle length is more than the programmed TDI. VT persists for 3.7 min until approximately interval 280 prior to detection, when sensed intervals become highly variable. This indicates degeneration of the rhythm to VF with undersensing that delays detection. During VT and VF, atrial flashback intervals (not shown) indicated lower rate limit bradycardia pacing at 40 bpm (1500 ms). The upper panel shows stored atrial and far-field ventricular electrograms immediately prior to detection with atrial and ventricular marker channels. Specific undersensed electrograms cannot be identified because the rate sensing electrogram was not recorded. However, long-sensed RR intervals ending with VS markers indicate undersensing and correspond to long interval in upper panel. “VF Therapy 1 Defib” at lower right (arrow) denotes detection of VF. (Reproduced with permission from Sverdlow C, Friedman P. Advanced ICD troubleshooting: Part I. PACE 2005; 28:1322–46., Blackwell Publishing.)
Fig. 10.76 Appropriate therapy for VT with 1:1 VA conduction. Bipolar atrial electrogram (RA), dual-chamber marker channel and rate-sensing (RV) electrogram are shown. Asterisk denotes onset of VT during sinus tachycardia, identified by abrupt acceleration of ventricular rate and change in electrogram morphology without change in atrial rate. Morphology discriminator requires five of eight match scores $\geq 60\%$ to withhold therapy. During sinus tachycardia, scores exceed 60%. (Five are 100%.) During VT, most scores are $< 60\%$. Check marks above marker channel indicate that morphology algorithm classifies beats as supraventricular. “X” marks indicate beats classified as VT morphology. The VT cycle length is moderately irregular. Changes in VV interval precede those in AA interval. Ventricular antitachycardia pacing (ATP) at right of lower panel results in transient VA block without acceleration of the atrial rate followed by 1:1 VA conduction. The near-simultaneous atrial and ventricular activation during tachycardia is more typical of typical (antegrade-slow, retrograde-fast) AV nodal re-entrant tachycardia than VT, but the shortening of the AV interval at the onset of tachycardia is inconsistent with this diagnosis. Pacing-induced VA block without acceleration of the atrial rate is also unusual in AV nodal reentry. “Trigger” in lower panel indicates detection of VT. “D=” at onset of ATP indicates that atrial rate = ventricular rate. ‘S’ denotes intervals interval the “Sinus” zone longer than the VT detection interval of 400 ms. “T” denotes intervals in VT zone. DDI, mode switch. Time line is in section. (Reproduced with permission from Swerdlow C, Friedman P. Advanced ICD troubleshooting: Part I. PACE 2005; 28:1322–46., Blackwell Publishing.)

Fig. 10.77 Inappropriate detection of rapidly conducted atrial fibrillation. Stability and morphology algorithms are combined with “ANY” in this St Jude ICD so that VT is diagnosed if either discriminator classifies rhythm as VT. Stability algorithm incorrectly classifies rhythm because ventricular cycle lengths regularize. Morphology algorithm correctly classifies rhythm as SVT. The rhythm would have been classified correctly if the morphology discriminator alone had been programmed. “F” markers indicate ventricular intervals in VF (Fib) zone. Other abbreviations as in Fig. 10.76. (Reproduced with permission from Swerdlow C, Friedman P. Advanced ICD troubleshooting: Part I. PACE 2005; 28:1322–46., Blackwell Publishing.)
Fig. 10.78 VT during paroxysmal atrial fibrillation. Interval plot (upper panel) and continuous stored electrogram are shown. VT at cycle length 230 ms is diagnosed by AV dissociation and rate as “Fast VT.” Change in morphology is clear on ventricular electrogram, but is not used for diagnosis. Antitachycardia pacing is delivered toward the right side of the first electrogram panel. It changes the morphology of VT, which becomes polymorphic and terminates toward the right side of the middle electrogram panel (type II break). VF is detected during this delayed termination (“VF Defibr Rx 1” in middle of middle panel), but shock is aborted (Aborted) in middle of bottom panel. Dual-chamber interval plot at top shows onset of VT during atrial fibrillation, persistence of VT after antitachycardia pacing and subsequent termination. Most VT that occurs during paroxysmal atrial fibrillation is rapid and has cycle lengths classified in the VF zone using traditional programming. Antitachycardia pacing therapy for “fast” VT reduces inappropriate shocks. Ventricular intervals are classified as TS (fast VT), VS (ventricular sensed—sinus zone or during capacitor charging), FS (VF zone), TP (antitachycardia pacing) and VR (ventricular refractory) period after end of capacitor charging (CE). (Reproduced with permission from Swerdlow C, Friedman P. Advanced ICD troubleshooting: Part I. PACE 2005; 28:1322-46., Blackwell Publishing.)
During troubleshooting, both types of electrograms are evaluated in real time with the patient performing provocative (straining) maneuvers in several positions. Real-time electrogram recordings may be used for diagnosing oversensing and lead malfunction. Noise on the shocking elements suggests lead failure affecting shocking electrodes. Conversely, electrical signals on the near-field electrogram that do not correspond to far-field (and surface ECG) events may indicate diaphragmatic myopotential oversensing or “make-break” noise related to lead failure (Fig. 10.68). When multiple electrogram sources are available, as with resynchronization systems, recording each of the possible pathways during provocative maneuvers may provide useful diagnostic information (Fig. 10.69). If a patient describes a stereotypical maneuver that reproduced the event (for example, reaching or coughing), repeating the maneuver during electrogram recording may be diagnostic (Fig. 10.68). Comparison of electrograms recorded in real time while the patient is known to be in normal sinus rhythm or atrial fibrillation with stored episode electrograms may distinguish SVT from VT. In contrast to stored ventricular arrhythmia episodes, the morphology of stored SVT episodes resembles that of the usual supraventricular rhythm in the absence of aberrancy or post-shock distortion41,61 (Fig. 10.70).

**Stored episode data**

Episode information including intervals and electrograms are stored when a sustained tachyarrhythmia is detected. Analysis of stored episode data frequently diagnoses the cause of therapy delivery. The use of stored data to diagnose lead fracture and oversensing is reviewed below. This section reviews analysis of stored episode data recorded in response to a
true tachycardia, in order to discriminate VT from SVT. Figure 10.71 summarizes methods for analyzing single-chamber and dual-chamber electrograms.

**Analysis of single-chamber electrograms**

The morphology, abruptness of onset, and regularity of ventricular electrograms form the foundation of single-chamber SVT–VT discrimination. These are reviewed in detail below.

**Morphology**

The clinician should analyze electrogram morphology from all recorded channels, and particularly from a far-field electrogram. A real-time, reference electrogram of conducted sinus or baseline rhythm should be recorded to compare morphology with that of the stored electrogram (Fig. 10.70). Ideally, it should be recorded in the posture in which the episode occurred. The rhythm is classified as SVT if electrogram morphology is uniform and identical to the sinus/baseline morphology. It is classified as VT if morphology is uniform and distinctly different from the sinus morphology. Rate-related bundle branch block occurs when the rapid rate from an SVT results in block in one of the bundles, and can lead to misclassification of the arrhythmia using morphology. After shocks, electrogram
Fig. 10.81 Patient with a single-chamber defibrillator (Medtronic 7227) and a dislodged lead. (A) Hospital monitoring shows pacing of the atrium. Atrial pacing in a single-chamber ventricular defibrillator indicates that a lead tip is in an inappropriate location. The varying pacing intervals are due to the ventricular rate stabilization algorithm, which is "on." (B) Trend report of an event. Note the rapidly alternating VV intervals (300 ms alternating with 150 ms) beginning at –4 s, giving the appearance of "railroad tracks." This alternating pattern is suggestive of far-field signal oversensing. The "railroad tracks" terminate at +4 s, before the shock is delivered at 8 s (labeled 32.2 J), suggesting that a non-sustained ventricular tachycardia (VT) episode was shocked. (C) Electrograms corresponding to the trend report shown in (B). From top to bottom are far-field electrograms, near-field electrograms, and markers with VV intervals. The first complex is a sinus beat, followed by a run of non-sustained VT. The last two complexes are sinus beats. The lead is dislodged, with the tip in the atrium (hence the atrial capture with pacing in (A)) near the tricuspid annulus, so that atrial and ventricular activity is sensed. With the onset of VT, the short 150-ms intervals represent the ventriculoatrial interval, and the longer 310-ms cycle lengths represent the atrioventricular interval. Atrial oversensing leads to detection of ventricular fibrillation (VF) (at FD). (D) Continuation of electrograms from (C). The non-sustained VT has terminated. After capacitor charging is completed (CE for "charge end"), the defibrillator attempts to reconfirm whether VT or VF is still present. Since atrial oversensing results in two short intervals after CE (each of 170 ms), the device assumes that VF is still present and delivers a shock during sinus rhythm (CD for "charge delivered"). The lead was surgically repositioned without further problems.
morphology cannot be used to discriminate VT from SVT until post-shock distortion due to lead polarization and local bundle injury resolves (Fig. 10.72B). SVT cannot be discriminated from VT unequivocally using near-field electrograms in 5–10% of VTs.\textsuperscript{61,62}

Interval stability

Typically, the ventricular rhythm is irregularly irregular in atrial fibrillation and regular during monomorphic VT. The variability in R–R intervals seen during atrial fibrillation is used to distinguish it from VT, but exceptions occur. Usually, atrial fibrillation is detected inappropriately during an ongoing atrial fibrillation episode when a sufficient fraction of conducted beats exceeds the programmed VT or VF rate criterion. Thus, stored intervals are irregularly irregular prior to and during detection. However, because R–R intervals in atrial fibrillation are more regular at ventricular rates above ~170/min, interval stability cannot reliably discriminate atrial fibrillation from VT at these rates.\textsuperscript{63,64} Furthermore, the conducted ventricular rhythm in atrial fibrillation may regularize at slower rates due to transient organization of the atrial rhythm. During rapidly conducted atrial fibrillation, R–R intervals may be quite regular and electrograms frequently demonstrate subtle beat-to-beat variation in morphology, the intracardiac correlate of rate-related aberrancy. In contrast, electrograms during VT tend to be more uniform. Important pitfalls exist in making a rhythm diagnosis using these criteria. Amiodarone or type IC
antiarrhythmic drugs may cause monomorphic VT to become markedly irregular or polymorphic VT to slow, causing irregular intervals during true VT in the VT rate zone.65,66

Onset
Sinus tachycardia accelerates gradually and is always detected at the sinus-VT rate boundary (Fig. 10.73). In contrast, the onset of VT or paroxysmal SVT (including atrial fibrillation) is abrupt unless it originates during sinus tachycardia or SVT (Fig. 10.74). However, if VT starts abruptly with an initial rate below the programmed VT detection rate, the beginning of the stored electrogram does not record the onset of the arrhythmia. Rather, it records the VT as it accelerates across the programmed, sinus-VT rate boundary. In Medtronic ICDs, stored (“flashback”) intervals preceding the stored electrogram may permit correct diagnosis of an abrupt-onset arrhythmia at a rate slower than the VT detection rate (Fig. 10.75). In the absence of flashback intervals, the few seconds of stored electrograms prior to initial detection are insufficient to make a categorical determination of a “gradual-onset” arrhythmia.

Analysis of dual-chamber electrograms
Analysis of atrial and ventricular rates and AV relationships are the foundations of dual-chamber rhythm analysis. The added information provided by atrial electrograms significantly improves rhythm discrimination.46 If the ventricular rate is faster than the atrial rate, the diagnosis is VT. Interpretive issues arise for tachycardias in which the atrial rate is ≥ the ventricular rate.

Tachycardias with 1 : 1 AV relationship
The vast majority of tachycardias with 1 : 1 AV relationship are SVT, primarily sinus tachycardia or atrial tachycardia. VT with 1 : 1 VA conduction accounts for <10% of VTs detected by ICDs.67 The principal differentiating features between SVT and VT with 1 : 1 AV relationship include morphology of the ventricular electrogram, chamber of onset, and response to ventricular antitachycardia pacing. Atrial tachycardia usually begins with a short P–P interval (i.e., a premature atrial complex) followed by a short R–R interval, and each R–R interval is determined by the preceding P–P interval in the absence of ventricular ectopy (Fig. 10.72). In contrast, VT usually begins with a short R–R interval (Fig. 10.76). A few beats of AV dissociation may occur until 1 : 1 ventriculoatrial conduction stabilizes. In sinus tachycardia, the atrial rhythm accelerates gradually with an approximately stable PR interval. Atrial electrogram morphology in sinus rhythm often differs from the morphology of retrograde atrial electrograms in VT and from that of atrial electrograms during SVT. But these differences may be subtle, and their absence should not be considered as confirmatory of sinus P waves. The response to antitachycardia pacing may provide additional evidence (Figs 10.76 and 10.79, and further discussion below).

Tachycardias with atrial rate > ventricular rate
The physician must distinguish between conducted atrial fibrillation or atrial flutter (Fig. 10.77) and VT during atrial arrhythmia (Fig. 10.78). Most VT during paroxysmal atrial fibrillation is fast enough to be classified in the VF or FVT zone.68 The single-chamber criteria of abnormal ventricular morphology and regular ventricular rate are most helpful for diagnosing VT during atrial fibrillation. Conducted atrial flutter may be diagnosed in the presence of abnormal ventricular morphology if consistent 2 : 1 AV association or Mobitz 1 AV block is present. In contrast, VT during atrial flutter is diagnosed based on abnormal morphology and AV dissociation.

Response to therapy
When the atrial rate exceeds the ventricular rate, termination of a tachycardia by a single trial of ventricular antitachycardia pacing favors the diagnosis of VT. However, during atrial fibrillation, retrograde concealed conduction from ventricular antitachycardia pacing may result in post-pacing pauses and/or slowing of antegrade conduction that must be distinguished from true termination of VT. In contrast to arrhythmias in which the atrial rate exceeds the ventricular rate, termination of tachycardias with 1 : 1 AV association by antitachycardia pacing may be diagnostic. In ICD patients, the vast majority of 1 : 1 SVTs treated by ventricular antitachycardia pacing are atrial tachycardias. They are terminated by ventricular antitachycardia pacing only if the atrial rate accelerates during pacing. Thus, VT can be diagnosed if a 1 : 1 tachycardia terminates while high-grade VA block occurs at the onset of antitachycardia pacing without acceleration of the atrial rate. The response to unsuccessful antitachycardia pacing may also be helpful. If the atrial
cycle length is unchanged by ventricular antitachycardia pacing (tachycardia in the atrium does not depend on retrograde conduction, so that the ventricle is dissociated from the atrium), the diagnosis is SVT. If the atrial rate accelerates to the ventricular rate during ventricular antitachycardia pacing, the response at the termination of unsuccessful pacing therapy may be helpful: an AA V response is diagnostic of atrial tachycardia (Fig. 10.79), a VV A response is diagnostic of VT. If the atrial rate accelerates to the ventricular rate during ventricular antitachycardia pacing, the response at the termination of unsuccessful pacing therapy may be helpful: an AA V response is diagnostic of atrial tachycardia (Fig. 10.79), a VV A response is diagnostic of VT. An AA V response is one in which following the termination of ventricular pacing, two sensed atrial events are seen before the next sensed ventricular event; similarly, a VV A response is one in which two sensed ventricular events occur following the termination of pacing followed by an atrial event. A VAV response may occur in VT, AV nodal re-entrant SVT and AV re-entrant SVT.

If multiple arrhythmias occur with similar electrocardiogram morphology and ventricular rate, one episode may permit definitive diagnosis for all episodes. In tachycardias with 1:1 AV association, transient AV block permits the diagnosis of SVT; transient VA block permits the diagnosis of VT. A regular tachycardia during paroxysmal atrial fibrillation may be identified as VT if a tachycardia with the same rate and morphology occurred during sinus rhythm. If an arrhythmia is terminated by a shock, termination of multiple other episodes by a single trial of antitachycardia pacing favors VT if the atrial rate is greater than the ventricular rate (i.e., this suggests VT during atrial flutter, tachycardia or fibrillation). The approach to stored episode analysis is summarized in Fig. 10.71.

**Surface electrocardiography**

Surface electrocardiography during shock delivery is frequently not available. However, when present, it confirms the rhythm diagnosis preceding therapy. Therapy delivered during normal sinus rhythm indicates oversensing [lead fracture noise, T-wave oversensing, electromagnetic interference (EMI), or other forms of oversensing], but sinus rhythm at a rate above the VT detection rate must be excluded (Figs 10.80 and 10.81).

**Differential diagnosis and management in patients with frequent or recurrent shocks**

A patient with an ICD may present with frequent or recurrent shocks. Appropriate diagnosis and treatment are critical, since repetitive shocks are associated with significant morbidity, recurrent hospitalizations, anxiety, depression, and post-traumatic stress disorder. Broadly, multiple shocks may be due to one of four causes:

- Ventricular rhythms
- Supraventricular arrhythmias
- Device malfunction (detection enhancement error, oversensing, or lead fracture)
- Electromagnetic interference.

Differentiating the causes is important. Shocks due to VT or SVT may be treated by medications for arrhythmia suppression or rate control, by device reprogramming for enhanced specificity or utilization of painless pacing therapies or by catheter ablation to modify the arrhythmogenic substrate or to control the ventricular response during atrial arrhythmias. In contrast, device malfunction is treated by reprogramming (to prevent oversensing or correct algorithmic rhythm “misinterpretation”) or by surgical correction (to reposition a lead, or to replace lead or pulse generator), and EMI may require inactivation of the device (e.g., during electrocautery) or avoidance of environmental sources.

Table 10.10 lists potential causes for frequent shocks in patients with minimal symptoms. These are discussed in greater detail below.

**Emergency management**

When a patient receives one shock and has no residual symptoms, elective evaluation and interrogation is arranged. In contrast, repetitive shocks represent a medical emergency, as multiple shocks are painful, frightening, and may result in proarrhythmia. While most episodes in which more than two shocks are delivered occur as a result of inappropriate therapy, VT storm may occur in up to 10–20% of ICD recipients. Older patients with severely depressed ejection fraction, chronic renal failure, and who do not use lipid-lowering drugs are at increased risk.

The patient receiving repetitive ICD shocks requires urgent management. In the absence of a programmer, treatment steps include monitoring, medication, and magnet application. Surface electrocardiography permits determination of whether therapies are delivered following VT, SVT, or during sinus rhythm (Fig. 10.80). If therapies occur during SVT or during sinus rhythm (suggesting dislodgment, lead fracture, oversensing, EMI, or sinus rhythm in the VT zone), placement of a magnet over the pulse generator typically disables
VT/VF detection (and automatic therapies) without altering pacing function. Depending on how they are programmed, Boston Scientific ICDs may remain inactivated after magnet application for > 30 s, so that electrocardiographic monitoring must continue until interrogation with a programmer is performed and appropriate ICD function assured.

Specific causes of and management for shocks

**Ventricular arrhythmias**
Ventricular arrhythmias may cause recurrent ICD shocks—despite lack of symptoms—in patients with hemodynamically tolerated VT, with very rapidly detected and treated VT, or with non-sustained VT triggering shocks (Table 10.10). The diagnosis of ventricular tachyarrhythmias is confirmed by diagnostic stored-event information as described above. When the frequency of ventricular tachyarrhythmias dramatically increases, medical causes are considered (Table 10.11), although frequently are not found.

There are three strategies for managing repetitive shocks due to ventricular tachycardia: device programming optimization, adjunctive antiarrhythmic drug use, and catheter ablation. Self-terminating VT may commonly trigger ICD therapy\(^{75,76}\) and lengthening detection from nominal values results in a high frequency of episode termination (approximately 30%) without significant detection delay.\(^{76,77}\) Antitachycardia pacing painlessly terminates up to 90% of VT episodes, with a risk of shock-requiring arrhythmia acceleration < 5%; it is effective when empirically programmed.\(^{78}\) Parameter optimization to prevent shocks is discussed in detail in Chapter 7.

Antiarrhythmic drugs significantly reduce the risk of ICD shocks, but benefit is balanced by adverse drug
CHAPTER 10 Troubleshooting

475

The effects of antiarrhythmic drugs on defibrillation and on VT detection are reviewed in Chapter 1. In patients with repetitive therapies due to VT, antiarrhythmic drugs are commonly the first line of defense. Catheter ablation was originally limited to patients with slow, hemodynamically stable refractory VT. Advances in mapping and ablation technology have enabled successful treatment of even rapid “unmappable” arrhythmias, resulting in a >90% reduction of appropriate ICD therapies. The role of drugs and catheter ablation in the management of ventricular arrhythmias has been reviewed in professional society guidelines.

Supraventricular arrhythmias

Supraventricular arrhythmia is the most common cause of inappropriate device discharges, responsible for 65–80% of inappropriate shocks. The diagnosis is suggested by exertion at the time of the event (sinus tachycardia), by a history of atrial arrhythmias, and by more than two shocks in a cluster, and it is confirmed by characteristic stored electrograms. The far-field electrogram morphology remains unchanged (in the absence of aberrancy), the rhythm may not respond to antitachycardia pacing (atrial fibrillation or sinus tachycardia) and the heart rate may increase after shocks (especially for sinus tachycardia). Sinus tachycardia shows a characteristic gradually increasing heart rate, and P waves precede QRS complexes, seen on the far-field electrogram or atrial channel. Atrial fibrillation is characterized by irregular ventricular intervals, and when an atrial lead is present, rapid, fractionated atrial electrograms are seen. However, if the ventricular rate becomes regular and the ventricular morphology changes from baseline, the possibility of a double arrhythmia—development of VT during atrial fibrillation—is likely. For a detailed review of diagnostic criteria, see Stored episode data, above.

Inappropriate therapies due to SVTs must be eliminated because of discomfort to the patient (if shocks are delivered) and the risk of proarrhythmia, as shown in Figs 10.73 and 10.82. There are three approaches to preventing recurrent inappropriate therapies due to SVTs: (i) reprogramming the detection rate or programming detection enhancements to prevent ventricular therapy (and to deliver atrial therapy appropriately in devices in which it is available), (ii) controlling the ventricular rate during SVT with medications or catheter therapy and (iii) preventing the supraventricular rhythm with medications or catheter therapy. The ideal approach depends in part on how well the supraventricular arrhythmia is tolerated. Many patients with significant structural heart disease do not tolerate rapidly conducted atrial fibrillation, even without ICD shocks, so that specific therapy is required. In contrast, sinus tachycardia infrequently causes symptoms, and elimination of ICD therapies by detection enhancements may be preferable to medical or invasive interventions. The role of drug therapy and catheter ablation for the treatment of supraventricular tachycardia is discussed elsewhere.

Device malfunction or “error”

Device malfunction in the present context refers to SVT–VT discriminator misclassification of SVT as VT, oversensing of cardiac or non-cardiac signals, or lead fracture, all of which may lead to inappropriate therapy delivery. Stereotypical mechanisms of failure exist for specific SVT–VT discriminators detection enhancements. These are reviewed in detail.

Interaction of detection zones and detection enhancements

Detection enhancements are designed to differentiate VT from VT when a tachyarrhythmia exceeds the VT detection rate. Arrhythmias with rates below the VT cut-off are not detected by the device, arrhythmias with rates above a programmable “SVT

<table>
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<tr>
<th>Table 10.11 Potential causes of recurrent ventricular tachyarrhythmias</th>
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<tr>
<td>Progressive heart disease, ventricular dysfunction</td>
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<tr>
<td>Thyroid dysfunction (particularly in patients receiving amiodarone)</td>
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<tr>
<td>Electrolyte abnormalities (consider in patients who are taking diuretics or who have acute gastrointestinal illness)</td>
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<tr>
<td>Ischemia (favored by polymorphic ventricular tachycardia and ventricular fibrillation)</td>
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<tr>
<td>Non-compliance with medications or with diet</td>
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<td>Over-the-counter medications, possible drug–drug interactions</td>
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“SVT limit” are presumed ventricular. Detection enhancements may fail to reject SVT leading to inappropriate therapy if: (i) they are not programmed “on”; (ii) the clinical tachycardia rate exceeds the “SVT limit” so that discriminators are not applied; (iii) discriminators erroneously classify SVT as VT. Programming a sufficiently fast SVT limit is critical to reliable rejection of SVT. In clinical trials, ~25% of inappropriate rejection incidents occurred due to an inappropriately treated supraventricular tachycardia.
therapy for SVT is caused by SVT faster than the SVT limit.

The performance of SVT–VT discriminators is linked to the boundaries between detection zones for ventricular arrhythmias. In Boston Scientific ICDs, this link is explicit—discriminators are only applied in the VT zones. In devices up to PRIZM 2®, SVT–VT discriminator may be programmed only to the entire slower VT zone (“VT-1”). Starting with Vitality® (2004), they are programmable to the entirety of either or both VT zones. In St Jude ICDs, the SVT–VT limit is programmable independently within the two VT zones, but not in the VF zone. In Medtronic ICDs, it is programmable independently of VT/VF zone boundaries. However, the performance of the SVT rejection algorithm changes based on whether the arrhythmia falls into the programmed VT or VF zone. In contrast to the VT zone, SVT with an irregular ventricular rate or AV dissociation is not rejected in the VF zone. Thus, in Medtronic devices, at ventricular cycle lengths that fall within the VF detection window and that are slower than the SVT limit (so that discriminators are applied), sinus/atrial tachycardia or 2:1 atrial flutter is rejected as SVT, but conducted atrial fibrillation is not. In our practice, we do not typically apply SVT discriminators in the VF zone. In Medtronic devices the Fast VT window may overlap either the VT zone or the VF zone; in order to reject atrial fibrillation in a Fast VT zone, it must be programmed as “Fast VT via VT.”

**Discriminator misclassification**

**Morphology algorithm errors**

All morphology algorithms share common steps (see Fig. 10.83). (1) Record a template electrogram of baseline rhythm. (2) Construct and store a quantitative representation of this template. (3) Record electrograms from a tachycardia. (4) Time align template and tachycardia electrograms during tachycardia. (5) Construct a quantitative representation of each tachycardia electrogram. (6) Compare the representation of each tachycardia electrogram with that of the baseline rhythm template to determine their degree of morphological similarity. (7) Classify each tachycardia electrogram as a morphology match or non-match with the template. (8) Classify the tachycardia as VT or SVT based on the fraction of
tachycardia electrograms that match the template. Steps 3–8 are performed in real time. Morphology algorithms differ in electrogram source(s), methods of filtering and alignment and details of quantitative representations, but they have common failure modes:

1. **Inaccurate template**
   The template may be inaccurate because the baseline electrogram has changed (e.g., intermittent bundle branch block) or the template was recorded during an abnormal rhythm slower than the VT detection interval (e.g., idioventricular rhythm or bigeminal premature ventricular complexes). If software permits (Medtronic and St Jude ICDs), the template match should be verified initially and during follow-up. Accurate SVT–VT discrimination requires periodic template updates. The template will not update automatically in the absence of an escape rhythm (Medtronic, Boston Scientific) or if any ventricular pacing occurs (St Jude).

2. **Electrogram truncation**
   This both removes electrogram features for analysis and alters the timing of tallest peak. The amplitude scale in Medtronic and St Jude ICDs should be adjusted so that the electrogram used for morphology analysis is 25–75% of the dynamic range (Figs 10.84 and 10.85).

3. **Alignment errors**
   These prevent match between similar electrograms. Since the template is not accurately aligned with the tachycardia, device comparison results in a “mismatch” despite electrogram and template similarity. Their mechanism depends on the method used for electrogram alignment. Accurate alignment in the St Jude algorithm is sensitive to the value of sensing threshold at the onset of the ventricular electrogram, as determined by Automatic Sensitivity Control\^TM. If a template electrogram is acquired at the most sensitive setting of Automatic Sensitivity Control\^TM (either because of a slow sinus rate or after a ventricular paced beat, both of which increase sensitivity), a low-amplitude peak at the onset of the ventricular electrogram may be used for alignment. An identical tachycardia electrogram may be acquired at a sufficiently fast rate that Automatic Sensitivity Control\^TM does not reach its most sensitive value at the onset of the R wave. If this occurs, the low-amplitude peak at the onset of the ventricular electrogram may not be used for alignment. If identical template and tachycardia electrograms are then compared, their representations in the morphology algorithm may not match. Usually, they are assigned morphology match scores of either 0% or 100% (Fig. 10.84B,C). In patients who have dual-chamber ICDs and intact AV conduction, the preferred solution is to acquire the template during atrial pacing at a rate closer to the tachycardia rate. In single-chamber ICDs, solutions may include altering the minimum sensitivity, threshold start or threshold delay.

Medtronic ICDs align electrograms based on their tallest peaks. If an electrogram has two peaks of nearly
Fig. 10.84 Inappropriate detection of SVT by St Jude MD™ morphology algorithm. (A) Electrogram truncation: template electrogram is truncated (arrow) with amplifier range of 9.8 mV. Truncation was corrected by increasing range to 14.4 mV. Inconsistent truncation may prevent SVT morphology from matching template. (B) Interaction of automatic sensitivity control and morphology analysis. Left panel shows stored electrogram of SVT inappropriately detected as VT. Right panel shows programmer strip of validated template in sinus rhythm. Despite identical ventricular electrograms, morphology match scores are 0% in SVT and 100% in sinus rhythm. Slanted line denotes slope of automatic sensitivity control. In sinus rhythm, automatic sensitivity control reaches minimum value before the next ventricular electrogram so that small peak at onset of electrogram (arrow) is used for alignment. In SVT, automatic sensitivity control does not reach minimum, and small peak is not used for alignment. See text for troubleshooting solutions. (C) Programmer strip showing ECG, marker, atrial and ventricular electrograms in sinus rhythm (left) and intermittent atrial-sensed ventricular paced rhythm (right). Insert shows identical ventricular electrogram morphology in two panels recorded seconds apart in time. Morphology match is 100% for consistently conducted sinus beats and 0% for sinus beats following ventricular paced beats. The most likely explanation for this discrepancy is an alignment error; automatic sensitivity control is more sensitive after paced beats than during consistently conducted sinus rhythm. Thus the small peak at the onset of the ventricular electrogram is used for alignment only after paced beats. Failure of morphology match only on postpaced beats does not degrade algorithm performance during tachycardia. Abbreviations as in Figs 10.77 and 10.78. (Reproduced with permission from Swerdlow C, Friedman P. Advanced ICD troubleshooting: Part I. PACE 2005; 28:1322–46., Blackwell Publishing.)
Fig. 10.85 Inappropriate detection of SVT by Medtronic Wavelet™ morphology algorithm. Tip–Coil, near-field electrogram; Coil–Can, far-field electrogram. (A) Electrogram truncation. Electrograms in rapidly conducted atrial fibrillation exceed the maximum amplitude range of 8 mV, resulting in varying degrees of truncation (clipping) of the signal compared with the template. Inappropriate antitachycardia pacing occurs at right. Snapshots below show the last eight electrograms prior to detection at higher resolution. The first of these electrograms shows minimal clipping (66% match). The next five electrograms show more clipping (50–59% match). The last two electrograms are unclipped and are classified as supraventricular (84% and 81% matches). The rhythm is classified as VT because six of the last eight electrograms have <70% match. This problem was corrected by expanding the electrogram scale 16 mV. (B) Myopotential interference. Exercise-induced interference from pectoral myopotentials combined with low-amplitude coil–can electrogram result in inappropriate detection of sinus tachycardia at asterisk. Inset shows sinus template. Snapshots of the coil–can electrogram result in inappropriate detection of sinus tachycardia at asterisk. Inset shows sinus template. Snapshots of the coil–can electrograms show baseline distortion by myopotentials. The tip–coil electrogram shows no interference. This problem was corrected by switching to the tip–coil electrogram for morphology analysis. (C) Alignment error. Sinus template, snapshots of last eight tip–coil electrograms prior to inappropriate detection of sinus tachycardia and detail of last two electrograms are shown. The algorithm time aligns template and tachycardia electrograms using the tallest peak prior to calculation of percent match. This electrogram has two peaks of nearly equal amplitude. In the template, the second peak is taller than the first. In the first seven snapshots, the first peak is as tall or taller than the second peak. The algorithm misaligns template and tachycardia electrograms, resulting in a low match percent even though the overall shape of the tachycardia electrogram is only slightly different from that of the template electrogram. Only the last tachycardia electrogram, in which the second peak is taller than the first peak, is classified as supraventricular (match = 81%). This problem was corrected by changing electrogram source to coil–can. Reprinted with permission from Swerdlow et al. (Swerdlow CD, Brown ML, Lurie K et al. Discrimination of ventricular tachycardia from supraventricular tachycardia by a downloaded wavelet-transform morphology algorithm: a paradigm for development of implantable cardioverter defibrillator detection algorithms. J Cardiovasc Electrophysiol 2002; 13:432–41.) (Continued.)
equal amplitude or such peaks are caused artificially by truncation of large electrograms that exceed the programmed dynamic range, minor variation in their relative amplitudes may result in an alignment error (Fig. 10.85A,C). An alternative source electrogram should be selected.

The Boston Scientific algorithm aligns high-voltage electrograms based on the peak of the rate-sensing electrogram. “Slow” automatic gain control adjusts dynamic range based on the amplitude of the sensed R wave and should minimize alignment errors due to truncation. Presently, data regarding performance of this newest algorithm are limited.91,92

(4) Pectoral myopotential interference
In Medtronic ICDs, this may prevent template matches on the coil-can electrogram if its amplitude is low (Fig. 10.85B). The effect of myopotentials on match percent can be tested by pectoral muscle exercise. Select an alternative source electrogram in these patients, such as distal coil to proximal coil. Pectoral myopotentials are also a possible source of error in Boston Scientific ICDs, which incorporate the high-voltage electrogram in morphology analysis. Pectoral myopotentials are not oversensed in St Jude ICDs, since they use near-field electrograms for morphology determination.

(5) Rate-related aberrancy
Complete bundle-branch aberrancy is rare in ICD patients. If it occurs reproducibly, the template may be recorded during rapid atrial pacing. Automatic template updating should then be deactivated to prevent automatic acquisition of a true baseline template. During rapidly conducted atrial fibrillation, subtle degrees of aberration commonly distort the terminal portion of the electrogram sufficiently that the percent match is less than the nominal threshold. In St Jude ICDs, reducing the fraction of electrograms required to exceed the match threshold from 5 of 8 to 4 of 8 may correct this problem without compromising detection of monomorphic VT. Reducing the match percent has more chance of misclassifying VT.

(6) SVT soon after shocks
ICD detection algorithms typically reclassify the rhythm as sinus and revert to their initial detection mode within a few seconds after a shock, but post-shock distortion of electrogram morphology persists for 30 s to several minutes depending on the electrogram source (see Fig. 10.72). If post-shock VT starts after the rhythm is classified as sinus but before post-shock electrogram distortion dissipates, any morphology algorithm will misclassify SVT as VT. One appropriate (or inappropriate) shock may be followed by a repetitive sequence of inappropriate shocks in which each shock perpetuates post-shock electrogram changes in SVT, resulting in inappropriate detection of VT and the next inappropriate shock. Thus, morphology algorithms provide inadequate SVT–VT discrimination in patients who have VT within a few minutes of a shock.

Stability and onset errors
Stability algorithms utilize the irregularity of RR intervals during atrial fibrillation to distinguish it from ventricular tachycardia, which is typically regular. However, at rates >170 or with organization to atrial flutter, atrial fibrillation becomes regular, and in the presence of membrane active drugs, ventricular tachycardia may
be irregular. Onset algorithms distinguish sinus tachycardia from ventricular tachycardia by the gradual onset of the former and abrupt onset of the latter. However, if VT starts below the detection rate, it may gradually accelerate into the VT zone. Factors that lead to misclassification using the stability and onset algorithms are discussed in the “Stored episode data” section, above.

Atrial sensing errors
Dual-chamber algorithms integrate single-chamber, ventricular discriminators with analysis of the atrial rhythm, with some variation among manufacturers.\(^{33,42,50-48}\) Comparison of the atrial and ventricular rates is a simple and powerful VT-VT discriminator if atrial electrograms can be identified reliably. Because the ventricular rate exceeds the atrial rate in > 90% of VTs in the VT zone of dual-chamber ICDs,\(^{49}\) algorithms that compare atrial and ventricular rates as their first step (Boston Scientific RhythmID \(^{9}\) and St Jude) apply single-chamber discriminators to only < 10% of VTs, reducing the risk that they will misclassify VT as SVT. Dual-chamber algorithms also include measures of stable 1:1 or N:1 AV association (Medtronic, St Jude).

Atrial lead dislodgments, oversensing of far-field R waves or undersensing due to low-amplitude atrial electrograms or atrial blanking periods may cause inaccurate identification of atrial electrograms, resulting in either misclassification of VT as SVT or of SVT as VT. Ideally, the atrial lead should be positioned at implant so that far-field R waves are minimized. Far-field R waves are common because the large mass of the ventricles generates a potential that can be sensed on the atrial lead; if sensed, the ICD must then determine whether the event is a true atrial event or a ventricular event. To date, atrial-lead and sensing problems have limited the degree to which dual-chamber algorithms improve VT-VT discrimination over single-chamber algorithms.\(^{99,100}\) Novel leads have been developed that might improve atrial sensing function by using closely spaced electrodes less prone to sensing far-field R waves, but validation of their role in improving rhythm discrimination is absent at the time of writing.\(^{101,102}\)

To prevent oversensing of far-field R waves, some dual-chamber ICDs have postventricular atrial blanking periods similar to those in pacemakers (Fig. 10.86). Because the blanking period is fixed, the blanked fraction of the cardiac cycle increases with rapid ventricular rates. Atrial undersensing caused by postventricular atrial blanking may cause underestimation of the atrial rate during rapidly conducted atrial flutter or atrial fibrillation, resulting in inappropriate detection of VT, since the device inappropriately calculates that the ventricular rate exceeds the atrial rate (Fig. 10.86, lower panel).\(^{46}\) However, without postventricular atrial blanking, atrial oversensing of far-field R waves may cause overestimation of the atrial rate during tachycardias with 1:1 AV relationship.\(^{46}\) This may cause either inappropriate rejection of VT as SVT if far-field R waves are consistently counted as atrial electrograms or inappropriate detection of SVT as VT if far-field R waves are inconsistently counted (Fig. 10.86).\(^{48}\)

St Jude ICDs provide programmable atrial blanking after sensed ventricular events to individualize the trade-off between oversensing far-field R waves and undersensing atrial electrograms in atrial fibrillation. They also provide programmable atrial sensing Threshold Start and Decay Delay, corresponding to the same features in the ventricular channel. Boston Scientific Vitality \(^{‘}\) ICDs have a 15-ms fixed blanking period followed by a period of auto-adjusting, reduced sensitivity (SmartSense\(^{‘}\)) designed to reject far-field R waves without preventing detection of atrial fibrillation. Older Boston Scientific ICDs have obligatory blanking periods after atrial sensed events, which often cause the atrial rate to be underestimated during atrial fibrillation.\(^{103}\)

Medtronic ICDs provide no atrial blanking after sensed ventricular events to ensure reliable detection of atrial fibrillation. Instead, they reject far-field R waves algorithmically by identifying a specific pattern of atrial and ventricular events that fulfill specific criteria (Fig. 10.87). Because intermittent sensing of far-field R waves or frequent premature complexes may disrupt this pattern, it is preferable to reject far-field R waves after sensed ventricular events by decreasing atrial sensitivity if this can be done without undersensing atrial fibrillation. Atrial sensitivity can be reduced to 0.6 mV with a low risk of undersensing atrial fibrillation. Less sensitive values should be programmed only if the likelihood of rapidly conducted atrial fibrillation is low. Far-field R-wave oversensing that occurs only after paced ventricular events (when auto-adjusting atrial sensitivity is maximum) need not be eliminated to prevent inappropriate detection of SVT as VT.

Programming atrial sensing is also discussed in Chapter 8, “Programming.”

Ventricular oversensing
Inappropriate therapy occurs in the absence of tachy-
cardias because non-physiological or non-arrhythmic, physiological signals are oversensed and detected as arrhythmias. Non-physiological signals are usually extracardiac. Physiological signals may be intracardiac (P, R, or T waves) or extracardiac (myopotentials). Oversensing presents distinctive patterns of stored electrograms (see Figs 10.88 and 10.89).

Implantable defibrillators use dynamic gain or sensing to appropriately sense relatively large ventricular electrogram complexes, while avoiding detection of the subsequent T wave, and maintaining adequate sensitivity to detect low-amplitude fibrillatory electrograms (Fig. 10.90). Since the sensitivity following R-wave detection progressively increases until the maximum sensitivity is reached (Fig. 10.90), oversensing is more likely to occur at slow heart rates. Additionally, due to prolonged blanking periods following pacing, sensitivity (or gain) is rapidly maximized after a paced event, also increasing the risk of oversensing (Fig. 10.91).

**Intracardiac signals**

Ventricular oversensing of physiological intracardiac signals results in two detected ventricular electrograms for each cardiac cycle.

**R-wave oversensing.** R-wave double counting occurs if the duration of the sensing electrogram exceeds the ventricular blanking period of 120–140 ms, which is programmable only in St Jude ICDs. It may be exacerbated by sodium-channel-blocking drugs, since they widen the QRS complex, particularly at high heart rates as use-dependent sodium-channel blockade increases. It is particularly common during VT or conducted supraventricular rhythms in Y-adapted or older biventricular blanking period. Lower panel shows ECG, atrial, and ventricular electrograms from atrial flutter with 2:1 AV conduction. Horizontal bars on ventricular channel denote postventricular atrial blanking, which results in atrial undersensing of alternate atrial flutter electrograms (in boxes). Resultant incorrect calculation of atrial rate causes inappropriate shock (arrow) for atrial flutter because the ICD interprets the ventricular rate to be more than the atrial rate. (Reproduced with permission from Swerdlow C, Friedman P. Advanced ICD troubleshooting: Part I. PACE 2005; 28:1322–46., Blackwell Publishing.)

**Fig. 10.86** Effect of postventricular atrial blanking. Upper panel shows ECG, atrial electrogram, and marker channel during atrial sensed–ventricular paced rhythm. First segment of lower horizontal bar denotes postventricular atrial blanking period (PVAB). Second segment denotes postventricular atrial refractory period (PVARP). FFRW denotes far-field R wave on atrial channel. With a short postventricular atrial blanking period (left), far-field R waves are oversensed. Longer postventricular atrial blanking period (right) prevents oversensing of far-field R waves. Lower horizontal line bar denotes postpacing
Fig. 10.87  Inappropriate therapy of sinus tachycardia caused by oversensing of far-field R waves in a Medtronic ICD. Upper panel shows dual-chamber electrogram and marker channel in sinus tachycardia. Large far-field R waves on the atrial channel are oversensed intermittently (AR markers simultaneous with ventricular electrogram). ST denotes withholding of VT therapy because pattern of sinus tachycardia is confirmed. Intermittent oversensing of far-field R waves results in failure to confirm sinus tachycardia pattern, resulting in inappropriate detection of VT and antitachycardia pacing at right of panel (VT Therapy 1 Burst). Lower left panel (page 483) shows alternating pattern of long (L)-short (S) atrial electrograms with 2:1 AV relationship correctly classified by far-field R wave rule as sinus tachycardia. Consistent sensing of far-field R waves results in algorithmic withholding of VT therapy. Lower right (page 483): interval plot shows intermittent oversensing of far-field R waves in sinus tachycardia resulting in three sequences of inappropriate antitachycardia pacing and a low-energy (delivered 4.9 J) shock. During oversensing of far-field R waves, sum of two sequential, sensed AA intervals equals the RR interval. Consistent oversensing gives “railroad track appearance,” seen on the interval plot at 20–26 s after antitachycardia pacing.
Types of oversensing resulting in inappropriate detection of VT/VF. (A–C) Oversensing of physiological, intracardiac signals. (D–F) Oversensing of extracardiac signals. (A) P-wave oversensing in sinus rhythm from integrated bipolar lead with distal coil near the tricuspid valve. (B) R-wave double counting during conducted AF in a biventricular sensing ICD. (C) T-wave oversensing in patient with low-amplitude R wave (note mV calibration marker). (D) Electromagnetic interference from a power drill has higher amplitude on widely spaced high-voltage electrogram than on closely spaced true bipolar sensing electrogram. (E) Diaphragmatic myopotential oversensing in a patient with an integrated bipolar lead at the RV apex. Note that noise level is constant, but oversensing does not occur until automatic gain control increases the gain sufficiently, about 600μs after the sensed R waves. (F) Lead fracture noise results in intermittent saturation of amplifier range denoted by arrow. RA, right atrium; RV, right ventricular sensing electrocardiogram; HV, high-voltage electrocardiogram. (Reprinted with permission from Swerdlow and Shivkumar. Swerdlow C, Shivkumar K. Implantable cardioverter defibrillators: clinical aspects. In: Zipes DP, Jalife J, eds. Cardiac electrophysiology: from cell to bedside, 4th edn. Philadelphia: W.B. Saunders, 2004:980–93.)
entricular ICDs that use extended bipolar sensing between the tips of the LV and RV electrodes, since these systems merge left ventricular and right ventricular activation into a common sensing channel. R-wave double counting is not common in current ICD systems, as they use right ventricular electrodes alone to determine the ventricular rate during tachyarrhythmias. R-wave double counting and oversensing of far-field P waves result in alternation of ventricular cycle lengths with an isoelectric interval between sensed events, producing a characteristic “railroad track” appearance on interval plots (Fig. 10.81B).

P-wave oversensing. P-wave oversensing may occur if the distal coil of an integrated bipolar lead is close to
Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach

T-wave oversensing. Oversensing of spontaneous T waves may cause inappropriate detection of either VT or VF, depending on the sensed R-T interval and programmed VF detection interval. T-wave oversensing is identified by alternating electrogram morphologies. Intervals may or may not oscillate on interval or counter plots.

Any condition that increases the QT interval or results in a relatively small ventricular electrogram increases the likelihood of T-wave oversensing.

T-wave oversensing by ICDs occurs because ICDs must sense VF electrograms reliably, which often have low amplitudes. To minimize the likelihood of T-wave oversensing, ICDs automatically adjust sensitivity in relation to the amplitude of the preceding R wave (Fig. 10.90). At the end of the blanking period after each sensed ventricular event, sensitivity is decreased based on the amplitude of the sensed R wave (ventricular electrogram) and then decreases (becomes more sensitive) with time to a minimum value. The specific behavior of this adjustment depends on the manufacturer. In Medtronic and St Jude ICDs, this minimum value is the programmed sensitivity. In Boston Scientific ICDs, the minimum value depends both on programmed sensitivity and “slow” Automatic Gain Control, which adjusts the dynamic range of the sensing amplifier.

T-wave oversensing may be divided into three classes: postpacing, large R wave (>3 mV) in spontaneous rhythm and small R wave (<3 mV) in spontaneous rhythm (Fig. 10.92). Postpacing T-wave oversensing can cause inappropriate inhibition of bradycardia pacing or delivery of antitachycardia pacing at the wrong rate. It does not cause inappropriate detection of VT/VF but it may cause inappropriately slow bradycardia or antitachycardia pacing and increment VT or VF counters, increasing the likelihood that non-sustained VT will be detected as VT or VF. It may be corrected by increasing the postpacing ventricular blanking period.
Oversensing of spontaneous T waves often occurs in the setting of low-amplitude R waves because sensitivity and/or amplifier gain is automatically adjusted in relation to the low-amplitude preceding R wave (Fig. 10.93). Further, patients with low-amplitude R waves may require lower minimum sensing thresholds to ensure reliable sensing of VF. T-wave oversensing in the setting of a low-amplitude R wave is a warning that detection of VF may be unreliable and should be assessed at non-invasive electrophysiological study. The ventricular lead should be revised if the safety margin for sensing VF is insufficient. Reprogramming may be able to reduce T-wave oversensing in the setting of low-amplitude R waves, provided detection of VF is reliable; however, lead revision is often necessary either by the addition of a separate pace-sense lead or by repositioning or replacement of the defibrillation lead. If the defibrillation lead is replaced, a true bipolar lead is preferred due to the smaller risk of T-wave oversensing compared with integrated bipolar leads.\textsuperscript{37} If T-wave oversensing occurs in the setting of large R waves, reprogramming to decrease sensitivity (Medtronic, Boston Scientific), or to adjust the “Threshold Start” and “Decay Delay” (SJM) may eliminate the problem, as shown in Figs. 10.93 and 10.94.

Myopotential oversensing may persist for variable fractions of the cardiac cycle. Oversensing usually occurs after long diastolic intervals or after ventricular paced events, when amplifier sensitivity or gain is maximal. It often ends with a sensed R wave, which abruptly reduces sensitivity. In pacemaker-dependent patients, diaphragmatic oversensing causes inhibition of pacing, resulting in persistent oversensing and inappropriate detection of VF (Fig. 10.58). Clinically, this may present as syncope from inhibition of pacing followed by an inappropriate shock. This is an exception
to the rule that syncope prior to a shock indicates an appropriate shock. It is most common in male patients who have integrated bipolar leads in the RV apex with Boston Scientific ICDs that utilize Automatic Gain Control™. Pectoral myopotentials are more prominent on a far-field electrogram that includes the ICD can rather than the near-field electrogram; because ICDs do not use this electrogram for rate-counting, oversensing of pectoral myopotentials does not usually cause inappropriate detection. An exception occurs if the morphology of the far-field electrogram is used for SVT–VT discrimination. In that setting, noisy far-field signals during SVT may be misinterpreted as being different from a clean template signal, or vice versa. Diaphragmatic myopotentials are most prominent on the sensing electrogram. Oversensing these potentials may result in inappropriate shock (Fig. 10.95).

Device–device interactions

Patients with a defibrillator and a separate pacing system are at risk for a number of device–device interactions. Some interactions, such as detection of both atrial and ventricular pacing spikes on the ventricular channel of the ICD, may result in inappropriate therapies. Since up to 50% of patients may have significant pacemaker–ICD interactions that can be avoided if properly identified, testing is warranted. This subject is covered in Chapter 13. Use of separate systems is uncommon and should be avoided.
Lead failure

A number of clinical risk factors for lead failure can be determined by history, review of the patient’s device identification card, device interrogation (which often retrieves information regarding specific leads used) and system radiography. Clinical factors predictive of lead failure include epicardial leads, abdominal pulse generator location, coaxial defibrillation leads, subclavian venous access (as opposed to cephalic access) and dual-chamber (as opposed to single-chamber) ICD systems. In epicardial systems sensing and defibrillation are performed by different leads, eliminating oversens-

**Fig. 10.95** Oversensing leading to inappropriate detection of ventricular fibrillation (VF), transient asystole, and shock delivery. The patient was performing deep breathing exercises when she felt dizzy and received a shock. These are stored episode data from the patient whose real-time telemetry findings are shown in Fig. 10.68. (B) From top to bottom are the atrial, ventricular near-field (rate) and ventricular far-field (shock) electrograms. (A) Diaphragmatic myopotential oversensing leads to inappropriate detection of VF. Pacing is withheld during ventricular tachycardia (VT)/VF detection, leading to asystole (note that QRS complexes are absent on the far-field electrogram [bottom tracing], but noise is present on the near-field electrogram [middle tracing]). (B) Oversensing resolves and pacing resumes (at VP 75), presumably because the patient stopped the deep breathing exercises after the dizzy spell. During reconfirmation (at the second “Chrg” marker), pacing is withheld to determine whether VT or VF is still present. Since this patient has high-grade atrioventricular block, asystole recurs and is shocked by the algorithm, which inappropriately assumes that asystole represents fine VF. Newer versions of this algorithm do not shock for asystole, but rather provide pacing support.
ing as an early warning for failure of defibrillation leads. Thus, periodic assessment of shocking lead impedance is essential. At present, epicardial systems are used only in patients with a univentricular heart, significant right to left shunt or mechanical tricuspid valve. Subcutaneous patches, which have similar construction to epicardial patches and similar high failure rates, also warrant careful observation.\(^\text{42,109}\) Abdominal pulse generator placement, independent of epicardial or transvenous approach, may cause lead stress associated with tunneling, as well as mechanical friction of the pulse generator against the lead.\(^\text{4,46}\) Cardiac resynchronization leads have a higher dislodgment rate than other electrodes.\(^\text{110}\) Adapters increase the number of mechanical connections and stress points and may increase the risk of system malfunction. A loose setscrew (in the header or an adapter) can mimic lead fracture, with make–break contact noise and elevated impedance.

Coaxial leads have higher failure rates than multilumen leads, in which conductors are longitudinally arrayed along the length of the lead body (Fig. 10.96). Medtronic Transvene coaxial leads (models 6936 and 6966) in particular warrant careful observation.\(^\text{47,61}\) In coaxial leads, failure occurs due to degradation of a middle insulation layer composed of 80A polyurethane, which is prone to metal ion oxidation. A drop in the ring-to-coil impedance is an early marker of this failure.\(^\text{51}\) Oversensing of characteristic non-physiological intervals caused by lead fracture noise increments the Sensing Integrity Counter. Use of ring-to-coil impedance, non-sustained episode log, and Sensing Integrity Counter may permit identification of early failure before oversensing of electrical noise causes inappropriate shocks (Fig. 10.97). A signature presentation of this type of lead failure is oversensing of electrical noise following shocks (Fig. 10.56).\(^\text{51}\)

**Electromagnetic interference**

EMI can result in electrical signals on ICD leads that are recorded on the sensing channel, resulting in suppression of pacing or inappropriate detection of VF. The signature of oversensing due to extracardiac signals is the replacement of the isoelectric baseline with high-frequency noise that does not have a constant relationship to the cardiac cycle.\(^\text{49}\) An example is shown in Fig. 10.88, and this is reviewed extensively in Chapter 12.

**Phantom shocks**

Some patients report experiencing shocks, at times associated with flashes of light, myoclonic jerks, verbal outcries, and chest soreness, without actual discharge of the defibrillator, as confirmed by device interrogation.\(^\text{111}\) This phenomenon usually occurs in the twilight preceding sleep, more commonly in patients with previous ICD discharges, and may represent anxiety or maladjustment to the defibrillator. Patients experiencing these symptoms should be reassured, and if symptoms persist, psychiatric evaluation should be considered.

**Delayed, absent, or ineffective therapy**

Absent or significantly delayed ICD therapy is rare, based on a series of observational studies demonstrating nearly uniform device effectiveness, analysis of post-mortem stored electrograms after sudden cardiac death in ICD patients,\(^\text{112}\) and prospective randomized trials showing reduced mortality compared with the best medical therapy in high-risk patients.\(^\text{113,114}\) This is particularly true when an appropriate follow-up program is used, which often detects important malfunctions before they result in clinical events. Nonetheless,
these serious complications may occur. Differential diagnoses are listed in Table 10.12. Patients with this problem should be carefully evaluated by the approach outlined earlier in this chapter. Often, this examination identifies the cause of the malfunction. For example, interrogation may show that the device is programmed “off,” radiography or impedance values may reveal a lead fracture, or the documented VT may be slower than the programmed detection cut-off. If the cause is not evident, electrophysiological study should be done through the device to assess sensing, detection and therapy delivery.

Inactivated implantable cardioverter-defibrillator

An ICD may be inactivated by being programmed “off”—e.g., preceding surgery to avoid overdetection of electrocautery—or, in some models, by prolonged contact with an external magnet. One study has reported an unexplained 11% annual incidence of transient suspension of detection.115 As inadvertent deactivation usually occurs in the hospital environment, close communication with other physicians and with the patient ensures that devices turned off intentionally for surgical interventions are reactivated before the patient is dismissed from a monitored hospital bed.116 Boston Scientific defibrillators offer a “Change Tachy Mode with Magnet” feature that enables the mode of the device to be changed by holding a magnet over it for at least 30 s. Initially, tones synchronous with R waves are heard (while the device is in the monitor plus therapy mode), and they become continuous when the device is off. Since environmental magnets (e.g., in stereo speakers, motors) have in rare instances inactivated devices, with life-threatening consequences, we routinely program this feature “off.” This prevents environmental
Undersensing of ventricular arrhythmias

Undersensing occurs when the ICD does not reliably sense electrograms representing true cardiac electrical activation. The cause may be low amplitude electrograms, parameter settings, rapidly varying electrograms during VF, post-shock tissue effects, drug and metabolic effects, lead malfunction, or device–device interactions (Table 10.12). In contrast, underdetection occurs when, despite appropriate sensing, VT/VF is not classified correctly (because the rate is too slow or specificity algorithms inappropriately determine that the rhythm is not ventricular) (Table 10.12).

The factors that can lead to R-wave diminution in pacemakers also apply to defibrillators: local inflammation and fibrosis, myocardial infarction at the lead tip site and medication effect. Additionally, electrograms in defibrillators may be further diminished after shocks from the effect of the high-voltage gradient on the nearby myocardium117 (Fig. 10.98). Although postshock electrogram diminution with failed redetection was a problem in some older ICD systems that included integrated bipolar leads with a distal coil near the lead tip, the issue is no longer significant in modern systems.117–121 There appears to be a correlation between the normal rhythm R wave and sensed electrogram amplitude during VF; clinically significant undersensing of VF is rare in modern ICD systems if the baseline R-wave amplitude is ≥5–7 mV.122

In the setting of a small R wave and uncertainty as to VF detection, redetection after a failed shock should be assessed at electrophysiological study. If, at sensitive program settings, impaired sensing delays detection or T-wave oversensing occurs, a separate sensing

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Table 10.12 Causes of absence or delay in effective implantable cardioverter-defibrillator (ICD) therapy with documented ventricular tachycardia (VT) or ventricular fibrillation (VF)

<table>
<thead>
<tr>
<th>Inactivated ICD</th>
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<tbody>
<tr>
<td>Programmed off</td>
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<tr>
<td>Sustained (possibly inadvertent) contact with an external magnet</td>
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<table>
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<tr>
<th>Undersensing of ventricular electrogram</th>
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<tbody>
<tr>
<td>Diminished amplitude of sensed ventricular electrogram (e.g., lead–tissue interface fibrosis)</td>
</tr>
<tr>
<td>Parameter settings (sensitivity, time to detection, others)</td>
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<tr>
<td>Drug and metabolic effects</td>
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<tr>
<td>Postshock electrogram diminution</td>
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<tr>
<td>Rapid electrogram amplitude fluctuation during VF</td>
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<tr>
<td>Lead malfunction or displacement</td>
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<tr>
<td>Generator malfunction</td>
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<tr>
<td>Device–device interaction (VF not detected because of pacing artifacts)</td>
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</tbody>
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<table>
<thead>
<tr>
<th>Underdetection</th>
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<tbody>
<tr>
<td>VT below detection cut-off rate</td>
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<tr>
<td>VT therapy withheld because of programmed specificity criteria</td>
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<tr>
<td>Onset</td>
</tr>
<tr>
<td>Stability</td>
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<tr>
<td>Electrogram width or template matching</td>
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<tr>
<td>Dual-chamber criteria</td>
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<tr>
<td>Algorithm “error” in rhythm interpretation</td>
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<tr>
<td>Detection in inappropriate zone (e.g., antitachycardia pacing for VF)</td>
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<tr>
<td>Pacemaker–ICD interaction</td>
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<tr>
<td>Intra-device interaction</td>
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<table>
<thead>
<tr>
<th>Mechanical failure preventing detection or delivery of therapy</th>
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<tbody>
<tr>
<td>Lead fracture or failure</td>
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<td>Poor lead connection in header</td>
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<table>
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<tr>
<th>Ineffective delivered therapy</th>
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<tbody>
<tr>
<td>Ineffective ATP</td>
</tr>
<tr>
<td>Pacing threshold increase (medications, metabolic)</td>
</tr>
<tr>
<td>Defibrillation threshold increase (medications or mechanical)</td>
</tr>
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</table>
lead should be implanted or the present lead should be repositioned or replaced. It is important to measure the R-wave amplitude on the near-field (rate sensing or pace-sense) electrogram, because this is the signal used by the device for sensing.

Presently, the most common causes ofVF undersensing are drug or hyperkalemic effects that slow VF into the VT zone, ischemia, and rapidly-varying electrogram amplitude.\(^{123,124}\) Undersensing due to hyperkalemia is shown in Fig. 10.99. ICDs that adjust dynamic range based on the amplitude of the sensed R wave (Boston Scientific) may be most vulnerable to the latter, extremely rare problem.\(^{123}\) Prolonged ischemia from sustained VT slower than the VT detection interval may cause undersensing of VF. Lead, connector or generator malfunction may also present as undersensing.

Undersensing may also occur because of lead failure or insulation breakdown in both epicardial and transvenous systems.\(^{12}\) Thus, system radiography, analysis of stored episodes, real-time telemetry with provocational maneuvers and assessment of pacing threshold and impedance may be diagnostic. Similarly, since defibrillators decrease sensitivity (or gain) immediately after a detected QRS to avoid T-wave oversensing, the large pacing spikes in patients with a separate pacing system may cause device–device interactions that mimic QRS complexes and result in significant undersensing of continuing VF. Electrophysiological testing to assess for device–device interactions is required in the patient with two separate systems and untreated ventricular tachyarrhythmias.\(^{40}\)

Undersensing may lead to detection of a tachyarrhythmia in an inappropriate zone. This may result in ineffective therapy, such as ATP delivery during VF (Fig. 10.100).

**Underdetection of ventricular arrhythmias**

VT slower than the programmed detection interval is potentially fatal in patients with severe LV dysfunction or ischemia. But in most ICD patients, VT with cycle lengths > 400–450 ms are tolerated well, while repeated inappropriate therapies are not. Most SVT–VT discrimination algorithms deliver less inappropriate therapies if the VT detection interval is programmed to a faster cycle length so that fewer SVTs are evaluated. To prevent underdetection of irregular VT, the VT detection interval should be set at least 40–50 ms longer than the slowest predicted VT for consecutive-interval counting (Medtronic devices in VT zone) and 30–40 ms longer for X-of-Y or interval + interval-average counting. (Boston Scientific or St Jude Medical). Consecutive interval counters require a programmed number of intervals faster than the VT cut-off rate to detect VT; a single slower event resets the counter to zero (Fig. 10.101). In X-of-Y counters, if a programmed proportion of intervals are faster than the detection rate, VT is detected, allowing greater tolerance for variability.

A long VT detection cycle length may be important in patients with advanced heart failure, in whom failure to detect slow VT may be catastrophic.\(^{103}\)
Cardiac Pacing, Defibrillation and Resynchronization: A Clinical Approach

This is particularly important in patients with known slow VTs. The VT detection cycle length should be increased if antiarrhythmic drug therapy is initiated, particularly with amiodarone or a sodium-channel blocking (Type 1) drug. It may be prudent to measure the cycle length of induced VT at electrophysiological testing after initiation of drug therapy, with the proviso that spontaneous VT often is slower than induced VT.

If therapy is not programmed for slow VT, the slowest rate zone may be programmed as a “monitor-only” zone with detection “on” and therapies “off.” However, in Boston Scientific, St Jude, and older Medtronic ICDs (up until Marquis), interactions between the counters in the monitor-only zone and the next zone may decrease the number of intervals required for detection in the next zone and restrict use of SVT–VT discriminators in the faster zone. Because every manufacturer

Fig. 10.99 Failure to capture, failure to sense, and underdetection of VF due to hyperkalemia. The top panel shows a surface ECG in a patient with a potassium level of 6.9 mmol/l with atrial flutter with variable conduction. Note that the first five spikes seen on the strip do not capture the ventricle. Also note that the second to last QRS complex (asterisk) is not sensed. The rhythm later degenerates to VF. The middle panel shows intracardiac recordings during VF. From top to bottom are atrial electrograms, ventricular electrograms, device markers and sensed intervals. Note the atrial bradycardia during VF. Following the shock (HV), VF nearly immediately recurs but is undersensed (as indicated by “return to sinus”). The bottom panel shows the surface telemetry and rescue shock during VF, confirming the intracardiac diagnosis.

(Fig. 10.75). This is particularly important in patients with known slow VTs. The VT detection cycle length should be increased if antiarrhythmic drug therapy is initiated, particularly with amiodarone or a sodium-channel blocking (Type 1) drug. It may be prudent to measure the cycle length of induced VT at electrophysiological testing after initiation of drug therapy, with the proviso that spontaneous VT often is slower than induced VT.
has a technique for dealing with “competing” counters, to avoid delayed or failed classification of true VT when rates overlap zones, events in a “monitor” only zone may actually increment a “combined count” counter in a fast zone and result in an inappropriate therapy even though the “formal” programmed detection criteria are never met. Present Medtronic ICDs (Marquis forward) provide the option of programmable independent monitor-only zones that avoids these limitations. Events in the designated “monitor” zone do not increment the “combined count” counter.

In St. Jude ICDs, monitoring zones nominally detect a monitored tachycardia if 12 intervals meet the detection rate for the monitoring zone. Once a monitored tachycardia is detected, bins in the VF zones are not cleared until “sinus” rhythm is redetected (nominally five consecutive intervals binned as sinus). Thus, all bins remain open for the duration of the monitored rhythm and the cumulative effect of 12 shorter intervals associated with PVCs may be detected as VF.128

SVT–VT discriminators
SVT–VT discriminators may prevent or delay therapy if they misclassify VT or VF as SVT.66,67,129,130 Discriminators that re-evaluate the rhythm diagnosis during an ongoing tachycardia, such as stability and most dual-chamber algorithms, reduce the risk of preventing detection compared with discriminators that withhold therapy if the rhythm is not classified correctly by the initial evaluation, such as onset or chamber of origin.

Single-chamber discriminators
Although spontaneous VT often begins with irregular R–R intervals, stability algorithms classify it as VT as soon as R–R intervals regularize, even transiently. Thus they rarely prevent detection of VT (~0.5%). Morphology discriminators also re-evaluate rhythm diagnosis during ongoing tachycardias. However, if they misclassify monomorphic VT initially, the error usually persists and prevents detection for the duration of the tachycardia. The St Jude morphology algorithm, which analyzes only the rate-sensing electrode, continuously misclassifies 5–10% of monomorphic VTs as SVT, but when it is restricted to the V = A and V < A rate branches, only 1% of VTs are misclassified.67 The Medtronic morphology algorithm, which usually analyzes high-voltage electrograms, misclassifies 1–2% of VTs as SVTs.131 Limited data regarding the Boston Scientific-Guidant algorithm report no episodes of VT misclassified as SVT.119 In contrast, the onset discriminator uniformly misclassi-
Fig. 10.101 Delayed detection of ventricular tachycardia (VT) due to an irregular VT with intervals falling outside the detection zone. (A) Trend report from a Medtronic 7271 dual-chamber defibrillator. The atrial cycle length (approximately 660 ms, open boxes) is stable, whereas VT with unusually variable VV intervals continues. At times −15, −12, −11 and −7, intervals > 500 ms occur. These are outside the tachycardia zone and reset the VT counter, delaying detection. After detection, a shock is delivered (9.6 J at +3 s), restoring sinus rhythm. Note the overlap of the VV and AA intervals after the shock. (B) Electrograms from the episode shown in (A). From top to bottom are the atrial electrogram, ventricular electrogram and atrial (upgoing) and ventricular (downgoing) markers. The ventricular rate is faster than the atrial rate, consistent with VT. However, note the long intervals (e.g., VS 550) and the shifting ventricular morphology. In the Medtronic algorithm, a single long interval outside the VT zone resets the VT counter to zero. This often prevents detection of atrial fibrillation, but when VT is irregular, it may delay its detection.
fies VT if it either accelerates gradually across the sinus–VT zone boundary or occurs during SVT with cycle length in the VT zone.

**Dual-chamber discriminators**

Discriminators that withhold VT therapy for 1:1 arrhythmias run the risk of withholding therapy if the atrial lead dislocates to the ventricle. These should not be programmed until the atrial lead is stable (Medtronic 1:1 VT rule and St Jude 1:1 Rate Branch without additional discriminators).

VT with 1:1 VA conduction is a difficult diagnosis for dual-chamber algorithms. Boston Scientific’s older Atrial View® misclassifies it as SVT unless onset is abrupt. Medtronic’s PR Logic® misclassifies it as VT in the < 1% of cases in which the VA interval is long and constant. Boston Scientific’s Rhythm ID® and St Jude’s Branch® misclassify it if a morphology match occurs.67,132 Single-chamber stability may be programmed in Medtronic dual-chamber ICDs to reject atrial fibrillation during redetection. In this case, stability is applied before the dual-chamber algorithm. Because stability responds to irregular rhythms by resetting the VT counter to zero, rhythms rejected by stability are not evaluated by the dual-chamber algorithm. Thus, stability may delay detection of VT even if the ventricular rate is greater than the atrial rate.

Sustained-duration override features deliver therapy if an arrhythmia satisfies the ventricular rate criterion for a long programmed duration even if discriminators indicate SVT. These include Sustained Rate Duration (Boston Scientific), Extended High Rate (St Jude), Maximum Time to Diagnosis (St Jude) or High Rate Timeout (Medtronic). The premise is that VT will continue to satisfy the rate criterion for the programmed duration while the ventricular rate during transient sinus tachycardia or atrial fibrillation will decrease below the VT rate boundary. The limitation is delivery of inappropriate therapy when SVT exceeds the programmed duration. The incidence of inappropriate detection of SVT is ~10% at 1 min and 3% at 3 min.136,137 Additionally, the SVT limit should be set to prevent clinically significant delay in detection of hemodynamically unstable VT.

**Mechanical, pulse generator, or interaction “failure” preventing delivery of therapy**

Isolated failure of shocking leads may prevent delivery of effective therapy despite appropriate detection. This phenomenon in the past occurred predominantly in epicardial systems, in which the leads for sensing and for shocking are mechanically distinct.46,132 Low-voltage impedance tests (discussed above) permit detection of shocking lead failure before clinically manifest. Infrequently, mechanical connections (e.g., an improperly inserted lead pin) can result in non-delivery of a shock; this defect is also detected with current low-voltage impedance tests.

Pulse generator failures are a rare cause of failure to deliver therapy. They may be random or systematic. The root causes of systematic failures include internal battery shorting46,134 and electrical overstress failures of high-voltage components. Electrical overstress failure is a term applied to semiconductor failure caused by application of extreme voltages or currents for a sufficient duration to cause a transistor to fail catastrophically.133 These failures may occur in the sealed components of the pulse generator or header.53,96,87,154,155

**Pacemaker–ICD interactions**

Interactions between ICDs and separate pacemakers have become rare since ICDs incorporated dual-chamber bradycardia pacing in the late 1990s. Nevertheless, a few such combined systems have not been revised at ICD generator change due to vascular access problems or for other reasons. The multiple potential interactions have been reviewed and testing protocols to detect them have been developed.136–138 The principal interaction that may delay or prevent ICD therapy occurs when high-amplitude pacemaker stimulus artifacts are oversensed by the ICD. If this occurs during VF, automatic adjustment of sensing threshold and/or gain may cause repetitive undersensing of VF electrograms.

**Intradevice interactions**

Today “intradevice interactions,” in which bradycardia pacing features of dual-chamber ICDs interact with and may impair detection of VT or VF, pose a greater challenge than pacemaker–ICD interactions.139 During high-rate, dual-chamber pacing, sensing may be restricted to short periods of the cardiac cycle because of the combined effects of blanking periods after ventricular pacing and cross-chamber ventricular blanking after atrial paced events, which is needed to avoid crosstalk. If dual-chamber pacemaker timing cycles blank a sufficient fraction of the cardiac cycle, systematic undersensing of VT or VF may occur. When pacing-scheduled blanking events occur at intervals
that are multiples of a VT cycle length, ventricular complexes are repeatedly undersensed, delaying or preventing detection (see Fig. 10.102).

Intradevice interactions have been reported most frequently with the use of the Rate Smoothing® algorithm in Boston Scientific ICDs. This algorithm prevents sudden changes in ventricular rate by pacing both the atrium and ventricle at intervals based on the preceding (baseline) R–R interval and may prevent sensing of VT/VF in some patients due to repetitive post-pace blanking. The algorithm applies ratesmoothing to baseline intervals independent of their cycle length, including intervals in the VT or VF zone. Intradevice interactions that result in delayed or absent detection of VT/VF are most common and most dangerous when VT is fast (> 220 bpm). The parameter interrelationships that result in delayed or absent detection of VT/VF are complex and difficult to predict. Generally, aggressive rate smoothing (a small allowable percentage change in R–R intervals), a high upper pacing rate, and a long and fixed AV interval favor undersensing and should be avoided. Most reports of intradevice interactions occurred with programmed parameters that generated programmer warnings. If rate smoothing is required, the AV delay should be dynamic, and parameter combinations that result in warnings should be avoided. This programming reduces, but does not eliminate, the risk of undersensing when Rate Smoothing is used.

A prospective clinical trial (VAST) used Rate Smooth-

![Fig. 10.102 Failure to detect VT due to an intradvice interaction. The rate-smoothing algorithm introduced atrial and ventricular pacing complexes with associated blanking periods that prevented detection of VT during post-implant testing. An external rescue shock was required. Shown from top to bottom are surface ECG, atrial electrogram, ventricular electrogram, and event markers. At top, VT is induced by programmed electrical stimulation with drive cycle length 350 ms and premature stimuli at 270, 250 and 230 ms (intervals labeled next to event markers). The first sensed ventricular event occurs 448 ms after the pacing drive ("PVC 448"). The rate smoothing algorithm drives pacing to prevent a pause after the "premature ventricular complex" (PVC), labeled AP↓1638. A ventricular-paced event does not follow the first AP↓ because a ventricular event is sensed (VT 415). Subsequent rate smoothing generated atrial and ventricular pacing pulses (indicated by AP↓ and VP↓ markers, respectively). The resultant post-pacing blanking periods are shown in the figure as horizontal bars. PAB denotes cross chamber (post-atrial-pace) ventricular blanking period. VBP denotes same-chamber (post-ventricular pace) blanking period. Together, they prevent approximately four of every six VT complexes from being sensed. Since the VT counter must accumulate eight out of 10 consecutive complexes in the VT zone for detection of VT to occur, VT is not detected.](image)
CHAPTER 10  Troubleshooting

...ing of 12% in a broad population of ICD patients and found that the algorithm was safe, but did not prevent VT/VF episodes by preventing rhythm irregularities, as had been hypothesized. The authors concluded that the algorithm should not be routinely programmed “on”, but that it may have a niche role in patients with long QT syndrome or symptoms due to ectopy.

Ineffective delivered therapy

Antitachycardia pacing (ATP) terminates 75–85% of VT in ICD patients, depending on the VT rate. Reduction in effectiveness of ATP may occur because the paced impulses cannot reach critical arrhythmogenic substrate as a result of medication or metabolic effects on conduction, emergence of new arrhythmia circuits, a change in the substrate, or induction of a new VT by pacing. Medication or metabolic effects on pacing thresholds may prevent capture during ATP. If repeated sequences of ATP fail to terminate VT, the device should be reprogrammed, and if new sequences of ATP are assigned, electrophysiological study can help assess ATP efficacy. In Medtronic devices, “Smart Mode” disables ATP therapies that have been unsuccessful in four consecutive episodes, to shorten the time to a therapy that may be effective.

A few patient-related causes that would otherwise require operative revision may be resolved by programming shock pathway and waveform parameters. For ICDs with fixed-tilt waveforms, waveform duration depends on output capacitance and pathway resistance. ICDs with programmable waveform duration or tilt (St Jude) permit optimization of waveform parameters independent of pathway resistance. Use of non-invasive device testing can help assess ATP efficacy. 

Defibrillation thresholds may spontaneously vary over time; in patients with implantation defibrillation thresholds < 15 J, failure to defibrillate is rare with current-generation devices (with maximum output ≥ 30 J). Importantly, medications may alter the defibrillation threshold. In particular, long-term oral administration of amiodarone increases the defibrillation threshold, so that reassessment is helpful in patients with borderline defibrillation margins. However, this is probably not necessary in patients with defibrillation thresholds < 15 J.

ICD system-related factors

Shocks may fail from insufficient programmed shock strength, battery depletion, component failure, epicardial patch crumpling, transvenous lead dislodgement, lead failure, device–lead connection failures or a pneumothorax, which adversely affects energy delivery across the myocardium (Table 10.13 and Figs 10.103 and 10.104). Device interrogation and chest radiographs diagnose these conditions. In evaluating data from the ICD interrogation, attention should be paid to arrhythmia duration, charge time, battery voltage, the relationship between programmed and delivered shock strength, impedance...
of the high-voltage lead at the time of shock, and trend plots of lead impedance. Prolonged episodes may increase the shock strength required to convert VT or VF. They may be caused by delayed detection and/or prolonged charge times.

ICDs misclassify effective therapy as ineffective if VT/VF recurs before the ICD detects post-therapy sinus rhythm. Decreasing the duration for redetection of sinus rhythm (St Jude) may correct this classification error. However, this type of misclassification usually does not constitute a clinical problem, while post-shock detection of non-sustained VT does, as it leads to additional (unnecessary) shocks.

<table>
<thead>
<tr>
<th>Troubleshooting cardiac resynchronization devices</th>
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<tbody>
<tr>
<td>In selected heart failure patients, combined ICD and cardiac resynchronization pacing improves exercise tolerance, reduces heart failure hospitalizations, and decreases mortality. The left ventricular electrode, most commonly placed via the coronary sinus and its tributaries, has a higher dislodgment rate than other electrodes (see Fig. 10.65). The two widely spaced ventricular leads in resynchronization ICDs and the high frequency of pacing employed in resynchronization introduce novel intradevice interactions between resynchronization pacing and detection of VT/VF. The troubleshooting implications are reviewed here; optimizing pacing parameters for hemodynamic benefit is covered in Chapter 2.</td>
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<table>
<thead>
<tr>
<th>Ventricular sensing problems</th>
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<tbody>
<tr>
<td>Sensing problems in CRT systems may lead to insufficient delivery of therapy, or to inappropriate shocks. The first commercially available dedicated system (Boston Scientific Contak CD) sensed between the LV and RV electrodes to determine R–R intervals. In contrast to standard ICDs and subsequent CRT ICDs which utilize closely spaced electrodes in the right ventricle alone to determine R–R interval timing, this “extended bipolar sensing” configuration “merges” events sensed in the LV or RV into a single recording channel. Significant intraventricular delay, present in most patients who receive CRT, can result in double counting of single ventricular complex. During pacing, RV and LV depolarizations are synchronized and refractory periods prolonged, preventing double counting. Any event that inhibits ventricular pacing even transiently</td>
</tr>
</tbody>
</table>

![Fig. 10.103 Pneumothorax, as seen in this chest radiograph (arrows), can result in ineffective defibrillation. Also present is a pneumopericardium (arrowheads).](image)
Troubleshooting

(e.g., premature complex, conducted sinus tachycardia or SVT) may lead to double counting and inappropriate therapy (Fig. 10.105). Use of pacemaker algorithms, such as those designed to terminate pacemaker-mediated tachycardia, may promote inappropriate shocks by temporarily suspending ventricular pacing, permitting double counting to occur (Fig. 10.105).

R-wave double-counting may result in shocks for SVT slower than the programmed VT detection interval due to the short interval between the two events sensed for a given R wave. Because every other “R–R” interval in any conducted rhythm increments the VF counter, all detected VT or SVT episodes are classified as VF and treated with shocks, regardless of cycle length. Because there are two detected intervals for each ventricular electrogram, transient non-sustained tachycardias may satisfy the VF detection criterion, resulting in aborted shocks.

Extended bipolar ventricular sensing also increases the risk of oversensing P waves and extracardiac signals. Oversensing of atrial arrhythmias on the ventricular channel has resulted in inappropriate shocks in adapted cardiac resynchronization systems despite a slow ventricular rate. Oversensing may also inhibit pacing, preventing resynchronization therapy of heart failure, or asystole, and this is most likely to occur when the LV lead dislodges into the main body of the coronary sinus, where (left) atrial oversensing is most likely to occur. All current biventricular systems utilize local RV bipolar sensing to determine R–R intervals to minimize the risk of double counting and far-field oversensing. Although less common in current systems that utilize local RV bipolar sensing, ventricular oversensing still occurs as with any ICD, leading to inhibited pacing output and spurious detections. Diagnosis and management of oversensing in ICDs is discussed in earlier sections of this chapter.

Atrial sensing problems can also impair resynchronization (Table 10.14). Atrial undersensing results in a loss of ventricular tracking. This permits intrinsic ventricular activation, which may inhibit ventricular pacing. Atrial oversensing may lead to inappropriate mode switch and loss of atrial synchronous ventricular pacing.

**Pacing problems**

In order to provide effective cardiac resynchronization, a sufficient dose of biventricular pacing must be delivered.
To achieve this, the frequency and hemodynamic effectiveness of pacing must both be optimized (Table 10.14). Hemodynamic optimization of pacing is covered in Chapter 2; assessment of pacing is covered here. A practical goal is to resynchronize 90% of R–R intervals. Figure 10.106 shows a systematic approach to confirming resynchronization. Loss of resynchronization occurs if ventricular pacing is interrupted or LV capture fails. If LV capture is intact, the most common causes of failure to deliver resynchronization are intrinsic AV conduction due to a long programmed AV delay or due to conduction of rapid atrial rhythms. Other causes include frequent premature ventricular complexes, sensing malfunction (see above), programming considerations and/or specific pacing algorithms (see below). Conducted atrial fibrillation may reduce the effectiveness of CRT. Interruption of cardiac resynchronization pacing is common, but can usually be restored when properly diagnosed. Restoration of resynchronization may require reprogramming or surgical lead revision.

**LV threshold and RV anodal capture**

Determining whether the LV lead is capturing the LV can be difficult. Even if they use RV sensing, most ICDs use “dual cathodal” extended bipolar pacing, in which the LV and RV tip electrodes serve as the combined cathode and the RV coil or RV ring as the common anode. Some ICDs permit true bipolar pacing in both the LV and RV (Fig. 10.107). In early-generation devices extended bipolar pacing systems applied the same voltage to the commonly wired LV and RV cathodes. As the local LV and RV thresholds may differ, the pacing pulse may be suprathreshold in one chamber and subthreshold in the other, leading to loss of resynchronization. In these ICDs, a change of ventricular-electrogram morphology during a threshold test indicates loss of capture in one chamber; the corresponding change in the surface ECG indicates which chamber (see below). Current systems permit independent programming of RV and LV outputs, facilitating determination of the RV and LV threshold.

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**Fig. 10.105** Multiple shocks in a patient with a cardiac resynchronization ICD with extended bipolar sensing (Contak CTM) due to double counting initiated by a pacemaker-mediated tachycardia prevention algorithm. Electrograms from top to bottom are atrial, ventricular near-field and ventricular far-field. The initial rhythm is sinus tachycardia, with P synchronous pacing at the maximum tracking rate (“VP-MT”). This algorithm periodically extends the post-ventricular atrial refractory period during upper-rate limit pacing to abort a pacemaker-mediated tachycardia (indicated by “PMT-B”). The following atrial-sensed event falls in the extended post-ventricular atrial refractory period (indicated by the parenthesis, “AS”) and is therefore not tracked. Cessation of ventricular pacing permits double counting of the intrinsic-wide QRS complex by extended bipolar sensing, leading to inappropriate detection of VF. VS, ventricular sense in sinus zone. (Reproduced with permission from Blackwell Publishing.)
Pacing leads are typically intended to capture at the cathode (negative terminal). "Anodal capture" occurs when the delivered voltage captures at the RV anode either in isolation or in conjunction with the RV or the LV cathode. If anodal RV capture is mistaken for LV capture, the pacing output may be programmed to a subthreshold value in the LV, resulting in loss of cardiac resynchronization (Figs 10.108 and 10.109). Anodal capture is much more common if the RV anode is a ring electrode (true bipolar pacing), which has a small surface area (high current density) that may be in direct contact with endocardium, than if the RV anode is a defibrillation coil (integrated-bipolar pacing), which has a large surface area (low current density). Thus,

### Table 10.14 Approach to non-responders to cardiac resynchronization

<table>
<thead>
<tr>
<th>Problem</th>
<th>Mechanism</th>
<th>Solution</th>
</tr>
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<tbody>
<tr>
<td>Resynchronization is not delivered (pacing &lt; 90%)</td>
<td>Loss of P synchronous pacing and conduction of intrinsic ventricular complexes</td>
<td>Increase atrial sensitivity or reposition atrial lead. If function, shorten PVARP, increase upper tracking limit, turn off PMT algorithm</td>
</tr>
<tr>
<td></td>
<td>Atrial undersense</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Atrial oversensing</td>
<td>Far-field R waves cause inappropriate mode switch and loss of atrial tracking</td>
</tr>
<tr>
<td></td>
<td>Ventricular oversensing</td>
<td>Ventricular pacing is inhibited</td>
</tr>
<tr>
<td></td>
<td>Algorithmic inhibition of ventricular pacing</td>
<td>Algorithms such as rate smoothing prevent tracking abrupt increases in atrial rate</td>
</tr>
<tr>
<td>Resynchronization is not delivered (pacing &gt; 90%, but no LV capture)</td>
<td>LV capture with slow exit from LV pacing site</td>
<td>Due to slow conduction of tissue around LV lead, most of ventricle activated from RV</td>
</tr>
<tr>
<td>Non-optimal vector</td>
<td>Capture may be present from some LV vectors and not others; anodal capture may be present</td>
<td>Recheck threshold using alternate vectors</td>
</tr>
<tr>
<td>Insufficient output</td>
<td>LV threshold may be elevated</td>
<td>Increase output, or reposition LV lead</td>
</tr>
<tr>
<td>Resynchronization is delivered &gt; 90% with capture</td>
<td>Multiple end-points have been used to define non-responders, details in text</td>
<td>Six-minute walk or oxygen consumption treadmill; assess ejection fraction; formal QOL assessment</td>
</tr>
<tr>
<td></td>
<td>Program parameters may not optimize intraventricular, interventricular or atroventricular mechanical function</td>
<td>If frequent atrial pacing (as opposed to sensing) is disrupting left AV mechanical synchrony, use VDD mode</td>
</tr>
<tr>
<td>Non-optimal lead position</td>
<td>Insufficient RV-LV separation to allow resynchronization</td>
<td>Reposition lead, particularly if in anterior vein, or small RV-LV separation radiographically and small V-V interval during intrinsic rhythm</td>
</tr>
<tr>
<td>Absence of dyssynchrony</td>
<td>Program to minimize ventricular pacing; CRT may not help</td>
<td></td>
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</tbody>
</table>

Discussed in more detail in Chapter 2, Hemodynamics.
Fig. 10.106 Systematic approach to insuring delivery of cardiac resynchronization therapy. CRT, cardiac resynchronization therapy; PVC, premature ventricular complex; VTns, non-sustained VT; AVN, AV node.

Fig. 10.107 Extended and true bipolar left LV pacing configurations (available in the Contak Renewal 3™ ICD). The top two panels depict extended bipolar pacing, in which pacing occurs between LV and RV electrodes. This configuration has the advantage of requiring only a single LV electrode, but the disadvantage of permitting anodal capture (Fig. 10.108). The ability to select either the distal or proximal LV electrode as cathode for pacing functionally permits “moving” the LV lead via non-invasive reprogramming. This can be advantageous postoperatively if the pacing threshold increases or phrenic nerve stimulation occurs. The bottom two panels depict true bipolar LV pacing, which eliminates the possibility of anodal RV capture, but requires a bipolar LV lead. (Reproduced with permission from Blackwell Publishing.)
anodal capture is extremely rare in Medtronic and Boston Scientific ICDs, which use integrated-bipolar pacing. St Jude ICDs use an RV ring during CRT if the LV lead is unipolar and the RV lead is true-bipolar (resulting in higher likelihood of anodal capture), and an RV coil as the anode when an integrated bipolar RV lead is used.

Phrenic nerve stimulation

The left phrenic nerve courses adjacent to the left ventricular free wall (Fig. 10.110). An epicardial pacing lead in a coronary sinus tributary in juxtaposition to the phrenic nerve may stimulate it at the pacing rate, which is clinically intolerable. Phrenic stimulation may become apparent only after implantation due to alteration of the electrode–phrenic nerve relationship with postural changes or minor lead migration. Decreasing the pacing output, reprogramming unipolar to bipolar LV pacing or changing the LV pacing vector (in systems with this capability, such as the Boston Scientific Contak Renewal® or Medtronic Concerto®) may eliminate phrenic stimulation non-invasively. If sufficient difference between the phrenic and left ventricular capture thresholds cannot be achieved with any of these maneuvers, the left ventricular lead is repositioned. Episodic phrenic stimulation may relate to body position. Alternatively, it may relate to transient increases in the pacing amplitude performed by some ICDs (Boston Scientific) in order to assess daily impedance to confirm lead integrity. Disabling this feature may eliminate symptoms in patients with phrenic capture at the higher output.
ICD programming and resynchronization algorithms

Any parameter setting that minimizes ventricular pacing or permits ventricular fusion will adversely affect cardiac resynchronization. Such settings include a long AV delay, a prolonged postventricular atrial refractory period or extension of the postventricular atrial refractory period after a premature ventricular complex, a low maximal-tracking rate, AV search hysteresis, rate smoothing up or down or use of a DDI pacing mode. Atrial undersensing and ventricular oversensing may similarly minimize ventricular pacing and limit resynchronization. Resynchronization ICDs have features designed to maintain LV stimulation. To prevent LV T-wave oversensing, Boston Scientific RenewalTM ICDs incorporate an LV refractory period, which may inhibit LV pacing. Additionally, they include an LV protection period after a sensed or paced event during which pacing will not occur. Although designed to prevent pacing in the LV vulnerable period, this parameter reduces the maximum LV pacing rate and may inhibit cardiac resynchronization.

Resynchronization ICDs also employ algorithms designed to maximize ventricular pacing during potentially disruptive events such as premature ventricular complexes or rapidly conducted atrial arrhythmias. In Medtronic resynchronization ICDs, the Ventricular Sense ResponseTM feature triggers pacing in one or both ventricles after each RV-sensed event in order to maintain resynchronization. Both Medtronic and Boston Scientific systems include algorithms that temporarily shorten the postventricular atrial refractory period to

Fig. 10.109 Anodal capture during testing of LV capture threshold. LV pacing occurs between a LV cathode and a RV ring anode. (A) Upper left panel: From top to bottom: surface ECG, electrogram markers and intervals, atrial electrogram and ventricular near-field electrogram. At left, pacing output is 2.5V and anodal capture is present, with capture occurring at both the LV electrode and the RV ring. No electrograms are visible on the LV channel due to the simultaneous RV and LV capture. With a decrement in pacing output to 2.25V, the QRS morphology changes on the ECG (circled complex); and electrograms appear on the ventricular channel, representing local RV depolarization. Inset at lower right shows that the time between the pacing stimulus and the RV electrogram represents the interventricular conduction time during LV pacing. (B) With further decrement in LV pacing output from 0.75 to 0.5V, true loss of LV capture occurs, with widening of the surface ECG due to left bundle branch block. The local RV depolarizations are similar during LV pacing (in A) and intrinsic AV conduction (in B).54 (Reproduced with permission from Blackwell Publishing.)
regain atrial tracking and biventricular pacing after premature ventricular complexes or during sinus tachycardia faster than the nominal upper rate limit. By shortening this refractory period, an early atrial complex has a higher chance of landing in a non-refractory window so that it will be followed by a tracked resynchronized (biventricular paced) complex after the AV interval times out. In contrast, atrial events sensed in the refractory period are not tracked. Medtronic’s Conducted AF Response™ resynchronizes conducted beats in atrial fibrillation up to a minimum R–R interval without increasing the average ventricular rate. Boston Scientific’s Ventricular Rate Regularization™ algorithm is intended to restore resynchronization and ventricular regularity by pacing the ventricle during irregular conduction of atrial fibrillation. Concealed conduction into the AV node from the paced events may slow AV nodal conduction, thereby limiting the pacing-induced increase in ventricular rate. These and other algorithms designed to maximize cardiac resynchronization therapy may result in pacing after QRS onset on surface ECGs and pacing at “unexpected” times (Fig. 10.111). Awareness of these algorithms prevents unnecessary intervention.

Algorithms designed to maintain a high percent of cardiac resynchronization pacing during atrial fibrillation may be confused with inappropriate pacing. (A) Hospital telemetry shows ventricular pacing occurring after the QRS onset due to the Medtronic Ventricular Sense Response™ algorithm, which introduces a triggered biventricular pacing pulse after each RV-sensed event. (B) Surface ECG lead II, event markers and the RV tip to LV tip electrogram are displayed during atrial fibrillation. The first three complexes represent biventricular pacing (“BV”). The last complex represents a conducted complex that was sensed (“VS”). The split marker associated with the “VS” indicates that a triggered pace pulse resulted. (C) Hospital telemetry shows pacing during rapidly conducted atrial fibrillation due to an algorithm designed to maximize cardiac resynchronization pacing. This pacing, with the patient at rest, is distinct from rapid, rate-responsive pacing that is triggered by a sensor. Reproduced with permission from Blackwell Publishing.)
Conclusion

Advances in ICD technology have greatly enhanced defibrillator reliability. Additionally, when troubleshooting is required, sophisticated event and data storage, coupled with real-time telemetry and automated assessment of pacing thresholds and impedance values, provides information leading to rapid diagnosis. The addition of self-diagnostic tests, audible and palpable warnings and remote monitoring permit earlier detection of potential problems. Despite these significant enhancements, the growing complexity of defibrillators, lead systems and algorithms mandates a systematic approach to troubleshooting and a detailed understanding of device function so that system malfunction can be diagnosed and effectively corrected.

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CHAPTER 10  Troubleshooting


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CHAPTER 11

Pacemaker, ICD and CRT Radiography

David L. Hayes, Paul A. Friedman

Introduction

The chest radiograph remains an important tool in the pre- and postoperative evaluation of a pacing, defibrillation and cardiac resynchronization therapy (CRT) system. Additionally, the chest radiograph is essential when assessing the integrity of a pacing or implantable cardioverter-defibrillator (ICD) system. Both a posteroanterior (PA) and a lateral view should be obtained. A systematic approach should be employed, evaluating various anatomical and device components in an orderly fashion.1–3

Preoperatively, the presence of clips, wires, prosthetic valves, and other hardware can provide clues to prior cardiac or thoracic surgery, which may be important when planning the operative procedure.

Fig. 11.1 Posteroanterior (A) and lateral (B) chest radiographs of a dual-chamber pacing system. The pulse generator is located in a left prepectoral position. The position of both atrial and ventricular leads is acceptable. The "J" on the atrial lead is adequate, and is best seen in the lateral view. The ventricular lead is not positioned in a true apical position but is well seated with adequate slack. In a true apical position, it would be seen closer to the sternum in the lateral view. (Reproduced with permission from Hayes DL. Radiography of implantable arrhythmia management devices. In: Kusumoto F, Goldschlager N, eds. Cardiac pacing for the clinician.)
After implantation of the pacing, ICD or CRT system, both PA and lateral radiographs should be obtained to confirm correct lead position(s) (Fig. 11.1) and to note potential surgical complications such as pneumothorax, pleural effusion, and pericardial effusion. (In addition, an oblique film may at times be helpful to determine more precisely the location of a coronary venous lead.) A chest radiograph should be performed as part of most device trouble-shooting assessments. Table 11.1 provides a systematic approach to assessment of the chest radiograph of the device patient. Comparison with any previous radiographs is frequently useful. As part of the “total” care of the patient, inspection of the entire radiograph should be done, including: bony structures, aorta, cardiac silhouette, trachea, diaphragm, and lung fields.

**Pulse generators**

Most pulse generators are placed in a prepectoral location, inferior to the clavicle and medial to the axilla. At one time, many implants in children as well as many early ICDs were placed in an abdominal position, either below or anterior to the rectus muscle. If the pulse generator is located in an abdominal position, radiographic evaluation requires an AP radiograph of the abdomen as well as a PA and lateral chest radiograph. In some patients a subpectoral position may be used in lieu of the more common prepectoral location. Subpectoral may occasionally be advantageous, either because of inadequate tissue to maintain the integrity of a system in a prepectoral position, or for cosmetic preferences.

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**Table 11.1 Systematic approach to radiographic assessment of pacemakers and ICDs**

<table>
<thead>
<tr>
<th>Systematic approach</th>
<th>Clinical considerations</th>
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<tr>
<td>Determine pulse generator site</td>
<td>Any suggestion that there has been a significant shift from intended position, e.g., displaced generator, could be associated with lead dislodgment or Twiddler’s syndrome.</td>
</tr>
<tr>
<td>Determine pulse generator manufacturer, polarity, and model if possible</td>
<td>Radiographic identifiers allow determination of manufacturer—helpful if the patient comes without ID card. Polarity of pulse generator should be determined and compared with polarity of leads</td>
</tr>
<tr>
<td>Inspect the connector block</td>
<td>Is connector pin(s) completely through connector block? Loose connection could explain intermittent or complete failure to output or intermittent failure to capture.</td>
</tr>
<tr>
<td>Consider venous route utilized</td>
<td>Especially important if a pacemaker system revision is being considered, i.e., can the same venous route be accessed and how many leads are already placed in a single vein.</td>
</tr>
<tr>
<td>Determine lead polarity</td>
<td>Does lead polarity match pulse generator polarity or has some type of adaptor been used to allow the system hardware combination?</td>
</tr>
<tr>
<td>Determine lead position</td>
<td>Determine where the lead was positioned, i.e., for the ventricular lead, is it in the apex, outflow tract, septal position, coronary sinus?; for the atrial lead, is it atrial appendage, lateral wall, septal position, coronary sinus?</td>
</tr>
<tr>
<td>Does lead position appear radiographically acceptable?</td>
<td>Inadequate lead position may explain failure to capture and/or sense. Compare current X-ray to previous X-ray if possible. Is ventricular lead redundancy or “slack” adequate; is atrial “J” adequate?</td>
</tr>
<tr>
<td>Inspect entire length of lead for integrity, i.e., fracture, compression, crimp, etc.</td>
<td>Intermittent or complete failure to capture/sense and/or output could be secondary to lead conductor coil fracture or loss of insulation integrity. Attempt to follow each lead along its course assessing the conductor coil. Also inspect for any “crimping” of the lead as it passes under the clavicle.</td>
</tr>
<tr>
<td>Any other chest X-ray abnormality that is potentially related</td>
<td>For a recent implant, be certain there is no pneumothorax or hemopneumothorax. For the ICD patient with a change in defibrillation thresholds, whether acute or chronic, remember that a pneumothorax can be responsible for alterations in DFT.</td>
</tr>
<tr>
<td>If no abnormality is appreciated radiographically but there is a clinical abnormality—re-assess the chest X-ray in a problem-oriented fashion</td>
<td>As an example, if the patient has intermittent failure to output, the differential diagnosis would include a problem with the connector pin, i.e., loose setscrew, or conductor coil fracture. Go back once again and inspect these elements of the pacing system.</td>
</tr>
</tbody>
</table>
The retro-mammary position was once advocated for a better cosmetic result. However, given the size of contemporary devices, this surgical approach is rarely necessary and long-term is more difficult because of the more involved operative techniques required (Fig. 11.2). True axillary position has also been used in an effort to obtain a better cosmetic result, but due to the somewhat more complex implant technique as well as potential discomfort of the pulse generator in this position, use of this location is uncommon.

The pulse generator manufacturer and model can usually be identified from the chest radiograph. At one time unique shape, size, and internal circuitry pattern were sufficient to identify a manufacturer and model. Although this no longer holds true, all current devices have a radio-opaque code identifying the manufacturer and model of the device or some alphanumeric code by which the company’s technical services group can identify the “family” of devices (Fig. 11.3, Table 11.2). After identification of the manufacturer, the technical support division of that manufacturer can identify the device and should be able to provide additional information obtained at the time of implant and kept on file with the pulse generator registration (leads utilized, configurations, thresholds, etc.).

Migration of a pulse generator is less common with the smaller devices used today. However, comparison of previous and current radiographs for pulse generator position is useful. The clinical concern about pulse generator migration is that tension may be placed on the sternum, whereas the CS leads wraps posteriorly in the CS, closer to the spine. (Reproduced with permission from Hayes DL. Radiography of implantable arrhythmia management devices. In: Kusumoto F, Goldschlager N, eds. Cardiac pacing for the clinician.)

Fig. 11.2 Posteroanterior (A) and lateral (B) chest radiographs of a CRT-D, system with the pulse generator located in a retromammary position. Note that the right ventricular coil is in a low outflow/high septal position. The preferred view for distinguishing outflow tract and coronary sinus (CS) positions is the lateral view. In the lateral film, the right ventricular coil is just behind the sternum, whereas the CS leads wraps posteriorly in the CS, closer to the spine. (Reproduced with permission from Hayes DL. Radiography of implantable arrhythmia management devices. In: Kusumoto F, Goldschlager N, eds. Cardiac pacing for the clinician.)
the lead, possibly causing dislodgment or fracture. It is not possible to determine from a radiograph whether the patient is a “Twiddler,” but a twisted appearance of the lead suggests Twiddler’s syndrome, be it secondary to true patient manipulation of the pulse generator or, more commonly, rotation of the pulse generator within an oversized pocket (Fig. 11.5).5

Inspection of the generator connector block should be performed. The lead connector pin should be advanced beyond the setscrew(s) in the connector block, and the screws should be in direct contact with the pin. One cause of intermittent pacing failure, failure to output and/or failure to capture, is a loose connection between the setscrews and the connector pin (Fig. 11.6).

Implantable loop recorders are implanted with increasing frequency and appear as a small device usually in the pectoral region without an associated lead (Fig. 11.7).

Leads

Lead assessment is a critical component of the radiographic assessment of a pacing and/or ICD system. Standard radiographic techniques may not clearly demonstrate lead components. At one time, higher radiographic penetration and/or coning of the radiographic field was suggested to achieve better visualization of leads. However, if the radiograph has been stored...
Fig. 11.5 Abdominal radiograph in a patient with a malfunctioning implantable cardioverter-defibrillator. Inspection demonstrated a tight twisting of the lead (note loops in lead just to the left of the can), which resulted in device malfunction. The most likely diagnosis was Twiddler syndrome. (From Hayes DL. Complications; and Lloyd MA, Hayes DL. Pacemaker and ICD radiography. In: Hayes DL, Lloyd MA, Friedman PA, eds. Cardiac pacing and defibrillation: a clinical approach. Armonk, NY: Futura Publishing, 2000:453–84, 485–517. Used with permission of Mayo Foundation for Medical Education and Research.)

Table 11.2 Radiographic identifiers by manufacturer

<table>
<thead>
<tr>
<th>Manufacturer</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biotronik</td>
<td>ICDs/CRT identified with a three-part code: year of manufacture, Biotronik logo and a two-letter code unique to each device family; pacemakers identified with a two-part code: Biotronik logo and a two-letter code unique to each device family</td>
</tr>
<tr>
<td>Boston Scientific</td>
<td>Letters “GDT” to identify the manufacturer (Guidant), followed by a three-digit number which identifies which model software application is needed to communicate with the pulse generator; three-digit number does not correlate with a device model number</td>
</tr>
<tr>
<td>Medtronic</td>
<td>Historically used a unique alphanumeric identifier for each device “model.” With current generation devices the radiographic identifier signifies a broader grouping of Medtronic devices. With the “group” identifier, technical services would be able to determine specific models that would reside within that grouping</td>
</tr>
<tr>
<td>St Jude Medical</td>
<td>Each device has an alphanumeric series that corresponds to a specific model and can be identified from the technical manuals or by calling technical services</td>
</tr>
<tr>
<td>Sorin/ELA</td>
<td>ELA devices: Current generation devices only the letters “ELA” appear with exception of the letter “O” for the Ovation ICD. The ELA software will recognize any ELA device. Previous generation devices had “ELA” and the model number. Sorin Biomedica devices: Include a three- to five-alphanumeric code that is specific for each model</td>
</tr>
</tbody>
</table>

Fig. 11.4 Examples of three pulse generator polarity configurations, as described in the text. Left panel, unipolar polarity; middle panel, bifurcated bipolar single-chamber pacemaker; right panel, dual-chamber bipolar configuration. (Reproduced with permission from Hayes DL. Radiography of implantable arrhythmia management devices. In: Kusumoto F, Goldschlager N, eds. Cardiac pacing for the clinician.)
digitally it may be possible to manipulate the images to better delineate the leads. Figure 11.8 demonstrates a PA chest radiograph before and after the contrast has been altered. Unfortunately, lead-insulating materials are not visualized radiographically, and loss of insulation integrity breech is a cause of lead failure that is not uncom-

Fig. 11.6 Posteroanterior radiograph (A) and close-up view (B) from a patient with intermittent failure to pace. Comparison of the upper and lower pins reveals that the lower of the two unipolar leads is not completely advanced. This difference is more evident on the close-up view. By convention, the lower of the two leads in the connector block is the ventricular lead, so that this patient must have had intermittent or permanent ventricular failure to output. An unrelated observation, noted by the arrowhead, is shallow positioning of the atrial lead, i.e., the “J” is much wider than 90°. (From Hayes DL. Pacemaker radiography. In: Furman S, Hayes DL, Holmes DR Jr, eds. A practice of cardiac pacing, 3rd edn. Mount Kisco, NY: Futura Publishing, 1993:361–400. Used with permission of Mayo Foundation for Medical Education and Research.)

Fig. 11.7 Posteroanterior chest radiograph demonstrating an implantable loop recorder.
mon. Occasionally it is possible to see what appears to be a lack of continuity of the lead surface, which may correlate with a breach in the insulation (Fig. 11.9).

The number, types, location and radiographic integrity of all leads should be determined after studying the PA and lateral radiograph. Unlike pulse generators,

**Fig. 11.8** Posteroanterior chest radiographs before (A) and after (B) digital manipulation of the contrast level. By manipulating the contrast the pacemaker leads can be more easily seen in (B).

**Fig. 11.9** Posteroanterior (A) chest radiograph and close-up (B) of a portion of the posteroanterior film demonstrating a disruption of the outer portion of the lead, presumably

the insulation, with the appearance that the conductor coil is intact (arrowhead). In this patient, chronic pacing thresholds were considered acceptable and unchanged.
leads do not have characteristic radio-opaque markers to aid in their identification; however, information regarding the type and functional status of various leads can be obtained by their radiographic appearance.

It is not uncommon for a patient to have a cardiac rhythm device implanted and have a variety of functional and abandoned leads, both epicardial, transvenous and/or coronary venous. If additional procedures are required and lead extraction is required, it may be necessary to determine the type, position and integrity of all leads.

**Pacemaker leads**
The vast majority of contemporary pacing leads are placed transvenously, although patients may still have epicardial (myocardial) pacing wires placed after certain types of cardiac surgical repair or due to congenital cardiac anomalies. Intravascular insertion of transvenous leads is usually through either the right or left axillary, subclavian, or cephalic veins. The internal or external jugular veins have been used in the past, but should not be considered a good alternative for lead placement at this time (Fig. 11.10). Jugular vein insertion sites are usually easily identified by the lead coursing over or under the clavicle.

Clues as to the insertion site may be obtained from the chest radiograph by a change in the directional contour of the lead (Fig. 11.11). A more medial insertion site suggests a subclavian approach; a more lateral site suggests a cephalic or axillary approach.

![Fig. 11.10 Posteroanterior chest radiograph. One lead is placed in the subclavian vein; a second lead inserted through the right jugular vein has been transected. (Reproduced with permission from Hayes DL. Radiography of implantable arrhythmia management devices. In: Kusumoto F, Goldschlager N, eds. Cardiac pacing for the clinician.)](image1)

![Fig. 11.11 Radiographs demonstrating different venous routes for permanent lead placement. Upper left, subclavian vein placement. Note compression of the lead as it passes between the first rib and the clavicle (arrow); upper right, axillary vein placement; bottom, cephalic vein placement.](image2)
A femoral approach may occasionally be needed if access via the superior veins is not an option. An X-ray of the pelvis and abdomen is necessary to assess adequately the integrity of the lead (Fig. 11.12).

The radio-opaque conductor coil should be inspected in its entirety. There should be no discontinuity of the coil; any kinking or sharp angulation may represent lead fracture (Fig. 11.13). Special attention should be paid to the area between the first rib and the clavicle, as this is a frequent site of lead fracture (subclavian crush syndrome) (Fig. 11.14).

A pseudofracture, shown in Fig. 11.15, is a finding that is becoming largely of historical interest. This intact bifurcated bipolar lead shows discontinuity at the point of bifurcation (black arrow). This is not a fracture, but rather the normal radiographic appearance of this lead, which simply reflects the two conductors of a bipolar lead come together. (When uncertainty exists as to whether a discontinuity is a pseudofracture, fluoroscopy and impedance assessment may also be helpful.) As noted previously, there are still a small number of these leads in service that had been used for both pacemakers and ICDs.

The term “pseudofracture” has also been inappropriately applied to a different circumstance: the indentations caused by ligatures compressing the insulating...
material of a lead (Fig. 11.16). These indentations do not usually have any clinical significance, i.e., they do not imply the presence of lead damage, although it is possible to affect lead integrity with excessively tight ligatures placed around the sleeve.

It is generally difficult to make a radiographic determination regarding the diameter or "French" size of the implanted leads. Traditionally leads have been "stylet-driven." Newer leads are "lumenless" and of significantly smaller size. Such leads may be more difficult to detect and "follow" on the radiograph (Fig. 11.17).

**Intracardiac position**

In order to appreciate abnormal lead position, a de-
tailed description of the normal radiographic appearance is necessary.

Leads should have a modest amount of redundancy present. Undue tension on leads may result in poor pacing thresholds or frank dislodgment from the endocardial surface. In Fig. 11.18, both atrial and ventricular leads are positioned in such a way that they are too shallow. The atrial lead is most likely in the right atrial appendage. The atrial lead is not optimally positioned and is best appreciated on the lateral view.

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**Fig. 11.17** Posteroanterior (A) and lateral (B) chest radiographs demonstrating a ventricular “lumenless” lead. The small diameter of the lead, 4 Fr, makes it somewhat more difficult to see on the radiograph.

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**Fig. 11.18** Posteroanterior (A) and lateral (B) chest radiographs in a patient with suboptimal positioning of both leads. The ventricular lead position is also inadequate, i.e., too little slack has been left on the lead.
The angle of the “J” is significantly greater than 90°. The ventricular lead is also much too shallow, and this can be appreciated in both views.

Generous lead redundancy is preferred in pediatric patients in an attempt to accommodate subsequent growth and minimize the number of lead revisions which will be required during a lifetime of pacing therapy (Fig. 11.19).10

Unipolar leads have a single electrode (cathode) at the tip, whereas bipolar leads will have two electrodes separated by a variable inter-electrode space (Fig. 11.20). The type of lead fixation can often be determined by the chest radiograph (Fig. 11.21). Active fixation leads have a radio-opaque screw, which is usually visible radiographically. The tines of a passive fixation lead cannot be visualized, so the absence of a “screw” would suggest a passive fixation mechanism.

**Transvenous atrial leads**

Atrial leads may have a preformed “J” shape, or the lead may be straight but positioned in the atrium in such a way that a “J” is formed. Pre-formed “J” leads are now used less frequently than standard leads for atrial application. Use of a pre-formed “J” may limit options to place the lead in alternative atrial positions.

Atrial leads have historically been most commonly positioned in the atrial appendage unless the atrial appendage had been surgically amputated. Regardless of whether a preformed “J” or a straight lead is implanted in the atrium, if implantation is in the right atrial appendage, the J portion of the lead is slightly medial on the PA projection and anterior on the lateral projection. Optimally, the limits of the “J” should be no greater than approximately 80° apart. Redundancy proximal to the J within the atrium or superior vena cava should not be seen.

There is growing interest and enthusiasm for positioning the atrial lead in a septal position to avoid or minimize the intra-atrial conduction delay that may occur when the lead is positioned in the appendage (Fig. 11.22). Any portion of the free wall may also be targeted (Fig. 11.23), as long as there is mechanical stability and satisfactory thresholds can be obtained. Unlike the appendage, the remainder of the atrium is not trabeculated, and active fixation leads are usually required to obtain mechanical stability.

Pacing for the reduction or prevention of atrial fibrillation remains somewhat controversial. However, techniques that have been used include positioning the lead in the atrial septum or the use of two atrial leads.11–14 In Fig. 11.24, one lead is positioned in the right atrial appendage and the other lead on the posterior septum near the coronary sinus os. Pacing the atrium via the coronary sinus has also been advocated, but has never been widely performed. A large posterior curve sug-

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**Fig. 11.19** Posteroanterior and lateral chest X-ray from a pediatric patient with a newly implanted pacemaker. Excessive redundancy has been left on the ventricular lead to allow for future growth.
gests placement in the coronary sinus, although it could also suggest placement across a patent foramen ovale or atrial septal defect into the left atrium. Special note should be made of the appearance of a type of atrial lead used many years ago, which maintained the "I" shape by incorporating a "retention wire" into the
Fig. 11.22 Posteroanterior (A) and lateral (B) chest radiographs of a dual chamber pacing system. This patient had long intra-atrial conduction time and in an attempt to normalize atrial activation the atrial lead was placed on the intra-atrial septum. Note that in the posteroanterior radiograph the atrial lead is not on the free wall and is located close to the tricuspid annulus. On the lateral radiograph the lead is seen to be posterior. This is the typical radiographic appearance of an atrial lead positioned on the low atrial septum just posterior to the coronary sinus. (Reproduced with permission from Hayes DL. Radiography of implantable arrhythmia management devices. In: Kusumoto F, Goldschlager N, eds. Cardiac pacing for the clinician.)

Fig. 11.23 Posteroanterior (PA) (A) and lateral (B) chest radiographs demonstrate an atrial position other than the atrial appendage. The lead is positioned laterally.
lead (Fig. 11.25).\(^{15}\) This lead design had the potential for the retention wire to fracture and protrude through the insulation, resulting in cardiac and/or vascular laceration, or may migrate into extracardiac locations. There are still small numbers of these leads in service and they require ongoing digital fluoroscopic surveillance because the retention wire itself may be difficult to visualize on a standard radiograph, even with significant protrusion or migration of the wire.

Transvenous ventricular leads

Transvenous ventricular leads are traditionally placed in the right ventricular apex. Radiographically, the lead should have a gentle contour with the tip of the lead pointing downward in the PA view and located between the left border of the vertebral column and the cardiac apex. The position of the heart, vertical or relatively more horizontal, largely determines the position of the lead in relation to the cardiac apex and varies among patients. The lateral view is necessary to distinguish an apical position in which the lead tip is anterior and caudally directed, is directed posteriorly in the right ventricle, or is on the posterior surface of the heart, i.e., within the coronary sinus. The ventricular lead should have a gentle curve along the lateral wall of the right atrium and cross the tricuspid valve to the ventricular apex (Fig. 11.1). It may be preferable to place the lead on the right ventricular septum or outflow tract (Fig. 11.26).

There is growing interest in placement of the leads in a non-apical position, given the potential for adverse hemodynamics from an apically placed lead. Although the data are inconclusive regarding the long-term hemodynamic advantages of a non-apical position and...
the potential superiority of one non-apical position over another, i.e., low septal vs. mid-septal vs. His bundle vicinity, there is enough experience with non-apical positioning to state that it is feasible and safe.16–18

A non-apical position may also be chosen for specific pacing applications. Figure 11.27 demonstrates a patient with a device placed specifically for monitoring right ventricular (pulmonary artery) pressures. The
lead is placed in the right ventricular outflow tract. The radiographs in Fig. 11.26(C) and (D) contain multiple leads in multiple right ventricular positions. The patient has a right ventricular apical lead that is part of an original ICD system. The leads connected to the device located in the right prepectoral region are part of a cardiac contractility modulation device placed as part of a clinical investigation. The leads are positioned in the posterior septum and the anterior septum. Figure 11.28 represents a combination of epicardial leads that

Fig. 11.27 Posteroanterior (A) and lateral (B) chest radiograph of a patient with a pulse generator and a single lead placed in the right ventricular outflow tract for the purpose of hemodynamic monitoring.

Fig. 11.28 (A) Posteroanterior (PA) chest radiograph of endocardial and epicardial pacing leads in a patient with D-transposition of the great vessel is status post Mustard procedure. From the right infraclavicular region the endocardial leads enter the heart through the superior vena cava, then into the atrium. The leads course anterior to the baffle connecting the pulmonary veins into the right ventricle. The atrial lead is in atypical left atrial location anterior to the baffle. The ventricular lead enters the morphological left ventricle proximal to its apex. The epicardial system is seen in the left chest with screw-in leads placed posteriorly and inferiorly into the ventricle. An atrial lead has not been placed. (B) A close-up from the PA radiograph notes the presence of a “Y” connector which connects two ventricular epicardial leads to this single chamber ventricular pacemaker. On this close-up there is a defect in the lead adaptor just as it exits the connector block (arrow).
are being actively used and abandoned transvenous new leads in a patient with a congenital abnormality.

A left ventricular endocardial position is generally undesirable\(^9\) (Fig. 11.29). Although there is a reported experience with left ventricular endocardial pacing for purposes of resynchronization,\(^8\) this technique has not gained popularity because of concern related to the thromboembolic potential of having a lead permanently positioned in the systemic circulation. However, leads intended for right-sided placement may reach the left ventricular cavity through perforation of the ventricular septum, the lead having inadvertently crossed a patent foramen ovale, atrial septal defect, or ventricular septal defect during transvenous placement, and in the pericardial space as a result of perforation. The preferred view identifying a lead as terminating in the left ventricle is the lateral projection, in which the lead will be excessively posterior (toward the spine) (Fig. 11.30). If there is early recognition that a lead has inadvertently been placed in the left atrium or left ventricle, the lead should be withdrawn and repositioned in the right side of the heart.

Lead dislodgment may occur and may result in failure to capture and/or sense. Dislodgment may be obvious, i.e., macrodislodgment. Such dislodgment can be anywhere other than its original position, i.e., the pulmonary artery, coronary sinus, ventricular cavity, or superior or inferior vena cava. Dislodgment may not be identifiable radiographically. This has been labeled “microdislodgment” but, in the absence of X-ray documentation, there is no evidence of its presence.

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**Fig. 11.29** Posteroanterior (A) and lateral (B) chest radiographs demonstrating a ventricular lead that courses over the spine, is relatively straight, and is positioned in an unusually high position when viewed on the posteroanterior (A) film. On the lateral film (B), the lead is seen to course posteriorly, which is consistent with a left ventricular position. On a still frame from the two-dimensional echocardiogram (C), the lead is seen crossing the aortic valve and residing in the left ventricle. The lead had been placed inadvertently via the subclavian artery. (Reproduced with permission from Hayes DL. Radiography of implantable arrhythmia management devices. In: Kusumoto F, Goldschlager N, eds. Cardiac pacing for the clinician.)
It is, therefore, a presumptive diagnosis. A “macrodislodged” atrial lead is shown in Fig. 11.31.

In Fig. 11.32 a large loop is seen in the ventricular lead on the lateral radiograph. The patient’s chronic thresholds were excellent and no problems had been encountered. Even though the positioning is suboptimal, if function is normal then no intervention is necessary in this patient. If the radiograph in Fig. 11.32
was taken the day following pacemaker implantation, i.e., less than optimal positioning noted soon after implant and/or pacing or sensing thresholds were poor, repositioning might be considered.

It is often helpful to compare serial chest X-rays to confirm a lead dislodgment. In Fig. 11.33, PA chest X-rays from two consecutive days demonstrate a change in coronary venous lead position.
Fig. 11.33 (A) Posteroanterior (PA) and (B) lateral chest radiograph taken the day after CRT device implantation. The coronary sinus lead appears to be adequately positioned. (C) PA and (D) lateral chest radiograph taken the day after CRT device implantation. The coronary sinus lead has definitely moved, with radiographic evidence of a less distal position on the second set of radiographs.

Note the absence of a right ventricular lead—all ventricular pacing is from the left ventricle in this system. (Reproduced with permission from Hayes DL. Radiography of implantable arrhythmia management devices. In: Kusumoto F, Goldschlager N, eds. Cardiac pacing for the clinician.)
Single-lead VDD systems are not commonly used, but can be identified by an additional bipolar sensing electrode in the atrialized portion of the lead, which may or may not have contact with the endocardial surface (Fig. 11.34).

**Epicardial leads**

Epicardial pacing leads are used in patients with specific congenital cardiac abnormalities, some pediatric patients, and in patients with right A-V valve prostheses (Fig. 11.35). The lead(s) are tunneled to the pulse generator either in the pectoral or abdominal area. Historically, epicardial leads have had a higher incidence of lead failure, and a transvenous approach is generally preferred if possible (Fig. 11.36).

When epicardial leads are present and the pulse generator is located in an abdominal position, it should be remembered that this position seems to increase the likelihood of lead fracture significantly. Although longevity of epicardial leads has yet to be equal to transvenous pacing leads, epicardial lead function has improved in recent years.

**ICD leads**

**Epicardial ICD leads**

Currently used infrequently (Fig. 11.37), such systems
Fig. 11.36  (A) Posteroanterior and (B) lateral chest X-ray from a patient with three epicardial pacing leads. There are two “stab-in” leads, one of which is fractured (arrow). There is also a single screw-in ventricular epicardial lead. (Reproduced with permission from Furman SF, Hayes DL, Holmes DR Jr. A Practice of Cardiac Pacing. Mt Kisco, NY. Futura Publishing Company, Inc., 1986.)

Fig. 11.37  (A) Lateral (LAT) chest radiograph (B) posteroanterior (PA) radiographs that demonstrate fracture of the epicardial patch (arrow). Note the posterior positioning of the patches in the lateral view consistent with idea location on the posterior and posterolateral left ventricle. (Reproduced with permission from Brady PA, Friedman PA, Trusty JM, Grice S, Hammill SC, and Stanton MS. J. Am. Coll. Cardiol. 1998;31:616–622.)
usually have easily recognizable patches positioned over the heart, as well as epicardial or transvenous pacing leads for sensing and pacing purposes. Depending on the manufacturer, the actual defibrillation coils may or may not be easily visualized. One manufacturer’s epicardial patches feature a radio-opaque marker around the periphery of the patch; this is not active, and a fracture of this marker does not reflect on the integrity of the patch. The actual coils of these patches are radiolucent and not visible on the radiograph. A frequent site of fracture in all epicardial patches is at the patch–lead junction, and this area should be carefully inspected.

**Transvenous ICD leads**
The leads most commonly have an active fixation mechanism, but may be passive, and like pacing leads should have a gentle redundancy. Leads may have a single high-voltage coil in the right ventricle, or may have an additional coil in the superior vena cava area (Figs 11.38 and 11.39). Rarely, an additional subclavian lead, subcutaneous patch, or array may be used in order to obtain satisfactory defibrillation thresholds (Fig. 11.40). As with pacing leads, the insertion into the connector block and the connection with the setscrews should be noted. All leads and patches should be inspected for obvious fracture, and for unusual bending or kinking. Subcutaneous patches and arrays are usually placed inferior and posterior to the axilla, and lateral and/or customized oblique views may be required to obtain satisfactory visualization of them.

Although no longer manufactured, a number of patients continue to use Transvene endocardial defibrillation leads, which utilize a coaxial coil design more prone to failure. This lead can be identified by the presence of fillers (the loops of conductor) along the body as opposed to longitudinal conductors (Fig. 11.41).

Due to the design of defibrillation leads, there are characteristic sites of pseudofracture with which the caregiver should be familiar (Fig. 11.42).

**Coronary venous leads**
Coronary sinus lead placement was used many years ago, but lost favor because of the high rate of lead dislodgment. However, with the advent of CRT, placing a permanent lead in the coronary venous system has become commonplace. Atrial pacing can also be achieved via the coronary venous system, but this is not commonly done for permanent pacing.

Radiographic evaluation of a CRT system requires the caregiver assessing the radiograph to have a familiarity with coronary venous anatomy and lead placement. The placement of the left ventricular lead will vary due to variations in coronary sinus anatomy. There is in-

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**Fig. 11.38** Posteroanterior (A) and lateral (B) chest radiographs of an older implantable cardioverter-defibrillator (ICD) system. The ICD is connected to a single-coil ventricular lead. Single-coil leads are now used less commonly for initial implants, but a number are still in use.
Fig. 11.39 Posteroanterior (A) and lateral (B) chest radiographs of an implantable cardioverter-defibrillator (ICD) system in the left prepectoral position. The ICD is connected to a dual-coil ventricular lead.

Fig. 11.40 Posteroanterior (A) and lateral (B) chest radiographs from a patient with an implantable cardioverter-defibrillator. Unacceptable defibrillation thresholds necessitated placement of a subcutaneous array.
increasing enthusiasm for radiographic evaluation of the coronary venous system with CT venography prior to attempt CRT implant. This provides additional pre-implant information regarding variations in coronary venous anatomy, potential target veins, etc. (Fig. 11.43).

The ventricle or atrium can be paced from the coronary sinus, depending on where the lead is positioned. The atrium is paced when leads are positioned in the coronary sinus itself; the ventricle is paced with very distal placement, or in any of the ventricular tributaries, as usually used for CRT. Figures 11.44, 11.45 and 11.46 demonstrate coronary sinus lead placement in the lateral anterior interventricular, and middle cardiac veins respectively.

If assessing dislodgement of a coronary sinus lead, the lateral chest X-ray is paramount, as differentiation of a right ventricular outflow tract from coronary sinus site is difficult on the PA image alone.

Miscellaneous considerations
Occasionally, it may be difficult to locate the subclavian vein, or the patency of the cephalic/axillary/subclavian vein may be in question. Contrast material injected into a peripheral intravenous line in the ipsilateral upper extremity will define the venous anatomy as it flows into the central circulation, determining patency and guiding access (Fig. 11.47). There is also evidence that using contrast-guided venous puncture on a routine basis will minimize the incidence of pneumothorax.
Fig. 11.43 Computed tomography (CT) venography (A) larger image of a single view and (B) a composite of images demonstrating coronary venous system by CT angiography.
Management of the patient with a prosthetic tricuspid valve that requires permanent pacing requires special consideration. A single ventricular pacing lead can be placed across a bioprosthetic valve, but there is no way to predict if the lead could potentially interfere with valve function or minimize the functional longevity of the bioprosthetic valve. Placement of the ventricular lead with echocardiographic guidance, as well as fluoroscopy, has been suggested in an effort to minimize trauma to the bioprosthetic valve. The goal of echocardiographic guidance would be to preferentially place the lead so that it was in a commissure of the bioprosthetic valve. Our ex-

![Fig. 11.44 Posteroanterior (A) and lateral (B) chest radiographs from a patient with right atrial, right ventricular and coronary sinus leads. In the posteroanterior view the coronary sinus lead is noted to be leftward and closer to the lateral wall, whereas on the lateral view the coronary sinus lead is seen to be the most posterior of the three leads. This is consistent with posterolateral cardiac venous positioning of this lead. The large distance between the left and right ventricular lead electrodes is desirable for cardiac resynchronization. (Reproduced with permission from Hayes DL. Radiography of implantable arrhythmia management devices. In: Kusumoto F, Goldschlager N, eds. Cardiac pacing for the clinician.)](image1)

![Fig. 11.45 Posteroanterior (A) and lateral (B) chest radiographs from a patient with right atrial, right ventricular (RV), and coronary sinus leads. The coronary sinus lead is positioned in a lateral branch of the anterior interventricular cardiac vein. (Reproduced with permission from Hayes DL. Radiography of implantable arrhythmia management devices. In: Kusumoto F, Goldschlager N, eds. Cardiac pacing for the clinician.)](image2)
experience with this approach is limited, but we have found that it is difficult, even with echocardiographic guidance, to be certain that the lead is within a commissure. Use of small French size leads that may be more “mobile” and preferentially settle in a commissure may be optimal.

A standard ventricular transvenous pacing lead cannot be placed across a mechanical prosthetic tricuspid valve. Several approaches have been used in this circumstance. If the need for permanent pacing is anticipated at the time of tricuspid valve replacement, some have advocated placing the transvenous lead outside the sewing ring of the tricuspid valve. The concern with this approach is the difficulties that would be encountered if the lead would need to be extracted at some future date or if the lead failed and another lead was necessary. Alternatively, transmural ventricular pacing lead placement can be considered. In this approach, a cardiac surgeon places an active fixation lead through the free wall of the right ventricle and the lead is actively fixated to the endocardial surface. A purse-string suture is placed around the lead where it passes through the right ventricular lead. Our limited but long-term success with this technique has been excellent29 (Fig. 11.48).

Another option in the patient who has undergone tricuspid valve replacement or surgery, in whom it is desirable to avoid placing a lead across the tricuspid valve, or in patients in whom ventricular access is limited because of congenital anomalies, would be to pace...
the ventricle via the coronary sinus. With newer, more reliable coronary sinus leads this should be a viable option (Fig. 11.49).

The most common cardiovascular congenital anomaly encountered by the implanting physician is persistent left superior vena cava, in which the leads are needed for atrioventricular block. The ventricular pacing lead was placed via the coronary sinus into a lateral vein position.

Fig. 11.48 Posteroanterior (A) and lateral (B) chest radiographs of a patient with prosthetic mitral and tricuspid valves. A ventricular lead passes through the free wall. (Reproduced with permission from Furman SF, Hayes DL, Holmes DR Jr. A Practice of Cardiac Pacing, 3rd Edition. Mt Kisco, NY Futura Publishing Company, Inc., 1993.)

Fig. 11.49 Posteroanterior (A) and lateral (B) chest radiographs of a patient with prosthetic aortic, mitral and tricuspid valves. A permanent pacemaker was
placed through the left vena cava, into the coronary sinus and then into the right atrium. The ventricular lead will usually need to be looped in order to direct it across the tricuspid valve into the right ventricle (Fig. 11.50). The diagnosis can be suspected by the finding of an enlarged coronary sinus at echocardiography, or by contrast injection into an ipsilateral intravenous line in the left upper extremity.

Congenital cardiovascular anomalies may require novel implantation techniques and may result in unusual lead placement. Knowledge of the native and corrected anatomy is essential when planning a procedure and when interpreting the chest radiograph; consultation with the congenital cardiologist or cardiovascular surgeon may be helpful (Figs 11.51 and 11.52).

Summary

The chest radiograph provides important information about pacing, ICD and CRT systems. As systems become more complex and specialized, and as indications for these devices expand, the importance of recognizing the normal and abnormal radiographic appearance of these systems is essential. A systematic evaluation of the anatomy and system components is necessary preoperatively, postoperatively, and during trouble shooting. When approaching a patient with suspected or known system malfunction, following the systematic radiographic evaluation in Table 11.1 can provide important clues to and/or the actual diagnosis. If a systematic radiographic approach is followed and no abnormalities are appreciated, the radiograph should be reassessed in a focused approach. For example, if the patient had presented with intermittent and/or consistent failure to output, both lead fracture and loose connector pin would be part of the differential diagnosis. The chest radiograph should then be reinspected with specific attention to the lead in question and the connector block.

Following this approach will allow the clinician to gain the most information possible from the radiograph.

Fig. 11.50 Posteroanterior (A) and lateral (B) chest radiographs of a DDD pacing system in a patient with a persistent left superior vena cava. The atrial lead courses through the left superior vena cava and the coronary sinus and into the right atrium. The ventricular lead follows the same path, but is then looped into the right ventricle. (From Hayes DL. Implantation techniques. In: Hayes DL, Lloyd MA, Friedman PA, eds. Cardiac pacing and defibrillation: a clinical approach. Armonk, NY: Futura Publishing, 2000:159–200. Used with permission of Mayo Foundation for Medical Education and Research.)
Fig. 11.52 Posteroanterior (A) and lateral (B) chest radiographs of a patient with dextrocardia, total situs inversus with congenitally corrected transposition of the great vessels. Due to progressive dysfunction of the systemic ventricle, a cardiac resynchronization device was placed. The coronary sinus lead is positioned in a right ventricular vein fairly basally in a lateral branch. With congenitally corrected transposition of the great arteries the RV is posterior and explains why the lead looks as if it is still in the coronary sinus when the lateral radiograph is examined. The ICD lead is in the morphological left ventricular apex with the left ventricle being more anterior than the right. The atrial lead is most likely located in the region of the anterior intra-atrial septum.
CHAPTER 11 Pacemaker, ICD and CRT Radiography

References


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CHAPTER 12
Electromagnetic Interference and Implantable Devices

David L. Hayes, Paul A. Friedman

Some of the most common questions patients ask have to do with potential sources of electromagnetic interference (EMI). Their concerns are often misdirected because of myth or sensationalism by the media. It is important to know not only what sources of interference are of potential concern, but also how external interference actually affects pacemakers, implantable cardioverter-defibrillators (ICDs) and cardiac resynchronization therapy (CRT) systems. Interference refers to an inappropriate device response to electromagnetic signals resulting in absent, modified, or inappropriate detection or therapy.

Implantable devices are subject to interference from many sources. Most sources of EMI are non-biological, but in addition, biological sources of interference, such as myopotentials and extremes of temperature, may cause pacemakers and defibrillators to malfunction. In general, contemporary devices are effectively shielded against commonly encountered EMI. There has always been some concern about EMI that patients may encounter in the non-hospital environment, but because of improvements in pulse generator shielding and design, EMI is now of less concern outside of military and industrial environments. The principal sources of interference that affect pacemakers and defibrillators are in the hospital environment.

The portions of the electromagnetic spectrum that may affect implantable devices are radio waves, with frequencies between 0 and 109 Hz, including alternating current electricity supplies (50 or 60 Hz) and electrocautery, and microwaves, with frequencies between $10^9$ and $10^{11}$ Hz (Fig. 12.1). Higher frequency portions of the spectrum, including infrared, visible light, ultraviolet, X-rays and gamma rays, do not usually interfere with implanted devices because their wavelength is much shorter. However, therapeutic radiation can damage pulse generator circuitry directly.

EMI enters a pacemaker, defibrillator or CRT device by conduction if the patient is in direct contact with the source or by radiation if the patient is in an electromagnetic field in which the lead acts as an antenna. Pacemakers and defibrillators have been protected from interference by shielding of the pacemaker circuitry, filtering of the incoming signal, and reduction of the distance between the electrodes to minimize the “antenna” size, thus maximizing nearby (myocardial) signals while minimizing far-field signals. Similarly, defibrillator systems utilizing true bipolar sensing (tip to ring) are less susceptible to EMI than those incorporating integrated sensing (tip to distal coil) right ventricular leads due to the smaller “antenna” with the tip to ring lead. The contemporary pulse generator is protected from most sources of interference because the circuitry is shielded inside a stainless steel or titanium case. In addition, body tissues provide some protection by reflection or absorption of external radiation.

Bipolar leads sense less conducted and radiated interference because the distance between anode and cathode is smaller than that for unipolar leads. Bipolar sensing configuration largely eliminates myopotential inhibition in pacemakers. Defibrillators incorporate variable signal amplification to permit detection of fine ventricular fibrillation (VF) electrograms while avoiding sensing of T waves, so that myopotential interference may on occasion occur. Since amplification
increases with time and is augmented following paced beats, oversensing is most likely to occur at slow heart rates, particularly while pacing. In pacemakers, studies have shown that with bipolar sensing, there is considerably less sensing of external electrical fields and less effect from electrocautery during surgery.

Sensed interference is filtered by narrow band-pass filters to exclude non-cardiac signals. However, signals in the 5–100 Hz range are not filtered because they overlap the cardiac signal range. These signals can result in abnormal pacemaker behavior if they are interpreted as cardiac.

The possible responses to external interference include:
• Inappropriate inhibition of pacemaker output (Fig. 12.2)
• Inappropriate triggering of pacemaker output (Fig. 12.3) and inappropriate tracking (if sensed on the atrial channel)
• Asynchronous pacing (Fig. 12.4)
• Reprogramming, usually to a backup mode (Fig. 12.5)
• Inappropriate initiation of “other” features, such as mode switching or rate drop response (Fig. 12.6)
• Damage to the pacemaker circuitry (Fig. 12.7)
• Inappropriate detection of EMI as ventricular tachycardia (VT)/ventricular fibrillation (VF) (Fig. 12.8)
• Failure to sense VT/VF

The most common responses to EMI are triggering or inhibition of pacemaker function, and spurious tachyarrhythmia detection in ICDs.

Pacemaker responses to noise

Asynchronous pacing
To protect the patient from inappropriate inhibition of pacemaker output, contemporary pacemakers have the capability of reverting to asynchronous pacing if exposed to sufficient interference. This change is usually activated by signals detected during a noise-sampling period (NSP) within the pacemaker timing cycle (Fig. 12.9A,B). The NSP (noise sampling period or resettable refractory period) occurs immediately after the ventricular refractory period (VRP), which
Fig. 12.3 Electrocardiographic recording taken at rest from a patient with a pacemaker programmed to the DDDR mode. During exposure to equipment emitting a radiofrequency signal close to that of the pacemaker, the external signal was sensed on the atrial sensing circuit of the pacemaker, resulting in tracking and a paced ventricular rate at the programmed upper rate limit.

Fig. 12.4 Electrocardiographic recording taken at rest from a patient with a pacemaker programmed to the DDDR mode. During exposure to equipment emitting a radiofrequency signal close to that of the pacemaker, the external signal resulted in asynchronous (DOO) pacing.

Fig. 12.5 Electromagnetic reprogramming. (A) Transtelephonic electrocardiographic tracing during magnetic application from a patient with a pacemaker programmed to the DDD pacing mode. The transmission reveals VOO pacing instead of the expected DOO response. (B) Electrocardiographic tracing obtained after reprogramming during magnetic application to the DDD mode; there is appropriate DOO pacing. A history obtained from the patient revealed that she had undergone magnetic resonance imaging of the head. This procedure had reprogrammed the pacemaker to the backup mode, i.e., VVI at a rate of 65 ppm.
Fig. 12.6 Electrocardiographic tracing from a patient with a dual-chamber pacemaker. The electrocardiogram is obtained during exposure to a source of electromagnetic interference. The interference is sensed on the atrial sensing channel, and the pacemaker responds by rapid ventricular tracking. Criteria are met for mode switching, and the rate begins to fall back to the programmed lower rate.

Fig. 12.7 Programmer printout after elective cardioversion in a patient with a DDD pacemaker. The telemetered data from the atrial lead are incomplete, with lead impedance noted to be "high" and output current, energy delivered, and charge delivered "low." In addition, there was failure to pace in the atrium. The device was explanted; direct measurements on the atrial lead were all within an acceptable range. A new pacemaker was attached and functioned normally. Destructive analysis of the explanted device showed damage to the atrial pacing circuit.

<table>
<thead>
<tr>
<th>RELAY DATA</th>
<th>TELEMETRY DATA</th>
</tr>
</thead>
<tbody>
<tr>
<td>PACING RATE</td>
<td>60 MIN⁻¹</td>
</tr>
<tr>
<td>PACING INTERVAL</td>
<td>1000 MS</td>
</tr>
<tr>
<td>CELL VOLTAGE</td>
<td>2.65 VOLTS</td>
</tr>
<tr>
<td>CELL IMPEDANCE</td>
<td>3.75 KOHMS</td>
</tr>
<tr>
<td>CELL CURRENT</td>
<td>19.0 UA</td>
</tr>
<tr>
<td>SENSITIVITY</td>
<td>0.5</td>
</tr>
<tr>
<td>LEAD IMPEDANCE</td>
<td>HIGH</td>
</tr>
<tr>
<td>PULSE AMPLITUDE</td>
<td>2.69</td>
</tr>
<tr>
<td>PULSE WIDTH</td>
<td>0.40</td>
</tr>
<tr>
<td>OUTPUT CURRENT</td>
<td>LOW</td>
</tr>
<tr>
<td>ENERGY DELIVERED</td>
<td>LOW</td>
</tr>
<tr>
<td>CHARGE DELIVERED</td>
<td>LOW</td>
</tr>
</tbody>
</table>

Fig. 12.8 Noise caused by electrical stimulation delivered during use of some type of chiropractic treatment equipment. This shock was ultimately aborted due to fortuitous discontinuation of energy delivery. (Top: atrial tip to ring EGM; Middle: ventricular tip to ring EGM; Bottom: marker channel.)
follows a ventricular sensed or paced event. During the VRP the ventricular sensing channel does not react to any signals and in particular prevents oversensing of the afterpotential of the ventricular pacing artifact or the evoked QRS and T waves. The VRP usually lasts between 200 and 400 ms, and events occurring during this period have no effect on pacemaker timing. The NSP lasts between 50 and 200 ms. If an event is sensed during this period, it is interpreted as noise, and either the VRP or the NSP is restarted. In addition, in a dual-chamber mode, the postventricular atrial refractory period and the upper rate interval, but not the lower rate interval, are restarted. If a further noise event is detected in the NSP, the VRP or NSP again is

Fig. 12.9 (A) Examples of noise reversion mechanisms from one manufacturer. Noise reversion occurs in a dual-chamber pacemaker when there is continuous refractory sensing in the atrial or ventricular refractory period. During noise reversion a pacemaker in a non-rate-adaptive mode, pacing will occur at the programmed lower rate. If the pacemaker is programmed to most rate-response modes, pacing will occur at the sensor-indicated rate. (B) Although there will be some variation among manufacturers, in the VVIR mode the pacemaker will most likely pace at the programmed lower rate during noise reversion. Reproduced with permission from Medtronic Adapta technical manual, p. 3-28.
restarted and the pacemaker does not recognize cardiac signals. Repetitive noise events eventually cause the lower rate interval to time out, and a pacing pulse is delivered. Continuous noise thus results in asynchronous pacing at the lower rate limit.

In some pacemakers, rather than timing out the lower rate interval, repetitive detection of noise in the NSP causes temporary switching to a specific “noise reversion mode,” which is usually an asynchronous mode (VOO or DOO). In some pacemakers with programmable polarity, the pacing output is unipolar in the noise reversion mode, even if the device is programmed to bipolar pacing.

In other devices there is technically not a noise reversion mode but designated operation during “noise detection.” When an intrinsic depolarization is sensed, a 60-ms noise rejection interval is started and is re-triggered in the presence of noise. Intrinsic events will not be detected during noise rejection and asynchronous operation can result if the noise continues (Fig. 12.10).

Whether noise causes inhibition or asynchronous pacing depends on the duration and field strength of the signal. As the field strength increases, there is a greater tendency to inhibition, because the noise may be sensed intermittently and may not be sensed in the NSP, but in the alert period between the NSP and the next pacing pulse. At higher field strengths, the noise is sensed continuously, including in the NSP, and asynchronous pacing occurs. Pacemaker models vary considerably in their susceptibility to noise.

Because VT/VF can be a high-frequency low-amplitude signal, ICDs necessarily interpret some noise episodes as VF. ICDs either have no noise reversion mode or very short noise sampling windows, providing imperfect protection from exogenous interference. If the noise is sufficiently sustained, VT/VF is detected and therapies delivered.

**Mode resetting (power-on reset, or electrical reset)**

Momentary, strong EMI can reprogram the pacing mode, as opposed to the transient changes already described. This is most commonly caused by external defibrillation or electrocautery, and does not resolve when the EMI is discontinued. This is usually the “backup mode” or “reset mode” and in pacemakers is often the same as the pacemaker elective replacement indicator or “battery depletion” mode (Fig. 12.5). Confusion can arise when the “backup mode” and the default settings at battery depletion are the same. Recognition of these parameters indicates that either the pacemaker has been affected by interference or has truly reached battery depletion. In both cases, careful attention to the programmer telemetry, when available, is helpful. If the telemetryed cell impedance remains low or the battery voltage is normal, the pacemaker battery is not exhausted and interference is the problem. In addition, if the pacemaker is reprogrammed to the original pacing mode with maximum output and an increased rate, it quickly reverts to the settings that indicate battery depletion if the pacemaker battery is truly near depletion.

The backup or reset mode is usually VVI, and if the pulse generator has programmable polarity, the backup polarity is unipolar. Some pacemakers may reset to VOO mode if subjected to interference, potentially resulting in competition with the intrinsic rhythm. In ICDs, a power-on reset will cause the device to revert to a backup pacing mode (such as VVI at 60 ppm) and shock only therapies with maximum energy shocks for heart rates greater than pre-defined detection rates, typically > 140–170 ppm. This occurs since only factory preset parameters stored in non-volatile memory are available following the power-on reset.

Exposure to low temperatures before implantation also may result in mode resetting. The cold tempera-
tures cause an increase in the internal battery resistance, and the subsequent decrease in the battery voltage causes the end-of-life indicator or reset mode to be activated. Because this effect occurs frequently during shipment in cold climates, all pacemakers should be routinely interrogated before implantation and reprogrammed if necessary (Fig. 12.11). If an ICD interrogation before surgical implantation indicates that an electrical reset has occurred, it is best to contact the manufacturer before implanting the unit.

**Environmental electromagnetic interference**

Electric and magnetic signals are emitted by certain industrial, hospital-medical and domestic sources. Each of these environments is discussed individually.

**Hospital environment**

The hospital is the most common environment with sources of potential EMI that can cause significant interference with implantable devices (Table 12.1).

**Electrocautery**

Electrocautery continues to be one of the most common potential sources of EMI for patients with implanted devices. Electrocautery involves the use of radiofrequency current to cut or coagulate tissues. It is usually applied in a unipolar fashion between the cautering instrument (the cathode) and the indifferent plate (the anode) attached at a distance to the patient’s skin. Bipolar cautery equipment is also available. The frequency is usually between 300 and 500 kHz (at frequencies of < 200 kHz.

![Fig. 12.11 Power on reset screen.](image-url)
Cutting diathermy uses a modulated signal, so that bursts of energy are applied, whereas coagulation diathermy uses an unmodulated signal to heat the tissue. Coagulation diathermy is used in radiofrequency ablation of cardiac tissue for the treatment of arrhythmias.

The current generated by electrocautery is related to the distance and orientation of the cautery electrodes relative to the pacemaker and lead. High current is generated if the cautery cathode is close to the pacemaker, and particularly high currents are generated in the pacemaker if it lies between the two cautery electrodes.

Electrocautery can result in multiple clinical responses from an implanted pulse generator (Table 12.2). The electrocautery signal may induce currents in the pacing lead and cause local heating at the electrode, leading to myocardial damage with a subsequent increase in pacing or sensing threshold or both. Threshold alteration is usually transient.

To prevent inappropriate inhibition of a pacemaker, a magnet can be applied over the pacemaker during cautery to convert it to the asynchronous mode. Although this maneuver may be successful, it may open some pacemakers to reprogramming by the electrocautery signal and is therefore controversial.

Pacemakers with rate-responsive functions may exhibit inappropriate responses during surgery due to vibration sensed from other intraoperative equipment or vibrations created by the surgical procedure. The electrocautery signal may overwhelm the impedance measuring circuit of a minute ventilation rate-responsive pacemaker and cause pacing at the upper rate limit.

There are other considerations when managing ICDs when there is potential exposure to electrocautery. Because electrocautery signals could be misidentified as cardiac activity, they could result in inappropriate delivery of antitachycardia pacing or defibrillation or result in failure to deliver appropriate defibrillation. For these reasons, the ICD patient should be placed on a monitor and therapies turned “off” prior to the surgical procedure. Personnel capable of recognizing tachyarrhythmias and responding to the rhythm abnormalities with external defibrillation should be available throughout the time that therapies are turned off.

If the ICD patient is pacemaker dependent, it is important to know the ICD response to magnet application. Magnet response varies between manufacturers and sometimes between models of the same manufacturer (Table 12.3). Most commonly, but certainly not universally, magnet application in ICDs disables tachyarrhythmia detection without altering pacing function. In this situation, it is best to use a programmer to

### Table 12.2 Potential effects of electrocautery

- Reprogramming
- Permanent damage to the pulse generator
- Pacemaker inhibition
- Reversion to a fallback or noise reversion mode, or electrical reset
- Myocardial thermal damage

### Table 12.3 ICD magnet response

<table>
<thead>
<tr>
<th>Manufacturer</th>
<th>Response Details</th>
</tr>
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<tbody>
<tr>
<td>Medtronic</td>
<td>Pacing mode, pacing rate and interval as programmed. VF, FT and VFT detection is suspended. Patient Alert audible tones will occur if applicable and enabled.</td>
</tr>
<tr>
<td>Boston Scientific</td>
<td>If “ENABLE MAGNET USE” is “on” (nominal), device will emit beeping synchronous tones on the R wave. If after 30 seconds the beeping does not change to a continuous tone, the magnet must be taped over the device to temporarily inhibit therapy. If beeping changes to a continuous tone after 30 seconds, tachy mode has gone to “off” and magnet can be removed. To turn device back to Monitor and Therapy, magnet should be placed back over the device for 30 seconds until R wave synchronous tones are heard. Magnet application does not affect pacing mode/rate. If “ENABLE MAGNET USE” is programmed “off” (nominal “on”), then a magnet will NOT inhibit therapy. No tones will be emitted and a programmer will be needed to turn device off.</td>
</tr>
<tr>
<td>SJM</td>
<td>Two programmable options for magnet response: NORMAL (Nominal) or IGNORE. In “NORMAL” response — when magnet is placed over the ICD it blinds detection and delivery of therapy. Bradycardia pacing is not affected by a magnet placed over the device and must be reprogrammed if asynchronous pacing is needed. If “IGNORE” is programmed the blinded is null and void.</td>
</tr>
<tr>
<td>Sorin/ELA</td>
<td>When magnet is applied it disables tachy therapy and arrhythmia detection. Brady function is to pace in the programmed mode at the magnet rate (corresponding to battery voltage); pacing outputs are set to maximum; rate hysteresis and AV extension are set to zero; AV delay is set to the programmed AV delay at rest.</td>
</tr>
<tr>
<td>Biotronik</td>
<td>When a magnet is applied, tachyarrhythmia therapy and detection will be suspended and rate-response is suspended.</td>
</tr>
</tbody>
</table>
confirm device function before dismissing the patient from monitored care. The caregiver also needs to know whether the ICD has a programmable asynchronous mode (Table 12.4). With this information, the device should be managed in such a way that inhibition is avoided in the pacemaker-dependent patient.

Patients with implanted devices who are to undergo surgery in which electrocautery may be used should be assessed preoperatively. Table 12.5 provides all the information necessary, but a few key points merit repetition. Preoperatively, it is most important to determine the programmed settings and whether the patient is pacemaker-dependent.

In the operating room, it is most important that the indifferent plate of the electrocautery be placed at a distance from the pulse generator, usually on the thigh, and that good contact be ensured. The effect of electrocautery may be difficult to assess, because it causes interference on the electrocardiographic (ECG) monitor. Another method of assessing cardiac rhythm should be used, i.e., pulse oximetry and/or arterial blood pressure monitoring. Cautery should be used with caution in the vicinity of the pulse generator and leads. The cathode should be kept as far from the pulse generator as possible, and the lowest possible amplitude should be used.

During electrocautery, device function and cardiac rhythm should be carefully assessed. The most likely response is transient inhibition or asynchronous pacing during electrocautery, which should not cause a signifi-
CHAPTER 12  Electromagnetic Interference and Implantable Devices

559

cant hemodynamic problem. Secure connection with good skin contact with the grounding pad is essential; disconnection of the pad may result in the implanted cardiac device serving as the anode for cautery, resulting in injury at the myocardial lead interface. Use of two dispersive pads protects against this risk.

Postoperatively, it is critical that the pulse generator be interrogated and if it is in the reset mode it should be reprogrammed to the original settings. Rechecking thresholds following exposure to electrocautery and comparison with preoperative values is reasonable, but not an absolute requirement. If problems are encountered during interrogation of the pacemaker or reprogramming to the original settings, the manufacturer should be consulted to determine whether a malfunction has occurred.

Defibrillation
External transthoracic defibrillation produces a large amount of electrical energy delivered in the vicinity of a cardiac device and has the potential to damage both the pulse generator and the cardiac tissue in contact with the lead. Cardiac devices are protected from damage by high defibrillation energies through special circuitry incorporating a Zener diode that electronically regulates the voltage entering the pacemaker circuit and that should prevent high currents from being conducted by the lead to the myocardium. However, the extremely high energies can overwhelm this protection and cause damage to the pacemaker or the heart. Internal defibrillation via epicardial or subcutaneous patches or intracardiac defibrillation electrodes delivers smaller amounts of energy but may also interfere with pacemaker function. Bipolar pacemakers are less susceptible than unipolar pacemakers to interference from defibrillation.

As with electrocautery, defibrillation may result in reprogramming to the backup or reset mode, a transient increase in pacing or sensing threshold, or damage to pacemaker circuitry.

The degree of damage may be related to the distance of the defibrillation paddles from the pulse generator. The paddles should be placed as far as possible from the generator; when possible, an anterior-posterior configuration is preferred (Fig. 12.12). However, in the anterior-anterior configuration, the paddles should be 10 cm away from the pulse generator if possible. After defibrillation, the pulse generator should be interrogated and the programmed settings compared with those before defibrillation and cardioversion. A transient rise in threshold should be managed by increasing the energy output if necessary (Fig. 12.13). Rarely, prolonged, severe threshold increases occur that necessitate lead replacement. In patients with ICDs, shock delivery via the ICD typically results in successful cardioversion and eliminates the need for external shock delivery and potential interactions. Using the smallest dose of external energy when it is required minimizes the risk of cardioversion-related implanted device complications; biphasic waveforms are more effective than monophasic waveforms, and are preferred.3 Recommendations for management of patients undergoing cardioversion and defibrillation are summarized in Table 12.6.

Catheter ablation
Catheter ablation of intracardiac tissues to control arrhythmias was first performed with direct current shock. This technique had a higher tendency to affect pacemakers than did external defibrillation, and patients frequently experienced problems, including reprogramming to the backup or reset mode, pacemaker circuit failure, and transient increases in pacing and sensing thresholds. Direct current catheter ablation is rarely used due to the superiority of radiofrequency ablation, and it should be avoided in patients with permanent pacemakers.

Catheter ablation most commonly uses radiofrequency current, which is the same as coagulation electrocautery, i.e., unmodulated radiofrequency current delivered at 400–500 kHz. Effects similar to those of surgical electrocautery have been reported, including
inappropriate inhibition, asynchronous pacing, and re-setting to backup mode.⁵,⁶

The ablation catheter is usually some distance from the pacing or defibrillation electrode, and radiofrequency ablation is commonly accomplished safely near implanted leads and pulse generators in bipolar systems without significant myocardial damage at the site of the pacemaker electrode.

When atrioventricular (AV) nodal ablation is performed, some operators prefer to place the permanent pacemaker before AV nodal ablation. Performing the procedure in this sequence obviates temporary pacemaker placement.

Before radiofrequency ablation is performed, however, it is essential to interrogate the pulse generator, record its programmed settings, and measure thresholds. Rate-adaptive sensing should be programmed off. A programmer should be available during the procedure. After the procedure, the device should be checked and reprogrammed if necessary. In our experience, pacing system damage or threshold changes are extremely rare in this setting.

**Magnetic resonance imaging**

In magnetic resonance imaging (MRI), three types of electromagnetic fields are present that may interact...
with implanted cardiac devices: a static magnetic field (which may exert mechanical force and/or activate the reed switch), a rapidly changing magnetic field, and a radiofrequency field (the latter two of which may lead to heating, electrical reset, and pacing inhibition or triggering). When a pacemaker is near an MRI scanner with the electromagnet “on,” the reed switch may close, resulting in asynchronous pacing. Although there may be competition with the underlying cardiac rhythm, asynchronous pacing rarely causes a clinical problem.

Animal studies of the effect of MRI on implanted devices have demonstrated the potential hazards. In some pacemakers, no effect other than asynchronous pacing occurred. In other pacemakers, cardiac pacing at the same frequency or a multiple of the frequency of the radiofrequency current occurred; for example, if the MRI device was operating at 200 ms, pacing rates at 300 ppm were observed in some dogs. The radiofrequency signal is detected by the leads acting as an antenna and is then amplified by the pacemaker circuitry to produce sufficient energy to pace the heart.

Reported interactions and/or problems of implantable devices in MRI scanners are magnet-activated asynchronous pacing, inhibition by the radiofrequency signal, rapid pacing induced by the radiofrequency signal, discomfort at the pacemaker pocket, reprogramming, and death due to induction of a ventricular tachyarrhythmia. Transient reed switch malfunction has been seen, and there are concerns that are more theoretical than real of the MRI resulting in heating of the conductor coil and tissue damage at the electrode/myocardial interface. Pacemaker circuitry damage by MRI is unlikely, but accelerated battery depletion could be seen in a device that is at or near elective replacement indicator parameters.

Recent human studies suggest that MRI can be performed safely in carefully selected patients with pacemakers and ICDs, undergoing specific imaging sequences. In the study by Sommer et al., there were no significant differences in immediate and long-term sensing amplitudes, pacing thresholds, or lead impedances. Tests were performed in patients with thoracic and non-thoracic MR examinations and diagnostic questions were answered in 100% of non-thoracic and 93% of thoracic studies. Pacemaker-dependent patients were excluded. In the study by Nazarian et al., only extrathoracic MRI examinations were performed, but were done in dependent and non-pacemaker-dependent patients and performed with an acceptable risk-to-benefit ratio.

In short, the interactions between implanted cardiac rhythm devices and MRI scanners are complex, and may be influenced by imaging factors (magnet strength, imaging sequence), device factors (device type, lead type, polarity, sensitivity and other parameter settings), and patient factors (pacemaker dependency, susceptibility to arrhythmias). While routine MRI imaging of all patients with implanted rhythm devices cannot be recommended, MRI imaging of patients with pacemakers and defibrillators can be safely performed in selected individuals with advanced preparation.

**Extracorporeal shock wave lithotripsy**

Extracorporeal shock wave lithotripsy (ESWL) is a non-invasive treatment for nephrolithiasis that delivers multiple, focused hydraulic shocks, generated by an underwater spark gap, to a patient lying in a water bath. The shock is focused on the stones by an ellipsoidal metal reflector. Because the shock wave can produce ventricular extrasystoles, it is synchronized to the R wave.

ESWL is safe to use with implanted pacemakers, provided that the shock is given synchronously with the ECG and that dual-chamber pacemakers have safety pacing enabled. In the pacemaker-dependent patient, it is recommended that a dual-chamber pacemaker be programmed to the VVI, VOO or DOO pacing mode to avoid ventricular inhibition. Programming of a DDD pulse generator to the VVI, VOO or DOO mode also avoids rare instances of irregularities of pacing rate, supraventricular arrhythmias that can be tracked or induced, and triggering of the ventricular output by electromechanical interference.

ESWL has not been reported to cause any damage to the pacemaker, except that if an older activity-sensing pacemaker utilizing a piezoelectric crystal is placed at the focal point of the ESWL, the crystal could be shattered. This has become less of a problem as piezoelectric crystal-based activity sensing has been replaced by accelerometer technology, but patients with piezoelectric crystal-based activity sensors can undergo lithotripsy safely if the device is implanted in the thorax; lithotripsy should be avoided in these patients if the device is located in the abdomen.

**Transcutaneous electrical nerve stimulation**

Transcutaneous electrical nerve stimulation (TENS) is a commonly used method for the relief of acute and chronic pain from musculoskeletal and neurological problems. A TENS unit consists of several electrodes...
placed on the skin and connected to a pulse generator that applies pulses of between 1 and 200 V and 0 and 60 mA at a frequency of 20–110 Hz. The output and frequency of the unit can be adjusted by the patient to provide maximum relief of pain.

The repetition frequency of the TENS output is similar to the normal range of heart rates, so it would be expected that TENS pulses might cause pacemaker inhibition. Although a study of 51 patients with pacemakers has shown no inhibition during TENS stimulation, cases have been reported of asymptomatic inhibition of pacemaker output by TENS. Interference is most likely to occur in significantly older pacemakers and pacemakers in the unipolar sensing configuration.

TENS can probably be used safely in most patients with bipolar pacemakers. However, it is reasonable to take special precautions in the pacemaker-dependent patient and monitor the response during initial TENS application. If TENS results in interference in patients with unipolar pacemakers, the testing can be repeated after reprogramming the sensitivity to a less sensitive value.

There is at least one published report of interference with a CRT device despite initial testing with the TENS unit and no evidence of interference. TENS units are best avoided in ICD patients. The TENS unit creates EMI that can be misinterpreted as ventricular fibrillation, leading to inappropriate device discharge. If the indication for TENS use is compelling, therapy could be delivered with the detections programmed “off” (in a monitored setting), or interaction testing should be considered.

Electroconvulsive therapy
Electroconvulsive therapy (ECT) appears safe with respect to implantable device function, as only a minimal amount of electricity reaches the heart because of the high impedance of body tissues. We would routinely place ICD patients on continuous monitoring and turn tachyarrhythmia detection “off” until after ECT therapy is completed. ECG monitoring during the procedure and interrogation of the pacemaker after the procedure are advisable. In pacemakers programmed to a unipolar sensing configuration, seizure activity may generate sufficient myopotentials to result in inhibition or ventricular tracking.

The equipment used may also generate an electrical field capable of causing 60-cycle interference (Fig. 12.14).

Diathermy
“Diathermy” can be used to refer to several therapies. Surgical diathermy is also known as electrosurgery or electrocautery and is discussed above. Diathermy is used in physical medicine and rehabilitation for the purpose of deep tissue heating. Ultrasonic diathermy heats tissues with ultrasound and electric diathermy utilizes high-frequency alternating magnetic or electric fields. Diathermy can be a source of interference, and because of its high frequency it should be avoided near the pulse generator implantation site. It has the potential to inhibit the pulse generator or damage the pulse generator circuitry by excessive heating.

Impedance plethysmography
Some patient monitoring systems can cause interference with minute ventilation sensing pacemakers. Specifically, interference can occur with a monitor in which impedance plethysmography is used to document the patient’s respiratory rate and detect ECG lead disconnection. Because the minute ventilation sensor also functions by detecting a change in intrathoracic impedance, the monitor may result in inappropriate sensor-driven pacing.
When connected to such a monitor, the pacemaker sensor measures the summated impedance signals coming from the monitor, falsely interprets the information as an increase in transthoracic impedance, and increases the heart rate accordingly. As soon as the patient is disconnected from the monitor, the heart rate returns to the programmed lower pacing rate. Figure 12.15 shows an example of such monitor-driven interference.

The Food and Drug Administration has issued an advisory about this phenomenon to manufacturers of monitoring equipment. It is important that physicians working with such patients in the operating suite and intensive care unit be familiar with this circumstance.

New technologies
Any new medical technologies that generate an electromagnetic field or deliver ionizing radiation, irrespective of whether they are cardiovascular in nature, should be assessed to determine whether they generate clinically significant interference with implanted pacemakers and defibrillators. For example, wireless endoscopic video capsules, relatively new technology, have been tested for potential interference when used in patients with permanent pacemakers. The video capsules were capable of causing a “reset mode,” but no other interference that was felt to be clinically significant.

Industrial environment
Conventional wisdom has been to advise patients to avoid “arc welding” and close contact with combustion engines. This advice needs to be re-examined as pacemakers become more resistant to external interference. However, as previously discussed, pacemakers using a unipolar sensing configuration remain more susceptible to EMI than pacemakers in a bipolar sensing configuration. For patients whose livelihood involves equipment with potential for EMI, a pulse generator with committed bipolar sensing configuration should be implanted.

Industrial environments with significant potential for clinically significant EMI with implantable devices include industrial-strength welding, i.e., welding equipment exceeding 500 A, use of degaussing equipment, and induction ovens. If a patient works in one of these environments or potentially some other even more obscure environment that suggests significant potential for EMI, the work environment should be carefully evaluated. If the patient is pacemaker-dependent, consideration should be given to assessment of the work environment by an engineer from the pacemaker manufacturer. Some manufacturers are willing to send an engineer to the patient’s work environment to conduct such testing. If the patient is not pacemaker-dependent, assessment may be achieved by ambulatory monitoring during exposure to the environment or by use of patient-triggered event records stored within the pacemaker (Fig. 12.16).

From a practical standpoint, most patients who claim to do “arc welding” use low-amperage equipment for hobby welding. If the patient uses welding equipment in the 100–150 A range, significant EMI is unlikely to occur. However, before giving the patient permission...
Fig. 12.16 Tracing from an ambulatory monitor from a man with intermittent high grade AV block. He worked in an industrial environment working with very powerful induction heaters. He would become intermittently symptomatic in various portions of the work area. Ambulatory monitoring correlated intermittent pacemaker inhibition with symptoms. It was difficult to be certain if the tracings represented true inhibition or artifact. However, each episode correlated with symptoms in the patient’s diary.

Fig. 12–15 Rate-adaptive pacemakers that utilize a minute ventilation sensor have been shown to interact with some electrocardiogram (ECG) monitors capable of documenting respiratory rate. In this example, upon connection to the monitor a paced tachycardia at a rate of 117 ppm occurs. This tachycardia stopped when the ECG leads were removed.
to return to this activity, the pacemaker clinician must consider the type of hardware implanted and the patient’s dependency status.

Testing methods have been designed to allow exposure of the patient with a pacemaker or ICD to progressively stronger fields of EMI. Although this testing is not practical for the individual patient, the study cited determined levels of interference at several programmed sensitivities (Table 12.7). This information could be applied to an individual patient if readings of EMI strengths in the work environment could be obtained.

**Non-industrial and home environments**

Many potential sources of EMI in the non-industrial and home environments are capable of one-beat inhibition of the pacemaker (Table 12.8). However, it would be unusual for any of these sources to cause EMI of clinical significance. It would also be unlikely that any of the devices in Table 12.8 could cause sustained EMI resulting in clinically significant interference with an ICD. However, anecdotal reports exist.

Although few sources can cause clinically significant EMI resulting in pacemaker malfunction, the potential for interference from cellular phones and electronic article surveillance equipment has been of intense interest because of possible public health issues. Before these are discussed in detail, several other potential sources, some of historical importance only, merit discussion.

**Microwave ovens**

One of the most common questions still asked by pacemaker recipients today is whether they can use a microwave oven. In many areas, signs are still in place warning the patient with a pacemaker not to use a microwave oven. The original warnings were posted because ineffective microwave shielding and less effective shielding of early pacemakers created the potential for pacemaker interference. Microwave ovens are no longer considered a significant source of interference, partly because they have effective shielding and interlocking circuitry that prevents them from being switched on while the door is open; moreover, significant advances have been made in shielding the pacemaker circuitry.

**Home induction ovens**

Induction cooktops have the potential to cause device interference. Investigations to date have established that patients at risk are those in whom the pulse generator is unipolar and implanted on the left side. It is most likely to occur in such patients if they are standing as close as possible to the cooktop and if the pan or pot is not concentrically lined up with the induction coil.

**Metal detectors**

This equipment is frequently mentioned as a potential problem for patients with implantable devices, and warning signs are often seen at airport security stations. Asynchronous pacing might occur for one or two beats without ill-effect to the patient. The major reason to warn patients about metal detectors is that the implanted device may “set off” the detector.

**Electronic article surveillance equipment**

The antitheft device (electronic article surveillance equipment) in many department stores and libraries consists of a tag or marker that is sensed by an electromagnetic field as the person walks through or by a “gate” (Fig. 12.17). Most systems consist of a “deactivator” that a cashier can use to remove or deactivate.

### Table 12.7 Electromagnetic interference levels in work environments capable of pacemaker interference

<table>
<thead>
<tr>
<th>Sensitivity setting, mV</th>
<th>Atrial*</th>
<th>Ventricular*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Unipolar</td>
<td>Bipolar</td>
</tr>
<tr>
<td>0.5</td>
<td>4509</td>
<td>17984</td>
</tr>
<tr>
<td>0.75</td>
<td>5744</td>
<td>20000</td>
</tr>
<tr>
<td>1.0</td>
<td>7679</td>
<td>20000</td>
</tr>
<tr>
<td>1.5</td>
<td>10143</td>
<td>20000</td>
</tr>
<tr>
<td>2.0</td>
<td>11790</td>
<td>20000</td>
</tr>
<tr>
<td>3.0</td>
<td>15034</td>
<td>20000</td>
</tr>
</tbody>
</table>

NA, not available.

*Values in milligauss units.
the tag after purchase of an item. This allows the customer to purchase an item and leave the store without activating an alarm. These electronic antitheft devices consist of multiple technological systems that generate electromagnetic fields in various ranges, including the radiofrequency range of 2 to 10 mHz, magnetic fields in the 50–100 kHz range, pulsed systems at various frequencies, and electromagnetic fields in the microwave range. Some systems continue to pose potential concern for the device patient.35

<table>
<thead>
<tr>
<th>Source</th>
<th>Pacemaker damage</th>
<th>Total inhibition</th>
<th>One-beat inhibition</th>
<th>Asynchronous pacing (noise)</th>
<th>Increased rate</th>
<th>Inappropriate shock</th>
<th>Possible inappropriate shock</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acupuncture</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Airport detector</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Antitheft equipment</td>
<td>N</td>
<td>?</td>
<td>Y</td>
<td>?</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Arc welder</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>Cardioversion</td>
<td>Y</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>Cautery, coagulation</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>Cellular phone</td>
<td>N</td>
<td>N</td>
<td>?</td>
<td>Y</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>CB radio</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>CT imaging</td>
<td>N</td>
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<td>Y</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
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<tr>
<td>Defibrillation</td>
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<td>N</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Diathermy</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>N</td>
<td>N</td>
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<tr>
<td>Drill, electric</td>
<td>N</td>
<td>N</td>
<td>Y†</td>
<td>N</td>
<td>Y‡</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>ECT, EST</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>Electric blanket</td>
<td>N</td>
<td>N</td>
<td>Y†</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Electric shaver</td>
<td>N</td>
<td>N</td>
<td>Y†</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
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<tr>
<td>Electric switch</td>
<td>N</td>
<td>N</td>
<td>Y†</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Electrolysis</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>N</td>
<td>N</td>
<td>Y†</td>
<td>N</td>
</tr>
<tr>
<td>Electrotonome</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
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<tr>
<td>Ham radio</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Heating pad</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Lithotripsy</td>
<td>Y†</td>
<td>Y†</td>
<td>Y†</td>
<td>Y‡</td>
<td>Y‡</td>
<td>Y§</td>
<td>N</td>
</tr>
<tr>
<td>Metal detector</td>
<td>N</td>
<td>N</td>
<td>Y†</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Microwave</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
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<td>N</td>
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<tr>
<td>MRI</td>
<td>Y</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
</tr>
<tr>
<td>PET scanner</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
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<tr>
<td>Power line</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Radar</td>
<td>N</td>
<td>N</td>
<td>Y†</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Radiation, Dx</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>N</td>
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<td>N</td>
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<td>Radiation, Rx</td>
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<td>N</td>
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<td>RF ablation</td>
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<td>N</td>
<td>Y</td>
<td>N</td>
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<td>TENS</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
</tr>
<tr>
<td>TV remote</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Ultrasound, Dx</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
</tbody>
</table>

CB, citizens band; CT, computed tomography; Dx, diagnostic; ECT, electroconvulsive therapy; EST, electroshock therapy; MRI, magnetic resonance imaging; N, no; PET, positron emission tomography; RF, radiofrequency; Rx, therapeutic; TENS, transcutaneous electrical nerve stimulation; TV, television; Y, yes.

*Impedance-based pulse generators.
†Piezoelectric crystal-based pulse generators.
‡Remote potential for interference.
§DDD mode only.
¶Capable of reprogramming the device.
In the Study of Pacemakers and Implantable Cardioverter-Defibrillator Triggering by Electronic Article Surveillance Devices (SPICED TEAS), 33 patients, in whom 18 pacemakers and 17 ICDs had been implanted, were exposed to six different electronic article surveillance (EAS) detectors.36 Of the six, three were radiofrequency devices, one was magnetoacoustic, and two were magnetic. No reprogramming of or damage to pulse generators was noted. Sixteen of the pacemakers demonstrated noise reversion or inhibition when they were exposed to a magnetoacoustic system at a range < 18 in. Reprogramming the sensitivity of the pacemaker could not abolish this effect. In addition, one epicardial unipolar pacemaker exhibited inhibition or noise reversion in each magnetic device. No EMI effects on any of the ICDs were demonstrated. No EMI was detected in any patients during exposure to the radiofrequency system.

In a multicenter study, 38,980 patients were tested with as many as six phones for a total of 55,333 phone exposures. A highly variable incidence of interference was observed. The overall incidence of interference, 20%, was high, but to quote this single percentage out of context would be misleading clinically. Interference at the “normal” use ear position was very low, and none was clinically significant, supporting the safety of “normal” use. The incidences of interference and, specifically, clinically significant interference were also would have occurred had the exposure continued. The authors concluded that it is safe for a patient with an ICD to walk through an EAS system, but that lingering could result in an inappropriate ICD discharge.

It is reasonable to advise patients to pass rapidly through any obvious EAS equipment and avoid leaning on or standing near the EAS equipment; that is, “Don’t linger, don’t lean.”

Cellular phones
A great deal of literature exists on cellular phones and the potential for pacemaker or ICD interference.36–41 At least one very early case report detailed injury that occurred when a pacemaker-dependent patient used a digital cellular phone.41 In a multicenter study,980 patients were tested with as many as six phones for a total of 5333 phone exposures. A highly variable incidence of interference was observed. The overall incidence of interference, 20%, was high, but to quote this single percentage out of context would be misleading clinically. Interference at the “normal” use ear position was very low, and none was clinically significant, supporting the safety of “normal” use. The incidences of interference and, specifically, clinically significant interference were also...
highly variable by combination of phone type, pacemaker manufacturer and pacemaker model. When one phone (not commercially available) was eliminated from the analysis, the incidences of interference and clinically significant interference dropped significantly to 13.1% and 2.8%, respectively (Fig. 12.18).

Although symptoms were present during 7.2% of the phone exposures, most were due to palpitations. The incidence of interference was highly variable by pacemaker manufacturer. Even for a given manufacturer, the incidence varied by pacemaker model, reflecting the effect of design on susceptibility to interference.

The highest incidence of interference occurred when the phone was directly over the pacemaker. Although this position is possible if the activated phone were carried in a pocket directly over the pacemaker, it is certainly not a "normal" phone use position and could be consciously avoided. As stated earlier, minimal interference occurred at the ear position. Most adverse effects are eliminated if the phone is kept 8–10 cm from the implanted device.

Even though specific changes in pacemaker design, such as feed-through filters, have significantly reduced rates of interference, new phone technologies could result in the potential for pacemaker interference, thus requiring further testing.

Fewer data exist on ICD interference from cellular phones. In a study of 43 ICD patients, no significant interference was seen with cellular phones. As with pacemakers, a number of complex variables including cellular technology (analog, PCS, CDMA, TDMA, GSM, Bluetooth, etc.), relative position of phone and ICD, and ICD technology (integrated vs. true bipolar leads, parameter settings including sensitivity, and others) govern the interactions between the cellular phone and the ICD. As a practical approach, advising patients not to place active cellular phones in pockets overlying the ICD and to preferentially speak with the phone using the contralateral ear make the risk of clinically significant EMI quite low.

**Therapeutic radiation**

Therapeutic radiation does not technically result in EMI. However, as an external source of potential damage to an implantable pulse generator, it is incorporated in this chapter. The dose of radiation used in diagnostic X-ray procedures, including coronary angiography, barium enemas and cerebral angiography, for example, does not significantly affect pulse generator function either immediately or cumulatively. However, computed tomography has been demonstrated to cause oversensing in implantable devices both *in vivo* and *in vitro*.

Therapeutic radiation can cause failure in contemporary pacemakers that incorporate complementary metal oxide semiconductor (CMOS) integrated circuit technology. ICDs have also been shown to fail when exposed to radiation. Radiation therapy may also result in inappropriate ICD discharge. Radiation therapy can also result in ICD "reset." The mechanism is believed to be a secondary neutron cloud that can interfere with the "memory cell" of the device and cause reset of the device. Some ICDs will result in a "patient alert" that therapies have been programmed "off." As long as the "reset" is recognized, the device can be reprogrammed to pre-existing parameters and remain in service.

The amount of therapeutic radiation that causes a device to fail is unpredictable and may involve changes in sensitivity, amplitude, or pulse width; loss of telem-
etry; failure of output; or runaway rates. If dysfunction occurs, device replacement is required. Although some changes may resolve in hours to days, the long-term reliability of the pacemaker is suspect, and it should be replaced. It should be emphasized that radiation therapy to any part of the body away from the site of the pulse generator should not cause a problem with the generator, but it should be shielded to avoid scatter. Centers that perform therapeutic radiation should have a protocol for patients with pacemakers or defibrillators. Before radiation begins, the device should be identified and evaluated. The most common clinical situation is development of malignant disease of the breast on the ipsilateral side in a patient with a permanent pacemaker. The pacemaker must be moved out of the field of radiation, because shielding the pacemaker would result in suboptimal radiation therapy. The pacemaker can be explanted and a new system implanted on the contralateral side. Alternatively, it is often possible to explant the pacemaker, tunnel the existing long-term pacing lead through the subcutaneous tissues to the contralateral side, and form a new pacemaker pocket on the contralateral side. The pacemaker is reattached to the now-tunneled lead and reimplanted (Fig. 12.19).

Clinical advice

Nearly all patients can be reassured that EMI will not affect their pacemaker, ICD or CRT device during the course of daily life. Patients in specialized industrial environments should be assessed individually. Improvements in pulse generator resistance to EMI should continue to minimize clinical concerns. However, the potential for EMI should never be taken lightly, and appropriate screening and monitoring should be done to avoid adverse clinical outcomes. In addition, despite improvements in pulse generator susceptibility to EMI, emerging technological advances result in new challenges for the patient with an implanted arrhythmia-control device. Newer wireless technologies or any technology with electromagnetic potential must be assessed for potential interference with implantable pulse generators.

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CHAPTER 12  Electromagnetic Interference and Implantable Devices


The complexity of device follow-up for pacemakers, implantable cardioverter-defibrillators (ICDs) and cardiac resynchronization therapy (CRT) systems has paralleled the increasing sophistication of the devices. Follow-up therefore requires a dedicated staff with a thorough understanding of implantable devices. Traditionally, the purpose of device follow-up has been to ensure appropriate device function. Increasingly, implanted devices record physiological information to permit assessing health status, with the goal of notifying caregivers before clinical decompensation occurs to permit intervention.

Follow-up of permanent pacemakers can be accomplished in more than one way: periodic visits to a pacemaker clinic, less frequent clinic visits in combination with transtelephonic monitoring (TTM), and/or surveillance via Web-based downloads. Follow-up of ICDs can likewise be accomplished in more than one way: periodic visits to an ICD clinic or less frequent visits in combination with remote access monitoring. In addition to routine checks, patients are able to transmit data if they are symptomatic, limiting travel time, clinic visits and costs. The relatively recent addition of remote access monitoring represents the most significant advance in device follow-up in decades, and further development of this technology is underway. Implanted devices have become part of a network of sensors that record health information, which can also include external sensors such as a scale or blood pressure cuff. Physiological information is wirelessly transmitted to a hub, from which it can be processed and sent to clinicians.

In the USA, TTM and remote access monitoring in combination with periodic clinic visits is the most common follow-up method for larger centers. Some centers that have a limited number of patients with implanted devices prefer to see the patient in the office on a regular basis and avoid the need for TTM or remote access. In a very large follow-up center, follow-up by clinic visits only would quickly monopolize a large proportion of the clinical resources.

TTM can be performed by the implanting center or by a commercial monitoring firm. Follow-up solely by TTM is suboptimal, because some patients undergoing pacemaker implantation may not be seen again until battery depletion indicators appear. Periodic clinic visits allow thorough evaluation and, if necessary, alteration of output settings, rate-adaptive settings and other parameters. Few patients would obtain maximum efficiency of their pacemaker if it remained at nominal or initially programmed values for the life of the pulse generator.

Remote access monitoring requires implantation of a compatible device. With inductive systems the patient holds a wand over the device to activate remote monitoring: the patient unit is typically connected to a phone line or to the cellular phone network to transfer data via the internet to a privacy-protected Web site. Wandless systems incorporate a radiofrequency transmitter that is positioned so as to be in regular patient proximity, e.g., on a night stand. The implanted device is automatically wirelessly interrogated on daily basis. Physicians are provided device information through fax, telephone, and/or Internet. Furthermore, physicians have access to the Web site to follow routinely scheduled patient transmissions as well as patient-initiated transmissions. Alerts are generated by the system if a malfunction is detected, such as a high impedance suggesting lead failure, with various
levels of alerts, and modes and times of physician notification available.

Requirements for a device follow-up clinic

Space
Because of the specialized equipment necessary for pacemaker ICD and CRT assessment and follow-up, dedicated space is desirable. There must be adequate space for:
- Patient assessment, programming and storage of all necessary programmers (Fig. 13.1)
- Electrocardiographic monitoring
- Space for informal exercise both for assessment of rate response and determination of appropriate rate-adaptive parameters and 6-min hall walk tests that may be used for follow-up of CRT patients
- Teaching tools, e.g., written educational information, DVDs, DVD recorder and screen, heart models (Figs 13.2 and 13.3)
- Transtelephonic receiving station or stations (the number of stations required is proportional to the overall volume of calls received.) (Figs 13.4 and 13.5)
- Remote access Internet stations to receive and review patient transmissions
- Record storage (even though most storage may be accomplished by computerized databases, in many centers some room is required for “paper storage”)
- Resuscitative equipment.

Personnel
Personnel requirements in the pacemaker clinic include:
- Allied professionals with expertise in pacemaker and ICD programming and follow-up [among them may be registered nurses, specially trained technicians, certified technologists (in some countries), physician assistants, and nurse practitioners.]
- Secretarial support.

Equipment
Equipment requirements include:
- Electrocardiographic (ECG) monitoring (this could be accomplished by the programmers. However, be-
cause some older programmers do not have this capability and independent monitoring is helpful in some situations, multichannel ECG monitoring should ideally be available.) Full 12-lead electrocardiography remains useful for assessment of CRT devices.

Fig. 13.2 Various teaching materials available in the outpatient area: we have an educational DVD about pacemakers and another about implantable cardioverter-defibrillators (ICDs). The patients see the DVD at the time of the device implant and are sent home with one for future reference. Also pictured are brochures that educate the patient about the basics of pacemakers, ICDs and cardiac resynchronization therapy devices.

Fig. 13.3 The patient is also sent home with a folder that includes educational brochures and a cover note reiterating postimplant restrictions, future transmission dates and important phone numbers related to device follow-up.
Fig. 13.4 Work area for nurses performing device follow-up, including transtelephonic monitoring. With digital storage of electrocardiographic tracings, reliance on the electronic medical record and availability of technical manuals on-line, the computer terminal and phone are the major requirements.

Fig. 13.5 Mayo Clinic Rochester has a large device follow-up area. Depicted here is the area where transtelephonic and remote monitoring is performed with stations for up to eight RNs.
- Programmers for all devices followed (Fig. 13.1)
- Reclining chair or examining table for patient evaluation (Fig. 13.1)
- Resuscitative equipment, including external cardioversion-defibrillation and external (transcutaneous) pacing
- Transtelephonic receiving station or stations
- Remote access Internet stations
- Access to technical manuals for all devices being followed. (Although most information can be obtained by calling the technical service department for the specific manufacturer, there should be access to manuals either via Internet-based resources or "hard-copy" of manuals.)

**Pacemaker follow-up**

**Transtelephonic monitoring**

TTM has been part of pacemaker follow-up since approximately 1970 (Fig. 13.6).² For many years, this follow-up method was used primarily in the USA and never gained significant popularity in other countries. Although still used widely in the USA, this technique will probably eventually be largely replaced by Web-based remote access.

TTM is an effective method to monitor pacemaker battery status and to demonstrate normal or abnormal function. Admittedly, transtelephonic assessment of atrial events is much more difficult than assessment of ventricular events. In large part the reason is simply the small amplitude of the atrial signal, whether paced or intrinsic. The pacemaker artifact may overwhelm the atrial event, whereas the usually larger ventricular event is not commonly overshadowed by the ventricular pacing artifact.

Obtaining TTM tracings of good quality is also an issue. Patients with pacemakers are often elderly, and without excellent initial teaching and possibly coaching during the TTM calls, they may have difficulty handling the transtelephonic equipment. We request that a

![Fig. 13.6 Typical equipment used by the patient for transtelephonic monitoring. In addition to the telephone, the patient requires a transmitter and electrodes. Various types of electrodes can be used. During electrocardiographic transmission, the patient is instructed to transmit for approximately 30 s. However, if the transmission needs to be interrupted for any reason, an alarm can be sent from the receiving center. If the patient hears the alarm, he or she is instructed to stop the transmission attempt and pick up the phone.](image-url)
family member or friend be present during the initial teaching session. It is often reassuring to the patients to know that someone else has the information necessary to complete the transmission should they forget a portion of the instructions. Transmission difficulties may be compounded if the patient has a significant hearing deficit. Incorrect use of the transtelephonic transmitter and improper magnet placement may impair the quality of the transmission (Fig. 13.7).

Some types of telephones may be suboptimal for TTM. For example, with cordless phones, the quality of transmission at times is decreased and there is sometimes a greater chance of being disconnected. Speaker phones and phones with altered volume controls may also present problems. Any source of electromagnetic interference close to the site of the patient’s transtelephonic transmission may induce significant artifacts (Fig. 13.8). However, with the popularity of cell phones, technology is being further developed to allow for cell phone transmission.

The frequency of TTM differs among centers. The Health Care Financing Administration [HCFA, now Center for Medicare and Medicaid Services (CMS)] established guidelines for pacemaker follow-up and reimbursement for follow-up in 1984 that have not been updated since that time. At the time of writing, major professional societies with an interest in device follow-up are establishing new guidelines for remote monitoring. It is likely that once the guidelines are endorsed by the professional societies they will be assessed critically by CMS and may serve as the basis for an update in CPT (Current Procedural Terminology).

\[\text{Fig. 13.7} \quad (A) \text{Upper tracing obtained during magnet application in a patient with a pacemaker programmed to a dual-chamber mode. Only the ventricular pacing artifact is seen. This tracing was obtained with the patient using "wrist bands" (bracelets) for the transmission. (B) Lower tracing obtained from the same patient but using a chest lead, i.e., removing the wrist band from the wrist and establishing contact between the electrode of the wrist band and the chest. Even though there is significant artifact present, both the atrial and ventricular pacing artifacts can now be seen.}\]
codes and reimbursement models for device monitoring and follow-up.

At this time reimbursement for TTM generally is not allowed for more frequent follow-up than that specified by the CMS guidelines. The guidelines that continue to be used at this time are shown in Table 13.1.

**Equipment**

To perform TTM, the patient must have access to the necessary TTM equipment. Typical transmitting equipment is shown in Fig. 13.6. Transmission requires contact with the patient’s skin, i.e., electrodes on the wrists or the chest. After calling the pacemaker clinic or commercial follow-up center, the patient places the telephone over the transmitting equipment.

In the pacemaker clinic, a receiving center is used to obtain the ECG tracings the patient transmits (Fig. 13.4).

For those centers with an electronic device database, multiple systems exist, including institution-designed, institution-specific systems; the most commonly used commercial system is the Medtronic Paceart® System. The Paceart® System will organize and archive relevant patient, device, programmer, transtelephonic, and remote management system information for follow-up of arrhythmia patients with implanted cardiac devices. Paceart® supports a common workflow by managing information for all leading manufacturers’ devices and serves as the link to electronic health record systems.

Paceart® standardizes patient and device information. The report format is consistent across all manufacturers’ devices (Fig. 13.9). It provides active device and lead information, current programming, data/telemetry information and trending device performance. The system also provides functionality for creating physician correspondence (Fig. 13.10) and scheduling pa-
CHAPTER 13 Follow-up

579

Patient follow-ups, for both in-clinic and remote systems (Fig. 13.11). The system will also assist in charge and billing management and provide documentation of in-clinic and remote patient management activities.

Transtelephonic monitoring sequence

A significant amount of information can be obtained transtelephonically. The order in which the information is collected may vary. The following sequence is used by our clinic.

- Brief discussion with the patient to determine general well-being and elucidate any problems the patient believes may be related to the pacemaker
- Nonmagnet “free-running” tracing (duration, 30 s)
- Magnet tracing (duration, 30 s)
- Patient informed of pacemaker status; next TTM transmission or clinic visit scheduled
- TTM data stored.

If the nonmagnet tracing displays intrinsic rhythm, the underlying rhythm should be noted and compared with previous transmissions. If there is intermittent pacing or pacing in only one chamber of a dual-chamber device, sensing can be assessed.

The magnet tracing should be used to assess
- Capture (for dual-chamber devices, capture should be determined for both chambers)
- Magnet rate
- Pulse width (for dual-chamber devices, pulse width should be determined for both chambers).

Magnet response varies not only from manufacturer to manufacturer but also among models from one manufacturer (Fig. 13.12). Specific magnet response for many different devices is difficult to commit to memory. Therefore, it is helpful to have the specific magnet response recorded on the patient’s records.

The healthcare professional taking the transmission should be familiar with the elective replacement indicators for the specific pacemaker (Table 13.2). Measurements from the patient’s previous transmission should be available for comparison.

Other specific information can be obtained from specific pacemakers. The proprietary Threshold Margin Test (TMT) provides some information on pacing threshold at the onset of magnet application (Fig. 13.12). The first three pacemaker artifacts occur at a rate of 100 bpm. As part of the TMT, the pulse duration of the third pace-

<table>
<thead>
<tr>
<th>Table 13.1 Transtelephonic monitoring guidelines</th>
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<tbody>
<tr>
<td><strong>Category I</strong> <em>(These apply to most contemporary pacemakers)</em></td>
</tr>
<tr>
<td>Single-chamber pacemakers:</td>
</tr>
<tr>
<td>• 1st month—every 2 weeks</td>
</tr>
<tr>
<td>• 2nd through 36th month—every 8 weeks</td>
</tr>
<tr>
<td>• 37th month to failure—every 4 weeks</td>
</tr>
<tr>
<td>Dual-chamber pacemakers:</td>
</tr>
<tr>
<td>• 1st month—every 2 weeks</td>
</tr>
<tr>
<td>• 2nd through 6th month—every 4 weeks</td>
</tr>
<tr>
<td>• 7th through 36th month—every 8 weeks</td>
</tr>
<tr>
<td>• 37th month to failure—every 4 weeks</td>
</tr>
<tr>
<td><strong>Category II</strong> <em>(These apply only to pacemaker systems (pacemaker and leads) for which sufficient long-term clinical information exists to assure that they meet the standards</em> of the Inter-Society Commission for Heart Disease Resources (ICHD) for longevity and end-of-life decay)*</td>
</tr>
<tr>
<td>Single-chamber pacemakers:</td>
</tr>
<tr>
<td>• 1st month—every 2 weeks</td>
</tr>
<tr>
<td>• 2nd through 48th month—every 12 weeks</td>
</tr>
<tr>
<td>• 49th through failure—every 4 weeks</td>
</tr>
<tr>
<td>Dual-chamber pacemakers:</td>
</tr>
<tr>
<td>• 1st month—every 2 weeks</td>
</tr>
<tr>
<td>• 2nd through 30th month—every 12 weeks</td>
</tr>
<tr>
<td>• 31st through 48th month—every 8 weeks</td>
</tr>
<tr>
<td>• Thereafter—every 4 weeks</td>
</tr>
<tr>
<td><strong>Mayo Clinic Guidelines</strong> <em>(Same guidelines apply to single- and dual-chamber pacemakers)</em></td>
</tr>
<tr>
<td>Single-chamber pacemakers:</td>
</tr>
<tr>
<td>• 1st month—every week</td>
</tr>
<tr>
<td>• 2nd month until first signs of battery depletion—every 3 months</td>
</tr>
<tr>
<td>• Onset of battery depletion to elective replacement indication—every 4 weeks</td>
</tr>
<tr>
<td>Dual-chamber pacemakers:</td>
</tr>
<tr>
<td>• 1st month—every 2 weeks</td>
</tr>
<tr>
<td>• 2nd through 30th month—every 12 weeks</td>
</tr>
<tr>
<td>• 31st through 48th month—every 8 weeks</td>
</tr>
<tr>
<td>• Thereafter—every 4 weeks</td>
</tr>
</tbody>
</table>

*The ICHD standards are (i) 90% cumulative survival at 5 years after implantation and (ii) an end-of-life decay of <50% decrease in output voltage and <20% deviation in magnet rate, or a decrease of ≤5 bpm, over ≥3 months.
Fig. 13.9 Pacert® report format providing active device and lead information, current programming, data/telemetry information and trending device performance.
maker stimulus is 75% of the programmed pulse duration. Failure to capture with the reduced pulse duration provides some information about threshold and pacing margin of safety.

Pacemaker clinic follow-up visit

The detail required during the pacemaker clinic visit depends on the follow-up technique or techniques used. If the pacemaker is not capable of Internet-based remote monitoring, our practice is to see the patient in the pacemaker clinic approximately 3 months after implantation, at yearly intervals, and at any time a problem is noted by TTM or the patient has a concern that may be pacemaker-related. At the time of each pacemaker clinic visit, the following steps are completed.

- Retrieval of previous data and follow-up records
- Discussion and interview with the patient
- Interrogation of the pacemaker
- Assessment of stored data (Fig. 13.13)
- Programming sequence
- Assessment of rate-adaptive parameters as needed
- Radiographic assessment if there are clinical concerns that might be addressed by a radiographic evaluation
- Data storage

Retrieval of previous data and records

At the onset of the pacemaker clinic visit, records should be available. These should include information from the patient’s previous clinic visits and most recent trans telephonic transmissions.

Discussion and interview

The patient should be interviewed in an attempt to elucidate any clinical problems that could potentially be related to pacemaker-related problems (Table 13.3). It is important to have some knowledge of the most
commonly noted pacemaker problems. These are discussed in detail in Chapter 10.

At our institution, the pacemaker clinic does not serve as the primary healthcare or cardiac care provider, and the extent of the physical examination is related to suspected problems. For example, if no clinical problem is suspected, the examination is limited to inspection of the pacemaker site.

At other centers, the pacemaker clinic may serve as the primary healthcare provider and may therefore provide a complete physical examination at periodic visits.

**Assessment of stored data**

In some pacemakers, initial interrogation results in a printout of stored data. In other pacemakers, these data must be specifically requested. It may be necessary to obtain stored data before programming, because a permanent change in programming may “clear” stored data.

Contemporary pacemakers may have the capability of storing a great deal of data. Although the storage capabilities increase with each successive pacemaker generation, at the time of manuscript preparation, devices have the ability to store up to 512K bytes of data. The data may provide invaluable assistance in achieving optimal programming and in diagnosing intermittent symptoms. In a large-scale, randomized, prospective study, the results of which are available in abstract only, the clinical use of pacemaker diagnostic data led to more and earlier diagnoses, less excessive testing when they presented for care, and had a decreased number of emergency room visits and hospitalizations compared with those patients whose diagnostic functions were not evaluated.4 The limitations of stored diagnostic function include low sensitivity and specificity as well as oversensing and undersensing. Stored intracardiac electrograms (EGMs) are a critical component in confirmation of stored diagnostic data and follow-up of stored events.
Fig. 13.12 Composite of magnet responses from three pacemakers. Magnet response, top to bottom: (A) Pacemaker goes to 100 bpm for three beats (Threshold Margin Test) followed by asynchronous pacing at programmed lower rate. (B) Asynchronous pacing at programmed lower rate. (C) Asynchronous pacing at 98 bpm.

Table 13.2 Beginning and end of service characteristics for major manufacturers

<table>
<thead>
<tr>
<th>Manufacturer</th>
<th>BOL</th>
<th>ERI</th>
<th>EOS</th>
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<tbody>
<tr>
<td>Biotronik</td>
<td>90bpm</td>
<td>80bpm</td>
<td>No output</td>
</tr>
<tr>
<td>Boston Scientific</td>
<td>100bpm</td>
<td>85bpm*</td>
<td>&lt;85bpm</td>
</tr>
<tr>
<td>Medtronic</td>
<td>65bpm§</td>
<td>No output</td>
<td></td>
</tr>
<tr>
<td>St Jude Medical</td>
<td>100bpm</td>
<td>85bpm*</td>
<td>&lt;85 bpm</td>
</tr>
<tr>
<td>Sorin/ELA</td>
<td>96bpm</td>
<td>80bpm</td>
<td>70bpm</td>
</tr>
</tbody>
</table>

* Refer to ‘magnet mode’ unless otherwise indicated.
† Elective replacement indicators.
‡ End of service.
§ 65 bpm nonmagnet and VOO 3 beats at 100 and then 65 bpm with magnet; there are specific Medtronic pulse generators that are exceptions to this, and the technical manual should be used to determine ERI response.
BOL: beginning of life.
Categories of stored information include:
- Event counters (Fig. 13.14)
- Rate histograms (Fig. 13.15)
- Electrograms (Fig. 13.16)
- Measured values (Fig. 13.17)
- Special diagnostic features (Fig. 13.18).

**Programming sequence**
A specific sequence should be adopted and followed for programming. This sequence is discussed in detail in Chapter 8. It is not important that this specific sequence be followed, but it is crucial that all steps be completed in some orderly manner to avoid deletion of any necessary steps.

**Rate-adaptive parameter programming**
As discussed in Chapter 9, the patient’s rate requirements may change over time. For example, in the chronotropically incompetent patient in whom rate response is restored, the newly found rate response...
may allow the patient to begin an exercise program and improve conditioning. With subsequent improvement in conditioning, a change in rate-adaptive parameters may be desired, such as higher paced rates and a faster increment in heart rate. Conversely, if symptomatic coronary artery disease were to develop in a patient with a rate-adaptive pacemaker, it may be desirable to make the rate-adaptive parameters less sensitive to avoid rate-related angina while the coronary artery disease is being evaluated and treated.

We routinely assess exercise informally at the first clinic visit, i.e., at approximately 3 months postimplant in most patients. On subsequent visits the rate response would be reassessed if stored rate histograms suggest that the patient is achieving a less than optimal rate distribution. If the rate histogram suggests suboptimal rate response, the rate-adaptive parameters are reprogrammed before informal exercise. The patient is also questioned about normal and desired activity levels. This is especially important the first time rate-adaptive parameters are initiated. However,
Fig. 13.16 Telemetered intracardiac electrograms from a patient with a biventricular pacemaker programmed to the DDDR mode. The atrial electrogram documents an atrial tachyarrhythmia. Biventricular pacing occurs at a regular rate consistent with mode-switching.

Fig. 13.17 Telemetered report of battery voltage, battery impedance, lead impedance and intrinsic amplitude measurement.
since activity levels change—increase with better conditioning and improvement in well-being or decrease because of associated medical problems—it is wise to inquire about any change in activity before assessing and possibly changing rate-adaptive parameters.

Some CRT devices provide an activity log, i.e., objective evidence of the patient’s activity level. This is especially important in patients with heart failure, because decreasing activity levels may presage the onset of clinical congestive heart failure (Fig. 13.19).

Informal exercise, when needed, can be accomplished in multiple ways depending on available equipment and monitoring. If telemetry is available then the patient should be connected to a wireless
monitor and asked to walk at a pace that feels "casual" for a minimum of 2 min. Rates are assessed via telemetry throughout the walk. If the paced rate achieved is felt to be inappropriately low or high for the patient, then parameters can be adjusted and the walk repeated. If the rate response during the "casual" walk seems appropriate for the patient and if the patient is capable of and describes more vigorous exercise as part of his or her daily activities, the process can be completed with the patient maintaining what they perceive as a "brisk" or "vigorous" pace. Paced rates are again assessed during this period. For any exercise assessment, formal or informal, the "onset" of the rate change as well as the rate response post-exercise should be assessed. The onset and offset of the rate-adaptive sensor is programmable in most devices and can be adjusted if the onset or offset appears too brisk or too slow.

Fig. 13.19 Partial printout from a cardiac resynchronization device with information regarding the number of hours of activity/day. In addition, there is information regarding the average ventricular rate, heart rate variability and % pacing/day.
If wireless telemetry is not available, the patient can be connected to the transtelephonic receiver by wrist electrodes to allow single-lead ECG monitoring and the cable disconnected from the receiver, bracelets left in place, and the patient asked to hold the end of the cable. The patient is then asked to perform the type of walk(s) described above. At the end of the walk, the cable is immediately plugged in, and with the receiving mode at “standby,” the ECG can be obtained at once and heart rate recorded that should be near the peak heart rate achieved. If telemetry is available, it is the preferred method of monitoring the patient during exercise. Alternatively, the rate histogram can be assessed for the focused period of informal exercise.

For the patient who exercises vigorously, formal exercise may be important. If formal (treadmill) exercise is performed and the rate-adaptive pacemaker being optimized has an activity sensor, the patient should avoid holding on to the treadmill. Holding the treadmill railing may blunt the sensor response and lead to inappropriate programming (see Chapter 8). Exercise protocols are shown in Fig. 13.20.

**Radiographic assessment**

Radiographic assessment of the pacemaker or ICD may provide critical information. This is discussed in detail in Chapter 11, and additional comments on radiography of ICDs are made later in this chapter. 

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**Fig. 13.20** (A) Exercise protocols including Bruce Protocol, Chronotropic Assessment Exercise Protocol (CAEP) and Naughton protocols are compared. (B) The CAEP protocol is preferred by some for assessment of chronotropic response. (From Hayes DL, Lloyd MA, Friedman PA. Cardiac pacing and defibrillation: a critical approach. Armonk: Futura Publishing Company Inc; 2000. p. 32546. Copyrighted and used with permission of Mayo Foundation for Medical Education and Research.)
do not routinely obtain a chest radiograph for every visit to the device clinic. For the patient with any implanted device, a chest X-ray is obtained in the event a clinical problem occurs and it is felt that an X-ray may be valuable in the evaluation and management of the patient or before any invasive procedure, such as replacement of the pulse generator.

**Data storage**

It is critical that the programmed data and battery and lead measurements be stored in some manner for future reference. A computerized database is preferable, but if a relatively small number of patients are being followed, paper storage may be manageable.

Although our data storage is entirely electronic, printouts of measured data and initial and final programmed parameters can be posted in a permanent record to allow comparison with subsequent pacemaker evaluations. This method may work well for centers with smaller volumes of patients. For large numbers of patients, paper storage becomes cumbersome. Computer storage of data is more efficient. The data can be entered into various data screens, or with some programs it may be possible to download data directly from the pacemaker programmer to the database.

If patient data are computerized, several additional functions may be available for data management, including

- Keeping track of follow-up schedules
- Automatic reminders of patients delinquent in follow-up
- Ability to query for outcome data or to assess performance of specific leads or pulse generators
- Billing functions.

**Implantable cardioverter-defibrillator follow-up**

Follow-up of patients with implantable defibrillators is in many respects similar to that of patients with pacemakers. Indeed, with the progressive integration of pacemaker and defibrillator technology, assessing the “pacemaker” function of the defibrillator has become a standard part of ICD evaluation. ICD follow-up has become more convenient for the patient and more efficient with the advent of remote access monitoring. The goals of ICD follow-up include assessment of patient health status, confirmation of system integrity and function, and ensuring optimal device-specific programming to prevent heart failure and minimize shocks. The approach to ICD follow-up is summarized in Table 13.4. Specific issues relating to the follow-up of patients with resynchronization devices are discussed further, below.

**Assessment of patient clinical status**

The vast majority of patients with an ICD have significant structural heart disease (Fig. 13.21), most commonly coronary artery disease followed by dilated cardiomyopathy. Since pharmacological therapy can significantly reduce mortality in patients with structural heart disease, an in-clinic evaluation provides an opportunity for review of medical status and for providing or arranging for additional medical evaluation as needed. Knowledge of the patient’s medical status may also guide management of arrhythmias (e.g., whether to add a β-blocking agent or reprogram an ICD to manage atrial fibrillation with rapid ventricular response). Assessment of clinical status is aided by the use of device-based physiological data. Information such as the presence of atrial or ventricular arrhythmias, the degree of physical activity, estimates of lung water and other measures may inform medication prescription or parameter adjustment (Fig. 13.22).

The role of diuretics, angiotensin-converting enzyme (ACE) inhibitors and β-adrenergic blockers in the management of ischemic heart disease and heart failure is well established. If a device clinic typically lacks the resources to make significant medical adjustments during a device check visit, appropriate referrals can be made.

**Pulse generator assessment**

The principle purpose of ICD generator assessment is to ensure that an adequate charge remains in the battery. Battery depletion is the single most common indication for device replacement. Early devices used lithium vanadium pentoxide batteries, which maintained a constant voltage throughout their lifetimes when under a low current load. Therefore, battery status was indirectly assessed by charging the capacitor and recording the charge time. ICDs now use lithium silver vanadium oxide chemistry, in which the unloaded voltage provides a reasonable estimate of remaining battery life. Battery voltages gradually decline from beginning of life (BOL), to middle of
Table 13.4 Routine follow-up evaluation of implantable cardioverter-defibrillators

| Assessment of patient clinical status | Interval history (myocardial infarction, new heart failure, syncope) Changes in medications, particularly anti-arrhythmic drugs Device-based logs of physical activity and physiology |
| Pulse generator assessment | Remaining battery life Capacitor formation and charge time Advisories/recalls (in many devices checked via manufacturer Web page by entering specific device serial number) Presence of any alerts |
| Lead status | Real time telemetry with maneuvers Evidence of lead failure on stored electrograms Radiographic assessment (if there is a sign of abnormality) Electrogram amplitudes, thresholds and impedances Parameter alerts (impedance) |
| Patient-specific programming and therapy | RV ventricular pacing minimized for non-CRT devices (ideally < 10%, consider intervention if >40%) For patients with CRT devices, assessment of response Defibrillation efficacy • Identify patients at risk for failed shocks • Device–device interactions • Specific situations suggesting additional testing • Medication effects |
| Strategies to minimize shocks | Sufficiently long detection in patients with nonsustained episodes Use of antitachycardia pacing Absence of T-wave oversensing on ventricular lead and far-field R-wave oversensing on atrial lead Appropriate use of detection enhancements |

Fig. 13.21 Heart disease in ICD recipients at Mayo Clinic. CABG, coronary artery bypass graft; CAD, coronary artery disease; HCM, hypertrophic cardiomyopathy; IDCM, idiopathic dilated cardiomyopathy; MI, myocardial infarction. (Modified from Trusty JM, Hayes DL, Stanton MS, Friedman PA. Factors affecting the frequency of subcutaneous lead usage in implantable defibrillators. Pacing Clin Electrophysiol 2000; 23:842–6. By permission of Futura Publishing Co.)

life (MOL), to appearance of the elective replacement indicator (ERI), and to end of life (EOL). Boston Scientific programmers show a "gas gauge" figure with labels BOL, ERI, and so on, graphically depicting the remaining useful battery life when the device is interrogated (Fig. 13.23A). Other manufacturers display the actual device voltage in tabular format, often with reference values for ERI and EOL (Fig. 13.23B). Since battery voltage may temporarily decline after a high-voltage charge, rechecking the unloaded voltage 24 h later may show return to normal.

The ERI voltage varies from manufacturer to manufacturer and even from model to model from the same manufacturer. The ERI voltage is a function of the power delivery of a particular battery, the current drain from the monitoring circuitry, and the capacitors used by the device. Thus, the ICD clinic must have readily available references indicating device-specific ERI voltages.
Once a battery is depleted to the ERI voltage, the time remaining until device malfunction varies, depending on the model, the degree of antibradycardia pacing, and the number of shocks delivered. Typically, plans are made for elective pulse generator replacement within no more than 3 months of ERI status,
and often sooner. To be certain that the ERI voltage is detected, patients are seen more often in clinic or are checked more frequently remotely as the voltage gets lower. Many devices will generate an audible tone or vibratory stimulus to alert the patient when ERI is reached. Patients are instructed to contact the clinic following any device alert.

In contrast to the ERI voltage, which is measured in the unloaded state, the EOL voltage is the minimum loaded voltage, recorded while the battery is maximally stressed. This usually occurs during capacitor charge. If interrogation shows that the voltage has declined to EOL at any time, this signifies that during a capacitor charge or other heavy current drain, the battery voltage dipped below acceptable levels. A device should be replaced before EOL is reached, and replaced immediately if an EOL voltage is seen, as subsequent function may be unreliable.

**Capacitor status**

Defibrillators require the use of capacitors to accumulate and store charge before shock delivery, since a bat-
battery is unable to deliver the high level of voltage and current needed over the shock interval. Implantable devices use electrolytic capacitors, since these have a high energy density and consequent small size. However, electrolytic capacitors develop relatively large leakage currents over time, which can be reduced by recharging (“reforming”) the capacitors. With early ICDs, patients had to be seen in the clinic every few months for capacitor reforming. All new devices offer either programmable or automatic capacitor reforming. In devices with programmable charge times, the charge frequency can depend on the specific device, the age of the device, or the most recent charge time (so that capacitor formation frequency is increased if the charge time becomes too long). “Smart” reforming devices recognize a full energy charge as a capacitor-reforming event. Generally, failure to reform capacitors with sufficient frequency can result in significant delays for the first shock during therapy delivery; subsequent therapies in the same episode are not affected, because the capacitor is reformed after the first charge. Excessively frequent capacitor formation does not harm the device or its functionality, although battery depletion may be accelerated. With many devices, capacitor formation is as important for the battery as for the capacitor. If the battery is not “pulsed” periodically, internal resistance develops, which can lead to a voltage delay (and delayed capacitor charge) when the capacitor is charged to deliver a shock therapy.

For the clinician, the most pertinent capacitor information is the charge time, which is actually a measure of both battery and capacitor function; it is provided on device interrogation (Fig. 13.23A,B). Some manufacturers have used charge time to indicate pulse generator EOL. Although acceptable charge time can vary from device to device, a full capacitor charge generally should not exceed 15–20 s.

Implantable defibrillators generate alerts that highlight parameters that are out of range (Fig. 13.24). These alert the clinician to battery, capacitor, impedance (lead)
and other potential problems. These are discussed further below.

Periodically, manufacturers may issue advisories regarding specific devices if evidence emerges that they fail to meet performance specifications or otherwise perform as intended. This is discussed in detail below in the section “Medical device advisories and recalls.”

Assessing lead function

Implantable defibrillators record serial measurements of lead impedance, and generate plots showing impedance trends (Fig. 13.25). As in pacemakers, elevated impedance suggests a conductor discontinuity, whereas low impedance suggests an insulation defect or short within the lead. Normal pacing impedance is a function of lead design, but for any lead (except for adapted lead combinations) impedance < 200 Ω indicates an insulation defect and impedance > 2000 Ω indicates conductor failure. A loose setscrew or faulty adapter can also cause abnormally high impedances. Normal shocking lead impedance ranges from 25 to 75 Ω for transvenous systems. Values outside this range suggest a conductor defect (high impedance) or insulation breach or short circuit (low impedance).

<table>
<thead>
<tr>
<th>Sound tone for:</th>
<th>Enable-Urgency:</th>
<th>Threshold:</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Pacing Lead Impedance Out of Range</td>
<td>On-High</td>
<td>&lt;200 ohms or &gt;2500 ohms</td>
</tr>
<tr>
<td>RV Pacing Lead Impedance Out of Range</td>
<td>On-High</td>
<td>&lt;200 ohms or &gt;2500 ohms</td>
</tr>
<tr>
<td>LV Pacing Lead Impedance Out of Range</td>
<td>On-High</td>
<td>&lt;200 ohms or &gt;2500 ohms</td>
</tr>
<tr>
<td>V. Defibrillation Lead Impedance Out of Range</td>
<td>On-High</td>
<td>&lt;20 ohms or &gt;2000 ohms</td>
</tr>
<tr>
<td>SVC Defibr Lead Impedance Out of Range</td>
<td>On-High</td>
<td>&lt;20 ohms or &gt;2000 ohms</td>
</tr>
<tr>
<td>Low Battery Voltage ERI</td>
<td>On-High</td>
<td>2.62 V(ERI)</td>
</tr>
<tr>
<td>Excessive Charge Time ERI</td>
<td>On-Low</td>
<td></td>
</tr>
<tr>
<td>Number of Shocks Delivered in an Episode</td>
<td>Off</td>
<td></td>
</tr>
<tr>
<td>All Therapies in a Zone Exhausted</td>
<td>Off</td>
<td></td>
</tr>
<tr>
<td>VF Detection Off, three or more VF Therapies Off</td>
<td>On-High</td>
<td></td>
</tr>
</tbody>
</table>

System Alert Time 10:00

Fig. 13.24 Programmable alerts. All current implantable cardioverter-defibrillators (ICDs) can generate alerts to warn of lead dysfunction, battery deterioration or other pertinent conditions. Patients may be notified of an alert condition by means of an audible tone or, in St Jude systems, a vibratory indicator. For ICDs with remote monitoring capability, some systems also include a patient Web page, at which alerts will be visible. In ICDs that are remotely monitored, physicians may be notified by fax, phone, page, Web notification or e-mail regarding alerts, depending on the programmable options and the particular monitoring system.

Fig. 13.25 Plot of high-voltage impedance of the superior vena cava (SVC) coil. The abrupt increase indicates lead malfunction. Since the SVC coil is used for shock delivery but not sensing, this type of malfunction does not cause inappropriate shocks, but can result in ineffective shock delivery.
During sinus rhythm, ventricular electrograms are generally > 5 mV to ensure appropriate sensing of ventricular fibrillation, which has variable and small amplitude electrograms. Automated capture assessment for implantable defibrillators may facilitate remote monitoring of thresholds.

Lead fracture often presents with characteristic non-physiological short intervals (< 150 ms) caused by make–break electrical contact noise. Their presence in stored episode recordings or during proactive maneuvers indicates lead failure (Fig. 13.26A). In the era before automated painless lead impedance tests, stored electrogram data were the most frequent indicators of lead malfunction10–13 (Fig. 13.26B). This occurred because the defibrillator’s sensing function provides continuous monitoring; if at any time enough electrical noise is created by failure to trigger detection of a “tachyarrhythmia” episode, it is recorded as a stored event or will declare itself as an inappropriate therapy. Intermittent automated impedance may permit detection before inappropriate therapies, but undetected malfunctions have been reported.14 The Medtronic sensing integrity counter detects short intervals and if the threshold of 300 is exceeded lead fracture, oversensing, or loose setscrew is suggested and the observation is highlighted at device interrogation (Fig. 13.23B).

System and patient characteristics exist that predict an increased risk for lead fracture. These include active use of epicardial leads,15,16 abdominal pulse generator location,17,18 coaxial defibrillation leads (Fig. 13.27),19 subclavian venous access (as opposed to cephalic access),19 and dual-chamber (as opposed to single-chamber) ICD systems.20 Patients with these characteristics warrant careful attention at follow-up. Risk factors for and evaluation of suspected lead malfunction are covered in greater detail in Chapter 10, Troubleshooting, which also reviews assessment of thresholds, provocative maneuvers and stored episode analysis in detail.

Radiography

Radiographic examination of the implantable defibrillator detects less than half of lead malfunctions in nonthoracotomy systems.19,21 It is useful for troubleshooting suspected or known lead malfunction. Due to radiography’s modest yield and improved, automated defibrillator impedance assessment, we do not routinely screen normally functioning ICD systems radiographically. The radiography of abnormally function systems is reviewed in Chapter 10, Troubleshooting and Chapter 11, Pacemaker, ICD and CRT Radiography.

Patient-specific programming and therapy

Ventricular pacing

Chronic right ventricular apical (RVA) pacing introduces ventricular dyssynchrony, and has been associated with increased risk for the development of atrial fibrillation and congestive heart failure.22–25 Moreover, RVA pacing appears to be poorly tolerated by patients with preexisting ventricular dysfunction and heart failure,26 with a correlation between the frequency of RV pacing and adverse outcomes.27–29 Since most ICD recipients have some measure of left ventricular dysfunction and heart failure, the frequency of pacing should be assessed at each follow-up. A pacing frequency of < 10% may be ideal, and a frequency > 40% may warrant intervention, although clinically validated guidelines are lacking.28 The frequency of pacing is displayed in device interrogation reports (Fig. 13.28).

In patients without a pacing indication, programming of ICD to backup low rate limit 40 bpm limits RVA pacing and its sequelae.29 In patients who need atrial pacing support (due to chronotropic incompetence or use of rate-slowing medications such as β-blockers and antiarrhythmic drugs), programming to a DDD pacing mode with a long AV delay, use of the AAI pacing mode, or use of specialized algorithms31,32 minimizes ventricular pacing. These approaches are discussed in detail in Chapter 8, Programming. If a high percentage of ventricular pacing in a patient with LV dysfunction is required, upgrade to a resynchronization device may be necessary if refractory, deteriorating heart failure intervenes.33,34

Follow-up of patients with CRT devices

Up to a third of patients who receive cardiac resynchronization devices fail to respond to therapy. Cardiac resynchronization results in numerous beneficial effects, including improved exercise tolerance, reduction in New York Heart Association class heart failure, reverse remodeling (including improvement in ejection fraction and reduction in mitral regurgitation and ventricular volume), improvement in quality of life, and
Fig. 13.26 (A) Real-time telemetry during provocative maneuvers. The patient had received a shock while doing physical labor. In clinic, while pressing hands together, noise is seen. From top to bottom are the far-field electrogram, marker channel, and near-field electrogram. Atrioventricular pacing occurs for the first three pulses. Pacing is interrupted and a pause occurs due to nonphysiological noise caused by make-break contact in a fractured lead (series of “VS” or ventricular sensed event). The noise is followed by an intrinsically conducted sinus beat (AR = atrial complex in refractory window, followed by VS). (B) Stored episode demonstrating inappropriate shock due to noise seen on both the far-field and near-field electrograms. After detection, the noise terminated. However, in Boston Scientific/Guidant devices before Prizm2™ the absence of spontaneous ventricular activity at charge end was treated as fine VF and shocked, necessarily committing pacemaker-dependent patients to shocks for nonsustained episodes that reach detection. This device operation has been modified in all implantable cardioverter-defibrillators from Prizm2™ onwards.
Fig. 13.27 Multilumen lead design (left) and coaxial lead design (right). Polytetrafluoroethylene (PTFE) is an insulation element. (Modified with permission from Friedman PA, Glickson M, Stanton MS: Defibrillator challenges for the new millennium: the marriage of device and patient—making and maintaining a good match. J Cardiovasc Electrophysiol 11:697–709, 2000.)

Fig. 13.28 Implantable cardioverter-defibrillator remote interrogation showing battery function, arrhythmic events, and percent paced. In this resynchronization device, note that ventricular pacing frequency is 99%, which is satisfactory assuming appropriate capture.
reduction in mortality. There is no clear consensus as to which of these effects should be measured in defining nonresponders. As a practical matter, baseline and follow-up 6-min walk tests or oxygen consumption treadmill tests are useful objective measures of exercise tolerance. Patients who fail to improve in these measures or who face clinical deterioration are considered nonresponders. In nonresponders, it is important to ensure that cardiac resynchronization is effectively delivered, and that it is optimized. A systematic approach to nonresponders is summarized in Table 13.5.

In order for cardiac resynchronization to occur, a sufficient “dose” of therapy must be delivered. A practical goal is to resynchronize at least 90% of R-R intervals. An R-R interval is resynchronized when a left ventricular (or biventricular) pacing pulse is delivered and it captures the left ventricle. The pacing frequency is reported at device interrogation (Fig. 13.28). Factors that prevent sufficient ventricular pacing include atrial undersensing (which results in loss of ventricular tracking and permits intrinsic ventricular activation), atrial oversensing (leading to inappropriate mode switch and loss of atrial synchronous ventricular pacing), ventricular oversensing (which inhibits ventricular pacing output), and conducted intrinsic rhythms (most commonly atrial fibrillation or frequent ventricular ectopy). Failure of left ventricular capture may be difficult to detect, and may reflect a suboptimal pacing vector or insufficient output. Solutions to insufficient pacing frequency and noncapture are summarized in Table 13.5 and discussed in detail in Chapter 10, Troubleshooting.

Some patients may receive a sufficient dose of captured resynchronized R-R intervals and yet fail to respond to therapy. Causes include nonoptimal programming (lack of atrioventricular synchrony or suboptimal V-V timing intervals), excessive atrial pacing (as opposed to atrial-synchronous pacing, the former may lead to left-sided atrioventricular dyssynchrony), nonoptimal lead position, or absence of dyssynchrony. Approaches to these nonresponders are also summarized in Table 13.5 and discussed in detail in Chapter 10.

**Defibrillation efficacy assessment**

In contrast to pacing thresholds, which are easily assessed in the office environment, defibrillation testing requires intensive monitoring due to the necessity or likelihood of arrhythmia induction and shock delivery. The well-documented rise in the defibrillation threshold (DFT) over time in monophasic systems has been nearly eliminated with the currently used biphasic active can systems. However, although the population DFT remains stable with biphasic systems, individual patients may have a critical increase in the DFT requiring system revision. Risk factors exist to predict which individual may have unreliable defibrillation due to a rise in threshold or system malfunction, summarized in Table 13.6. Risk factors for an increase defibrillation threshold at follow-up include an implant threshold > 14 J, and lack of an “active can” system in which the pulse generator serves as a defibrillating electrode (these “cold can” systems are rarely used today). Predictors of lead malfunction are discussed above and included in Table 13.6. In biphasic, active can, pectoral systems that incorporate low-voltage test shocks to confirm ongoing lead integrity, implant DFTs are typically < 10 J, and the risk of failed defibrillation is low. In these systems, routine inductions are performed only at the time of pulse generator change out. Specific clinical situations do arise in which a defibrillator should be evaluated and inductions considered. These are summarized in Table 13.7.

**Device–device interactions**

Because current-generation ICDs are capable of fully functional antibradyarrhythmia therapy, implantation of separate pacemaker and defibrillator pulse generators has become exceedingly rare. However, patients with separate systems require continuing follow-up, and occasionally patients may receive two devices in the setting of novel heart failure pacing devices (cardiac contraction modulation pacing).

Numerous interactions between pacemakers and ICDs can occur, and testing protocols have been developed to detect these interactions. Since up to 50% of patients may have significant pacemaker–ICD interactions that can be avoided if properly identified, testing is warranted. Among the more common interactions described are inhibition of ventricular fibrillation detection by the pacemaker pacing artifacts, inappropriate detection of tachycardia by the defibrillator because of overdetection of pacemaker stimulus artifacts, and various postshock phenomena. Most of these interactions can be avoided if meticulous testing is done at implantation and follow-up. Our recommended testing is summarized in Table 13.8.46

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**CHAPTER 13 Follow-up**

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599
Table 13.5 Approach to nonresponders to cardiac resynchronization

<table>
<thead>
<tr>
<th>Problem</th>
<th>Mechanism</th>
<th>Solution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resynchronization is not delivered (pacing &lt; 90%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sensing errors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Atrial undersensing</td>
<td>Loss of p synchronous pacing and conduction of intrinsic ventricular</td>
<td>Increase atrial sensitivity or reposition atrial lead. If functional,</td>
</tr>
<tr>
<td></td>
<td>complexes</td>
<td>shorten PVARP, increase upper tracking limit, turn off PMT algorithm</td>
</tr>
<tr>
<td>• Atrial oversensing</td>
<td>Far-field R waves cause inappropriate mode switch and loss of atrial</td>
<td>Reduce atrial sensitivity; increase PVAB; reposition lead</td>
</tr>
<tr>
<td></td>
<td>tracking</td>
<td></td>
</tr>
<tr>
<td>• Ventricular oversensing</td>
<td>Ventricular pacing is inhibited</td>
<td>Reduce ventricular sensitivity; adjust ventricular refractory periods</td>
</tr>
<tr>
<td>• Algorithmic inhibition of ventricular pacing</td>
<td>Algorithms such as rate smoothing prevent tracking abrupt increases in</td>
<td>Turn algorithms off. Shorten left ventricular protection period</td>
</tr>
<tr>
<td></td>
<td>atrial rate</td>
<td>(limits upper LV pacing rate)</td>
</tr>
<tr>
<td>Resynchronization is not delivered (pacing &gt; 90%, but no LV capture)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV capture with slow exit from LV pacing site</td>
<td>Due to slow conduction of tissue around LV lead, most of ventricle from</td>
<td>Confirm capture by pacing LV lead alone; positive QRS complex in lead</td>
</tr>
<tr>
<td></td>
<td>RV</td>
<td>III, negative in I, and RBBB morphology in V1 indicate LV capture;</td>
</tr>
<tr>
<td></td>
<td></td>
<td>if capture present, adjust V-V timing to pre-excite LV</td>
</tr>
<tr>
<td>Nonoptimal vector</td>
<td>Capture may be present from some LV vectors and not others; anodal capture</td>
<td>Recheck threshold using alternate vectors; anodal stimulation discussed</td>
</tr>
<tr>
<td></td>
<td>may be present</td>
<td>in Chapter 10, Troubleshooting</td>
</tr>
<tr>
<td>Insufficient output</td>
<td>LV threshold may be elevated</td>
<td>Increase output, or reposition LV lead</td>
</tr>
<tr>
<td>Resynchronization is delivered &gt; 90% with capture</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Confirm patient is nonresponder</td>
<td>Multiple end points have been used to define nonresponders, details in</td>
<td>Six minute walk or oxygen consumption treadmill; assess ejection fraction;</td>
</tr>
<tr>
<td></td>
<td>text</td>
<td>formal QOL assessment</td>
</tr>
<tr>
<td>Nonoptimal programming</td>
<td>Program parameters may not optimize intraventricular, interventricular</td>
<td>Optimize V-V and AV timing with echo guidance (see Chapter 2,</td>
</tr>
<tr>
<td></td>
<td>or atrioventricular mechanical function</td>
<td>Hemodynamics of Device Therapy) If frequent atrial pacing (as opposed</td>
</tr>
<tr>
<td></td>
<td></td>
<td>to sensing) is disrupting left AV mechanical synchronicity use VDD mode</td>
</tr>
<tr>
<td>Nonoptimal lead position</td>
<td>Insufficient RV–LV separation to allow resynchronization</td>
<td>Reposition lead, particularly if in anterior vein, or small RV–LV</td>
</tr>
<tr>
<td></td>
<td></td>
<td>separation radiographically and small V-V interval during intrinsic</td>
</tr>
<tr>
<td>Absence of dyssynchrony</td>
<td></td>
<td>rhythm</td>
</tr>
<tr>
<td></td>
<td></td>
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</tbody>
</table>
Follow-up

Specific situations suggesting further evaluation or arrhythmia induction

As noted above, a number of clinical situations may suggest the need for further ICD testing (Table 13.7). The diagnosis and correction of frequent ICD therapies (symptomatic or asymptomatic) are discussed in Chapter 10, "Troubleshooting". Other situations are discussed below.

Medications

The interactions between medications and defibrillators require special mention in follow-up, because medications may be modified by other physicians without full appreciation of the interaction with ICDs. In general, the medications demonstrated to promote longevity in cardiovascular patients—β-blockers, ACE inhibitors or angiotensin receptor antagonists, anti-hyperlipidemic agents, and aspirin—have no significant interaction with defibrillators and should be used as indicated. However, membrane-active antiarrhythmic drugs can affect pacing function, defibrillation function, and the rate and regularity of intrinsic arrhythmias. The effects require special mention in follow-up, because medications may be modified by other physicians without full appreciation of the interaction with ICDs. In general, the medications demonstrated to promote longevity in cardiovascular patients—β-blockers, ACE inhibitors or angiotensin receptor antagonists, anti-hyperlipidemic agents, and aspirin—have no significant interaction with defibrillators and should be used as indicated. However, membrane-active antiarrhythmic drugs can affect pacing function, defibrillation function, and the rate and regularity of intrinsic arrhythmias. The effects
of medications on defibrillator function are summarized in Table 13.9.

Use of antiarrhythmic drugs can affect pacing thresholds, so that thresholds should be rechecked when membrane-active drugs, particularly class IC agents, are prescribed. Class IC agents have use dependency, so that their effects are amplified at higher heart rates; thus, antitachycardia pacing (ATP) may be more affected than standard antibradycardia pacing. Most defibrillators permit independent programming of ATP outputs, which should generally be set for values greater than those of the standard pacing therapies to ensure capture. A fuller discussion of the effects of medication on pacing function is found in Chapter 1.

Table 13.9 Interactions of antiarrhythmic medications (membrane active drugs) and implantable cardioverter-defibrillators (ICDs)

<table>
<thead>
<tr>
<th>ICD function</th>
<th>Potential medication effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensing</td>
<td>Diminished slew rate could affect detection (rare)</td>
</tr>
<tr>
<td>Detection</td>
<td>Ventricular tachycardia rate slowed below detection cut-off rate</td>
</tr>
<tr>
<td></td>
<td>QRS widening can affect morphology detection enhancement criteria</td>
</tr>
<tr>
<td></td>
<td>Variability in R-R intervals during VT may lead to stability detection enhancement error</td>
</tr>
<tr>
<td>Pacing</td>
<td>Increase pacing threshold</td>
</tr>
<tr>
<td></td>
<td>Increase threshold at rapid pacing rates, affecting antitachycardia pacing (use dependency, particularly class IC agents)</td>
</tr>
<tr>
<td></td>
<td>Induce bradycardia or atrioventricular block necessitating antibradycardia pacing</td>
</tr>
<tr>
<td>Defibrillation</td>
<td>Proarrhythmia with increased shock frequency</td>
</tr>
<tr>
<td></td>
<td>Increase or decrease defibrillation threshold</td>
</tr>
</tbody>
</table>
The most important effect of antiarrhythmic drugs in patients with ICDs is the slowing of ventricular tachyarrhythmias. Ventricular tachycardia may slow below the cut-off rate and remain undetected; a useful rule of thumb is to increase the detection cycle length when initiating these medications by 30–50 msec. Drugs may also increase or decrease the defibrillation threshold, adversely affecting fibrillation termination, although clinically important elevation of defibrillation thresholds is uncommon with current biphasic systems. Due to these effects, initiation of antiarrhythmic drug therapy is followed by ICD parameter assessment, with reprogramming and testing commonly performed in our practice. The effects of medication on defibrillation function are discussed in greater detail in Chapter 1.

Strategies to minimize shocks

A number of programming strategies exist to reduce the likelihood of defibrillator shock, thus to improve the tolerance of device therapy. These include use of sufficiently long detection times in patients with non-sustained arrhythmias to minimize the risk that these trigger detection or therapy; liberal use of ATP to terminate arrhythmias; programming sensing functions to prevent T-wave oversensing on the ventricular lead and far-field R-wave oversensing on the atrial lead; and appropriate use of detection enhancement to minimize the risk of inappropriate detections. Chapter 8, Programming, reviews these features in detail. Screening patients for use of these strategies at follow-up minimizes the risk of shock.

Remote access monitoring

Remote monitoring of implantable devices is a concept that has been present for many years. However, recent advances permitting the acquisition of more sophisticated device and patient data have reinitiated the field. Remote monitoring in its current and rapidly developing iterations may improve patient safety by detecting device malfunction or physiological changes heralding clinical deterioration (such as incipient heart failure) before they become manifest, permitting intervention. Internet-based remote monitoring saves patients clinic visits, and increases healthcare efficiency by requiring less provider care for routine follow-up, and focusing attention on potential problems.

Remote monitoring sessions acquire nearly all of the information obtained from the device in a clinic visit, including real-time electrograms, programmed parameters, stored episode data, detailed therapy histories including electrograms, as well as plots of device function (impedance values, alerts) and patient physiological data (Fig. 13.29). Some systems such as Boston Scientific’s Latitude™ also permit tracking of “external” data such as blood pressure and weight by means of wireless communication with blood pressure cuffs and scales, and patient symptoms by means of patient-completed surveys (Fig. 13.30).

Remote monitoring may be completely automated or, in the case of inductive systems, may require patient intervention. Automated systems utilize radiofrequency transmitters integrated into the implanted device to transmit to a hub, which then communicates to secure data servers via a cellular or land line connection. The hub may be a transceiver kept on the nightstand or worn on a belt, which can receive implanted device data independent of patient action. In inductive systems, the patient holds a wand over his or her device to active a telemetry session, which is then transmitted via a phone connection to a secure internet site. Irrespective of the mechanism used, data are transmitted to a protected Web site that can be accessed by healthcare providers, and other sites (with limited data) available to the patients themselves.

As previously noted, guidelines for follow-up using remote access monitoring are currently being developed by professional societies. At this time the Mayo Clinic follow-up schedule for single- and dual-chamber ICDs in patients with fully automated systems is as follows:

- Nightly: lead and battery check with automatic transmission with radiofrequency devices
- 3rd month: clinic visit
- 3rd month through failure: every 3 months remote access monitoring with yearly in-clinic visits.

More frequent follow-up may be required early after implantation and as the device approaches elective replacement indicators. Other factors accounting for increased frequency of follow-up include patient-initiated transmissions secondary to symptoms, system alerts, or therapy delivery. Some systems incorporate two levels of alerts—low urgency (or yellow) for less time-critical warnings such as elective battery replacement indicators and atrial lead malfunction, and high-urgency (red) alerts for ventricular lead malfunction and other critical warnings. For the clinician, adjustable alert preferences include notification frequency and mode (fax,
The Latitude™ and CareLink™ systems allow patients to log in to a patient-specific Web site to see their alerts and battery status. Un-scheduled patient transmissions are available through all manufacturers, but with varying degrees of physician control. The HouseCall Plus™ (St Jude Medical) system requires the call center or physician to be available to receive the data. Table 13.10 summarizes remote monitor-

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**Fig. 13.29** Remote-access internet-based screen summarizing arrhythmia episodes, from the Medtronic CareLink™ system. Note the plots summarizing atrial and ventricular arrhythmia episode frequency, and patient activity. Also reported is the frequency of ventricular pacing. AS-VS, atrial sense, ventricular sense; AS-VP, atrial sense, ventricular pace; AP-VS, atrial pace, ventricular sense; AP-VP, atrial pace, ventricular pace.

**Fig. 13.30** Implantable cardioverter-defibrillator Internet-based remote monitoring: patient-completed questionnaire to record cardiovascular symptoms. This screen is from the Boston Scientific/Guidant Latitude™ system.

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phone, page, and/or Web-based). The Latitude™ and CareLink™ systems allow patients to log in to a patient-specific Web site to see their alerts and battery status. Un-scheduled patient transmissions are available through all manufacturers, but with varying degrees of physician control. The HouseCall Plus™ (St Jude Medical) system requires the call center or physician to be available to receive the data. Table 13.10 summarizes remote monitor-
Because remote programming is not available at this time, a clinic visit is required for parameter changes. As experience grows with remote monitoring, remote programming will likely become available.

Heart failure trials using non-ICD-based Internet home telemonitoring systems that incorporate a scale and blood pressure cuff have demonstrated survival benefit using these systems compared with usual care, and shortened hospitalization compared with nurse telephone support. The 2005 ACC/AHA heart failure guidelines recommend weight, blood pressure, activity level and clinical symptom and clinical sign assessments be recorded in all patients with CHF. At the time of writing, the Boston Scientific Latitude™ system permits recording of these parameters, and it appears other systems will soon incorporate them. In addition to intermittent weight and blood pressure, which are recorded via sensors that wirelessly transmit information to an ICD’s remote monitoring hub, implantable devices analyze continuous, physiological parameters. The role of various novel physiological parameters in predicting heart failure is the subject of active investigation. One trial has shown that activity (recorded by the accelerometer present in all ICDs) drops and weight increases prior to hospitalization. Decreased heart rate variability (a measure of the variability of R-R intervals associated with changes in autonomic tone) precedes hospitalization by 16 days, and automated detection had a 70% sensitivity for detection of cardiovascular hospitalizations, with 2.4 false positives per patient-year follow-up. The CareLink™ system provides intrathoracic impedance-based estimates of pulmonary congestion in defibrillators with the OptiVol® system (Fig. 13.31). In a small trial, a drop in impedance (suggesting incipient pulmonary edema) was associated with early detection of clinical symptoms and predicted hospitalization by 15.3 ± 10.6 days with a sensitivity of 76.9%. The role of remote monitoring in care is rapidly evolving.

### Equipment

To perform remote access monitoring, the patient must have access to the necessary equipment. Typical transmitting equipment is shown in Fig. 13.32. For wireless telemetry, the patient’s radiofrequency monitor can typically be within 10 ft of the implanted device for transmissions to occur. Inductive telemetry requires a wand to be placed over the device within.

<table>
<thead>
<tr>
<th>Name</th>
<th>Biotronik</th>
<th>Boston Scientific</th>
<th>Medtronic</th>
<th>St Jude Medical</th>
</tr>
</thead>
<tbody>
<tr>
<td>Remote monitoring</td>
<td>Home Monitoring™</td>
<td>Latitude™</td>
<td>CareLink™</td>
<td>Housecall Plus™</td>
</tr>
<tr>
<td>connection</td>
<td>Standard analog</td>
<td>Standard analog</td>
<td>Standard analog</td>
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<td>Access</td>
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<td>Secure Internet</td>
<td>Secure Internet</td>
<td>Secure Internet</td>
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<td>Clinicians</td>
<td>Clinicians</td>
<td>Patient access</td>
<td>Clinicians</td>
<td>Service provider</td>
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<td>Patient access</td>
<td></td>
<td></td>
<td>Patient access</td>
<td></td>
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<tr>
<td>Data available</td>
<td>Complete device</td>
<td>Complete device</td>
<td>Complete device</td>
<td>Complete device</td>
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<tr>
<td>EGM</td>
<td>EGM</td>
<td>BP and weight</td>
<td>EGM</td>
<td>EGM</td>
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<td></td>
<td></td>
<td></td>
<td>Hemodynamic—</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>OptiVol®</td>
<td></td>
</tr>
<tr>
<td>Transmission:</td>
<td>Daily and event</td>
<td>Fixed schedule</td>
<td>Fixed schedule</td>
<td>Fixed Concordant</td>
</tr>
<tr>
<td>Schedule</td>
<td>MD schedules</td>
<td>PRN MD schedules</td>
<td>PRN MD schedules</td>
<td>Concordant</td>
</tr>
<tr>
<td>Time to transmit</td>
<td>1–3 min</td>
<td>1–3 min</td>
<td>2–3 min</td>
<td>15 min</td>
</tr>
<tr>
<td>Cost</td>
<td>At implant</td>
<td>At implant</td>
<td>At implant</td>
<td>Clinic or 3rd party</td>
</tr>
<tr>
<td>Additional features</td>
<td>Wireless monitoring</td>
<td>Wireless implant</td>
<td>Wireless implant</td>
<td>Live interface with medical professionals</td>
</tr>
</tbody>
</table>
Electromagnetic interference

Although patients do not use the term “electromagnetic interference” (EMI), they ask whether anything in the environment interferes with pacemakers and defibrillators. The reader is referred to Chapter 12 for a complete discussion. Here, a few specific recurrent patient concerns merit repeating.

Microwave ovens have not been a concern for many years, despite the signs that remain posted near some microwave ovens in public places. Patients should be told that there were concerns with older pulse generators that were not as well shielded and older microwaves that were not as well sealed. Interference is no longer a problem. Although induction cooktops have not become widely used, they may produce clinically

Patient concerns during follow up

It is impossible to predict all concerns that might be raised by the patient with an implantable cardiac device. However, several specific issues are invariably raised, and they should be included in the information, whether written or oral, provided to the patient.

a range of a few inches. Transmissions may require a cellular or landline connection. The information is transmitted to a privacy protected Web site managed by the device company. Caregivers may easily access this information and it is similar to data obtained from in-clinic programmers. Typically, a list of clinic patients is presented on a Web page, with those with alerts highlighted for attention.
significant EMI and patients should be cautioned if they use such an appliance.

Cellular telephones should not be a concern for the patient with an implantable device. As discussed in Chapter 12, the patient should avoid holding the activated (“on”) phone near the pacemaker or ICD. Cellular calling and reception should be avoided in both the theoretical and practical sense during programming and interrogation, as loss of telemetry can occur if the cell phone is near the device or the programming head. Cell phones have not been associated with false arrhythmia, delayed recognition or inappropriate treatment. Ideally, the patient should use the phone at the ear contralateral to the pulse generator, at least 20 cm away from generator and not carry an activated phone in a pocket over the ICD.

Electronic article surveillance equipment has been a controversial issue in recent years. Although pulse generator interference is possible, some simple advice for the patient with an implantable device should suffice. Patients should be aware of the location of surveillance equipment and avoid lingering within or near any antitheft device. The phrase “don’t linger,
“Don’t lean” has been popularized to summarize this advice. Somewhat surprisingly, questions regarding welding equipment are not uncommon. For the non-pacemaker-dependent device patient, the use of most hobby welding equipment should not be a problem. Patients should be questioned about the strength of the welding equipment they use. Previous work has demonstrated that alternating current hobby welding equipment in the range of 100–150 A should not cause a problem. Industrial strength welding, i.e., alternating current of 200–500 A, definitely has the potential for clinically significant EMI. Patients with pacemakers or ICDs who work with or close to such equipment should be individually assessed, especially if they are pacemaker-dependent. With ICDs, oversensing of the EMI could result in a false-positive interpretation that delivers a shock to the patient.

Specific work environments that contain equipment capable of producing EMI require individual assessment. Work environments with degaussing equipment, such as the television industry, induction ovens, and industrial welding, are of particular concern. If a patient’s livelihood depends on working in such an environment, the issues must be considered carefully; the patient should not be glibly told that return to this job is not possible. Testing procedures can be performed to determine whether the work environment is indeed hostile. For the non-pacemaker-dependent patient, the risk posed by EMI is obviously less significant. It may be possible to assess nondependent patients by ambulatory monitoring or patient-triggered event records.

Medical advisories and recalls
Dealing with medical advisories and recalls from the manufacturer or the Food and Drug Administration (FDA) and reporting device failures are responsibilities of the physician or the institution, or both, providing follow-up care. The FDA categorizes recalls into three classes:

- **Class I**—Situations in which there is a reasonable probability that the use of, or exposure to, a violative product will cause serious adverse health consequences or death
- **Class II**—Situations in which the use of, or exposure to, a violative product may cause temporary or medically reversible adverse health consequences or in which the probability of serious adverse health consequences is remote
- **Class III**—Situations in which the use of, or exposure to, a violative product is not likely to cause adverse health consequences.

Safety advisories or safety alerts are sometimes issued and are, in general, less significant than class III recalls.

When informed of a recall or advisory, the physician or institution involved in follow-up of the patient with a pacemaker or ICD is responsible for making certain that the patient is aware of the potential problem and that appropriate steps are taken. Necessary action depends on the type of problem identified. Action may range from pulse generator or lead replacement to lead extraction, intensified follow-up, or patient notification only. Patient notification and advice should be documented in the medical chart. With remote access monitoring, recall information is transmitted to the patient through alerts on their home monitors.

Pacemaker clinic personnel should be available to discuss the alert or advisory with the patient after notification. They should also be knowledgeable about the specific problem and be able to explain the problem in a way the patient can understand.

Under the Safe Medical Devices Act of 1990 (Public Law 101–629) and the Medical Device Amendments of 1992, hospitals, ambulatory surgical facilities, nursing homes, and outpatient treatment facilities that are not physicians’ offices must report to the FDA or the manufacturer any death, serious illness, or serious injury caused or contributed to by a medical device. Such incidents should be reported within 10 working days of the event. Patient deaths must be reported to the FDA, and serious illness and injury must be reported only to the manufacturer. (If the manufacturer is unknown, the report should be made to the FDA.)

Despite their overall high level of reliability, over the years there have been periodic safety alerts and “recalls” of ICDs affecting thousands of patients. The number of pacemakers and ICDs affected has increased dramatically since 1995, probably due to increased awareness, greater enforcement of reporting policy, lay press coverage, and increased device complexity. ICD malfunction rates peaked in 2001 with 36 replacements per 1000 patient-years according to a multiregistry meta-analysis. Advisories lead to patient concern and impaired quality of life, more intensive follow-up, and surgical device replacement. They involve tremendous cost to and burden on the healthcare system, and operative morbidity and mortality for patients.
ment has a higher morbidity than new implantation and for some “recall” events likely exceeds the morbidity caused by the device malfunction itself.

A device malfunction is said to occur when it fails to meet its performance specification, or otherwise fails to perform as intended. Device malfunction may compromise or degrade therapy, or may only impact ancillary features such as diagnostics (see Table 13.11 for definitions of device malfunction). The approach to “recall” management depends on the mechanism of malfunction and its predictability, the impact should malfunction occur, patient factors (such as pacemaker dependency), and the risk of surgical interventions to correct the malfunction (such as device replacement).

Professional societies provide guidance for the management of device alerts and recalls that aim to detect problems early, to minimize patients’ confusion by providing timely and appropriate information, and to avoid unnecessary device replacements.71,72

Suggested methods of increasing patient safety while minimizing inconvenience and anxiety include:

- Increased follow-up frequency for devices that are not replaced
- More widespread use of automatic alerts and home monitoring systems for early detection of malfunctions in patients with devices under alert
- Efficient, quick and responsible communication between manufacturers, physicians and patients so that relevant information will reach patients through their caregivers rather than via mass media. The US FDA has issued a guidance document for “Dear Doctor” letters to facilitate such communications (http://www.fda.gov/cdrh/ocer/guidance/1645.html).

Recommended measures to decrease unnecessary device replacements include:

- Careful individual risk assessment, taking into account not only the probability of intrinsic device failure but also potential consequences of failure related to current device indication in the individual patient. For example, a pacemaker-dependent ICD patient with recurrent life-threatening arrhythmia is at much higher risk in the event of device failure than a patient

<table>
<thead>
<tr>
<th>Device malfunction</th>
<th>Device malfunction with compromised therapy</th>
<th>Induced malfunction</th>
<th>Malfunction without compromised therapy</th>
<th>Normal battery depletion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Failure of a device to meet its performance specifications or otherwise perform as intended. Performance specifications include all claims made in the labeling for the device. The intended performance of a device refers to the intended use for which the device is labeled or marketed (FDA Regulations 803.3(n)). Whenever possible, device malfunction should be confirmed by laboratory analysis</td>
<td>A device (pulse generator or lead) that has malfunctioned in a manner that compromises pacing or defibrillation therapy (including complete loss or partial degradation). Some examples include: sudden loss of battery voltage, accelerated current drain such that low battery voltage is not detected before loss of therapy and sudden malfunction resulting in nondelivery of defibrillation therapy</td>
<td>A malfunction caused by external factors (e.g., therapeutic radiation, excessive physical damage, etc.) including, but not limited to, hazards that are listed in product labeling. Damage to a pulse generator caused by a lead malfunction is considered to be a lead rather than a pulse generator malfunction</td>
<td>A device that has malfunctioned in a manner that does not compromise pacing or defibrillation therapy. Some examples include: error affecting diagnostic functions, telemetry function, data storage; malfunction of a component that causes battery to lose power prematurely, but in a timeframe that is detectable during normal follow-up before normal function is lost, and mechanical problems with connector header that do not affect therapy</td>
<td>1. A device is returned with no associated complaint and the device has reached its elective replacement indicator(s) with implant time that meets or exceeds the nominal (50th percentile) predicted longevity at default (labeled) settings 2. A device is returned and the device has reached its elective replacement indicator(s) with implant time exceeding 75% of the expected longevity using the longevity calculation tool available at the time of production introduction, calculated using the device’s actual settings</td>
</tr>
</tbody>
</table>

From Carlson, Wilkoff et al. 2006.72
implanted for primary prevention of sudden death. The concept of current device indication (as opposed to original indication for implantation) is important, as the clinical condition may have changed since implantation with newly developed arrhythmias or pacemaker dependence.

- The risks of the replacement operation and time to elective replacement of the device must also be considered for the individual patient. Overall, a risk of malfunction below 1/1000 is considered low when considering replacement in a patient who is not at particularly high risk should the device malfunction.

- In some specific situations, consideration of alternative noninvasive measures of management, such as reprogramming, frequent monitoring, or daily magnet application may be applicable.

Given the small risk of device malfunctions in most circumstances, the physician must allay patient anxiety and confusion and objectively balance the risk of operation with continued observation. Table 13.12 summarizes the Heart Rhythm Societies guidelines for decisions on device recalls and notifications.

### Lifestyle and personal concerns

Return to driving has traditionally been less of an issue with pacemakers than with ICDs. Both the Heart Rhythm Society (formerly North American Society of Pacing and Electrophysiology) and the Canadian Cardiovascular Society have established guidelines for return to driving after pacemaker implantation. These are summarized in Table 13.13. Regulations for return to driving differ from state to state.

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**Table 13.12: Guidelines for decisions on device recalls and notifications**

<table>
<thead>
<tr>
<th>Situation</th>
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</thead>
<tbody>
<tr>
<td><strong>Consider device/lead replacement if:</strong></td>
</tr>
<tr>
<td>- the mechanism of malfunction is known and is potentially recurrent</td>
</tr>
<tr>
<td>- the risk of malfunction is likely to lead to patient death or serious harm, and</td>
</tr>
<tr>
<td>- the risk of replacement is less than or at least not substantially greater than the risk of device malfunction</td>
</tr>
<tr>
<td><strong>Consider device/lead replacement in:</strong></td>
</tr>
<tr>
<td>- patients who are pacemaker-dependent</td>
</tr>
<tr>
<td>- patients with an ICD for secondary prevention of sudden death, and</td>
</tr>
<tr>
<td>- patients with an ICD for primary prevention of sudden death who have received appropriate device therapy for a ventricular arrhythmia</td>
</tr>
<tr>
<td><strong>Consider device replacement if the predicted end of life (EOL) is approaching</strong></td>
</tr>
<tr>
<td><strong>Consider conservative management with periodic noninvasive device monitoring when the rate of device malfunction is very low in:</strong></td>
</tr>
<tr>
<td>- patients who are not pacemaker-dependent and</td>
</tr>
<tr>
<td>- patients with an ICD for primary prevention of sudden cardiac death who have not required device therapy for a ventricular arrhythmia</td>
</tr>
<tr>
<td><strong>Provide routine follow-up for patients with a device malfunction that has been mitigated or corrected by reprogramming the software</strong></td>
</tr>
<tr>
<td><strong>Consider conservative management with periodic noninvasive device monitoring in patients where operative intervention risk is high or in patients who have other significant competing morbidities even when the risk of device malfunctions or patient harm is substantial</strong></td>
</tr>
</tbody>
</table>

From Carlson, Wilkoff et al. 2006.
We advise patients not to drive for 2 weeks after pacemaker implantation or revision of the ventricular lead, explaining that it is a medicolegal concern. Although rules vary among various countries and even states within the USA, general principles have been adopted by both American and European expert panels. Patients with ICDs are prohibited from any commercial driving. Patients who have received an ICD for primary prevention may drive within a relatively short period of time. At our institution we ask pacemaker and CRT patients and ICD patients implanted for primary prevention to avoid driving for 10 days. If patients with an ICD subsequently receive an appropriate therapy for ventricular tachycardia or fibrillation, especially with syncope, they are for practical purposes reclassified as having the device for secondary prevention and as such will be restricted from driving for 6 months. Furthermore, patients may have contraindications to driving without any tachycardia because of unstable medical issues, and driving privilege will need to be assessed on an individual basis.

Another relatively minor but driving-related issue is whether a seat belt interferes with the pacemaker. The seat belt may be over the pacemaker site for the driver with a left pectoral implant or a passenger with a rightsided implant. Seat belts are not an issue unless they result in irritation at the implant site in the early weeks after implantation. If irritation is a concern, the patient can place some padding over the pulse generator or around the seat belt in the area of pulse generator contact. This should not be an excuse not to wear a seat belt and would not be considered a justifiable reason for not wearing a seat belt if cited by the authorities.

Limitation of physical activities after device implantation must be addressed. We recommend that ipsilateral arm movement be limited to 90° abduction for 3–4 weeks. Admittedly, this may be overcautious and impossible for some patients, especially pediatric patients. The lead is secured to the pectoral muscle near the venous insertion site, and it is unlikely that more vigorous arm movements would dislodge the lead. However, some guidelines should be given, and this approach has been successful for us, with most adult patients seeming to follow the advice without difficulty. We frequently send patients home with a sling to be worn loosely for 5–7 days as a reminder to limit arm motion. This is especially helpful in the pediatric population.

Although we restrict arm motion on the side of the pulse generator implant, we are also careful to caution the patient not to immobilize the arm. Complete immobility could lead to reflex sympathetic dystrophy. We also recommend that patients limit lifting to no more than 10 lb with the ipsilateral arm for the first 2 weeks after implantation.

Table 13.13 Guidelines for driving after pacemaker implantation

<table>
<thead>
<tr>
<th>Condition</th>
<th>Non-commercial*</th>
<th>Commercial*</th>
</tr>
</thead>
<tbody>
<tr>
<td>No symptoms, no pacemaker</td>
<td>A</td>
<td>A</td>
</tr>
<tr>
<td>Syncope or near-syncope, no pacemaker</td>
<td>C</td>
<td>C</td>
</tr>
<tr>
<td>Pacemaker, not pacemaker-dependent†</td>
<td>A</td>
<td>A</td>
</tr>
<tr>
<td>Pacemaker, pacemaker-dependent†</td>
<td>B, 1 week</td>
<td>B, 4 weeks</td>
</tr>
</tbody>
</table>

A, no driving restrictions; B, driving permitted after controlled arrhythmia is documented for a specified period and an adequate pacemaker follow-up regimen is followed; C, driving completely prohibited.

*Guidelines from the Canadian Cardiovascular Consensus Conference differ only in that all patients for private driving are restricted for 1 week after pacemaker implantation, must not have evidence of cerebral ischemia, and must have a pacemaker that is performing normally with normal sensing and capture. For commercial driving, the same guidelines apply, except that the waiting period to drive is 4 weeks and the pacemaker output pulse must be at least three times the measured stimulation threshold.

†For these purposes, “pacemaker-dependent” is applied to patients who have lost consciousness in the past due to bradycardias. The term may also be applied to patients immediately after atrioventricular junction ablation and to any other patient in whom sudden pacemaker failure is likely to result in alteration of consciousness.

From Epstein et al. By permission of the American Heart Association.
Issues of return to work and disability arise. Once again, the advice should be individualized. Patients who have jobs that do not involve heavy physical exertion can return to work shortly after the procedure. It is unusual for the patient to experience postoperative pain significant enough to require more than ibuprofen or acetaminophen and to limit the ability to perform the job. Patients who have jobs that involve heavy physical exertion in which performance depends on upper body strength, or activities that require that the arm ipsilateral to the new implant be lifted above shoulder level may need to wait longer to return to work. In these circumstances, it is justifiable to keep the patient away from work for up to 4 weeks.

Sports activities are important for many patients. Patients are told that they can return to most sports activities. For the younger pacemaker patient, most competitive sports are reasonable, with the exception of contact sports having a significant potential for injury. Specifically, football, wrestling and boxing carry some risk. Patients should be informed of the risk and be given counseling to weigh the ratio of risk to benefit. The predominant concern with contact sports is direct trauma to the lead at or near the connector block. If the lead(s) has been implanted via the subclavian vein, it may be somewhat more susceptible to injury from any repetitive motions that has the potential of narrowing the interspace between the first rib and clavicle, e.g., weightlifting and basketball. Again, the issues should be discussed with the patient and the importance of the activity weighed against the risk.

Golf and swimming are two relatively common athletic activities for the average patient with a pacemaker. We suggest waiting 4 weeks after implantation before returning to golfing. Swimming can be resumed as soon as the incision is healed, but the recommendation is to limit some strokes for 4 weeks to stay within the abduction guidelines already discussed.

Hunting and marksmanship also seem to be relatively frequent activities for our patient population. The pacemaker should be implanted on the side contralateral to that from which the patient shoots a rifle. We allow patients to return to these activities at any time so long as they stay within the shoulder movement guidelines outlined.

Activity restrictions for ICD or CRT-D recipients are, for the most part, the same as those for pacemaker or CRT-P patients. Patients with slow ventricular tachycardia and young or active patients should undergo exercise testing to determine their peak heart rate with activity so that inappropriate detection of physiological tachycardia as an arrhythmia can be avoided. ICD recipients are currently ineligible for competitive sports; however, noncompetitive athletics and physical activity are generally encouraged. Swimming is avoided, particularly in long QT patients, due to the unique drowning risk associated with an event in the water. Detailed guidelines regarding sporting activities for ICD recipients have been published.

Concerns about resumption of sexual activity are frequent. Patients are often reluctant to ask questions about resuming sexual activity, and ideally the information should be offered. Patients are told that they may resume sexual activity whenever they like, so long as they observe the shoulder motion guidelines.

Similar to the seat belt issue previously discussed, some women are concerned about irritation to the device site by their bra strap. Our patients are advised either not to wear a bra until the incision is well healed or not to wear a bra at all until the incision is well healed and nontender, or to place extra padding around the strap. Rarely is this a significant problem or concern.

Many unanticipated concerns arise. Initial education about the pacemaker or defibrillator and how it works is the best way to facilitate the patient’s return to health.

Conclusion

Appropriate follow-up of an implantable device is required to ensure continuing integrity of the system and to detect failures before they become clinically manifest. This requires a thorough understanding of both device function and interpretation of the extensive telemetered data provided. However, with the incorporation of routine device self-assessment, with the ability of many pulse generators to alert patients effectively to potential problems, and with the advent or remote monitoring, device surveillance has changed dramatically in recent years. Furthermore, with continued maturation of device technology and monitoring, the focus of follow-up is evolving from detecting device malfunction to predicting disease progression before it becomes manifest in order intervene to maintain health.

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CHAPTER 13 Follow-up

52 Bonow RO, Bennett S, Casey DE Jr et al. ACC/AHA Clinical Performance Measures for Adults with Chronic Heart Failure: a report of the American College of Cardiology/


76 Okada M, Suzuki K, Hidaka T et al. Complex regional


Index

Page numbers in italics indicate figures. Page numbers followed by $t$ indicate tables.

AAI, see atrial inhibited pacing

AAT, see single-chamber triggered-mode pacing

abandoned, nonfunctioning, noninfected leads, 221–2, 224–5

abnormal physiology, 43–5, 45

accelerometer, 382–4, 383

activity sensors, 382–4

acupuncture, electromagnetic interference, 566

acute myoccardial infarction, 88–9, 89t

algorithms
to manage atrial fibrillation, 374, 375
to manage PVCs, 373–4, 374
to promote continuous tracking, 373, 373
alignment errors, 478–81, 479, 480–1
alteration in programmed pacing rate, 442–3

amiodarone, 36, 87

amyloidosis, 87

analysis of dual-chamber electrograms, 472–3

analysis of single-chamber electrograms, 469–72

anesthesia, 144

angiographic wires vs. deflectable electrophysiological catheters to engage coronary sinus, 164–8, 165–7

ankylosing spondylitis, 87

anodal stimulation, 288–97

antero-interventricular vein, 281–2, 282–3

anti-tachycardia pacing, 19–22, 21, 367–9, 368

AOO, see atrial asynchronous pacing

approach to biventricular paced electrocardiogram, 276–97

arrhythmia induction, 601–6, 601t

arrhythmias, 210, 210

arrhythmogenic right ventricular dysplasia, 110, 110–12, 111, 111t

arrhythmogenesis, 71

ARVD, see arrhythmogenic right ventricular dysplasia

ascending ramp waveform, 31

assessing lead function, 593–5, 595–6, 597–8

assessing pacing rate, 410, 410–12, 411–13

assessing ventricular synchrony with ECG, 279, 285–6, 286–8

assessment of patient clinical status, 590, 591–2

assessment of stored data, 582–4, 585–7

asynchronous pacing, 551–5, 554–5

atenolol, 36

atrial, ventricular-based timing compared, 247–51, 250, 251t

atrial asynchronous pacing, 235, 235–40, 236

atrial defibrillators
detection, 369, 369–70, 370
therapies, 369, 369–70, 370

atrial fibrillation, 100

atrial inhibited pacing, 236–7, 237–8

atrial sensing errors, 482–3, 484–5

atrial synchronous (P-tracking/P-synchronous) pacing, 241, 241

atrioventricular block, 83–8, 84–6, 87t, 87t

atrioventricular dissociation, ventriculoatrial conduction, 45–7, 46–7, 47t, 48

atrioventricular interval, 243–4, 246, 273

atrioventricular interval hysteresis, 247, 248, 249t

atrioventricular sequential non-P-synchronous, rate-modulated pacing with dual-chamber sensing, 240–3

non-P-synchronous pacing with dual-chamber sensing, 240, 240

ventricular inhibited pacing, 239–40
<table>
<thead>
<tr>
<th>Index</th>
</tr>
</thead>
<tbody>
<tr>
<td>atrioventricular sequential asynchronous pacing, 235,</td>
</tr>
<tr>
<td>235–40, 236</td>
</tr>
<tr>
<td>atrioventricular synchrony, 47–50, 48t, 49–50</td>
</tr>
<tr>
<td>atrioventricular synchrony on electrocardiogram, 287–8</td>
</tr>
<tr>
<td>atropine, 409</td>
</tr>
<tr>
<td>AV block, see atrioventricular block</td>
</tr>
<tr>
<td>AV dissociation, 366</td>
</tr>
<tr>
<td>AV node ablation, 87</td>
</tr>
<tr>
<td>AV optimization, 50–1</td>
</tr>
<tr>
<td>avoiding adverse effects from lead, frequent, unnecessary pacing, 137</td>
</tr>
<tr>
<td>avoiding atrial pace/sense competition, 254–5, 256</td>
</tr>
<tr>
<td>axillary (extrathoracic subclavian) approach, 147, 147–50, 148–51</td>
</tr>
<tr>
<td>battery depletion, 227, 227, 407, 455, 502</td>
</tr>
<tr>
<td>beta-blockers, 87</td>
</tr>
<tr>
<td>benefits of left ventricular, biventricular pacing, mechanisms underlying, 64–6, 65</td>
</tr>
<tr>
<td>biatrual pacing, 286–8</td>
</tr>
<tr>
<td>Biotronik device, 139, 249, 251, 402, 521, 557–8, 583, 605</td>
</tr>
<tr>
<td>biphasic waveforms, 29, 29–30</td>
</tr>
<tr>
<td>bipolar vs. unipolar pacing, sensing, 11, 11–12</td>
</tr>
<tr>
<td>biventricular devices, electrocardiographic interpretation, 276–84</td>
</tr>
<tr>
<td>biventricular paced electrocardiogram, 276–97</td>
</tr>
<tr>
<td>biventricular pacing consistent, 261–3, 262–4</td>
</tr>
<tr>
<td>ECG characteristics, 281, 284</td>
</tr>
<tr>
<td>blanking periods, 323–30</td>
</tr>
<tr>
<td>bretylium, 409</td>
</tr>
<tr>
<td>Brugada syndrome, sudden unexplained death syndrome, 108–9</td>
</tr>
<tr>
<td>calcific valvular disease, 87</td>
</tr>
<tr>
<td>calcium-blocking agents, 87</td>
</tr>
<tr>
<td>cannulating coronary sinus, 169–71, 171</td>
</tr>
<tr>
<td>capacitor status, 593–4, 593–5, 595</td>
</tr>
<tr>
<td>cardiac conduction system, 1, 1–2</td>
</tr>
<tr>
<td>anatomy, physiology, 1, 1–2</td>
</tr>
<tr>
<td>cardiac resynchronization devices, 372–4</td>
</tr>
<tr>
<td>cardiac resynchronization therapy, 137–8</td>
</tr>
<tr>
<td>cardiovascular physiology, 43–5, 44</td>
</tr>
<tr>
<td>cardioversion, electromagnetic interference, 566</td>
</tr>
<tr>
<td>cardioversion-defibrillation, 556</td>
</tr>
<tr>
<td>catheter ablation, 559–60</td>
</tr>
<tr>
<td>cautery, electromagnetic interference, 566</td>
</tr>
<tr>
<td>cellular phone, electromagnetic interference, 566</td>
</tr>
<tr>
<td>cephalic approach, 151, 152</td>
</tr>
<tr>
<td>Chagas disease, 87</td>
</tr>
<tr>
<td>channelopathies, 109–10</td>
</tr>
<tr>
<td>chest leads, 278</td>
</tr>
<tr>
<td>choosing lead or leads, 124–32, 125–6</td>
</tr>
<tr>
<td>choosing rate-adaptive sensor, 124</td>
</tr>
<tr>
<td>choosing specific programmable options, 124</td>
</tr>
<tr>
<td>chronic bifascicular, trifascicular block, 89–90, 90t</td>
</tr>
<tr>
<td>chronotropic response, 45, 45–6</td>
</tr>
<tr>
<td>class 1C agents, 87</td>
</tr>
<tr>
<td>clinical approaches to V–V optimization, 70, 70–1</td>
</tr>
<tr>
<td>clinical assessment, 402, 402t–403t</td>
</tr>
<tr>
<td>clinical troubleshooting, 406–12</td>
</tr>
<tr>
<td>collagen-vascular diseases, 87</td>
</tr>
<tr>
<td>committed, noncommitted shocks, 350, 351</td>
</tr>
<tr>
<td>comparison of atrial, ventricular rates, 357–9, 361</td>
</tr>
<tr>
<td>compatibility of lead, pulse generator, 128, 129</td>
</tr>
<tr>
<td>complications associated with coronary sinus cannulation, 172, 172–3</td>
</tr>
<tr>
<td>complications of lead extraction, 196</td>
</tr>
<tr>
<td>complications related directly to implant procedure, 202–27</td>
</tr>
<tr>
<td>component failure, 502</td>
</tr>
<tr>
<td>congenital heart disease, 112–13</td>
</tr>
<tr>
<td>congestive heart failure, 96–100, 97–8, 99t</td>
</tr>
<tr>
<td>contraindications to implantable cardioverter-defibrillator</td>
</tr>
<tr>
<td>therapy, 113, 113t, 114</td>
</tr>
<tr>
<td>coronary artery disease, 87, 102–4, 105</td>
</tr>
<tr>
<td>coronary sinus cannulation, 164–68</td>
</tr>
<tr>
<td>lead placement, 161–75, 163–64</td>
</tr>
<tr>
<td>musculature, 287, 289–90, 291–3, 294–6</td>
</tr>
<tr>
<td>venography, 168, 168–70, 169–70</td>
</tr>
<tr>
<td>coronary venous leads, 540–2, 543–5</td>
</tr>
<tr>
<td>corticosteroids, 409</td>
</tr>
<tr>
<td>creating defibrillation waveform, 28–9, 29</td>
</tr>
<tr>
<td>critical mass, 16–17</td>
</tr>
<tr>
<td>CRT, see cardiac resynchronization therapy</td>
</tr>
<tr>
<td>crumpling of epicardial patch, 502</td>
</tr>
<tr>
<td>data storage, 590</td>
</tr>
<tr>
<td>DDD, see dual-chamber pacing, sensing with inhibition and tracking</td>
</tr>
<tr>
<td>DDI, see atrioventricular sequential, non-P-synchronous pacing with dual-chamber sensing</td>
</tr>
<tr>
<td>DDIR, see atrioventricular sequential, non-P-synchronous, rate-modulated pacing with dual-chamber sensing</td>
</tr>
<tr>
<td>defibrillation, 367–9, 559, 559–60, 560t</td>
</tr>
<tr>
<td>efficacy assessment, 599, 601t</td>
</tr>
</tbody>
</table>
Index

619

far-field R wave, 366, 366
First phase as “conditioning” pulse, 31
flecainide, 409
focused troubleshooting, 415–45
follow-up, 572–616
implantable cardioverter-defibrillator follow-up, 590–6
pacemaker clinical follow-up visit, 581–90, 584
pacemaker follow-up, 576–81
patient-specific programming, therapy, 596–612
requirements, device follow-up clinic, 573–6
follow-up of patients with CRT devices, 596–9, 598, 600

general considerations, 172–3, 173–4
generator, lead selection in defibrillators, 132–5
Guidant, 521
guiding sheath, 164
hardware adaptations, 184, 184–6, 185, 185r, 186
heating pad, electromagnetic interference, 566
hemochromatosis, 87
hemodynamic benefits of pacing in first-degree atrioventricular block, 65, 75
hemodynamic benefits of pacing in neurocardiogenic syndromes, 73–5, 74
hemodynamic pacing, overview of, 45–55
hemodynamics, 43–81
cardiovascular physiology, 43–5, 44
less common indications for pacing for hemodynamic improvement, 72–5
overview, 45–55
pacing in congestive heart failure, 62–6
ventricular timing optimization (V–V optimization), 66–72
hemodynamics of device therapy, 43–81
home induction ovens, 565
homegoing instructions, 193–4
hospital environment, 556–63, 556r–558r
hospital stay after implantation, 190–1
hypertrophic cardiomyopathy, 96, 112, 112r
ibutilide, 36
ICD, see implantable cardioverter-defibrillator
identifying pulse generator, 402–3, 403r
idiopathic (senescent) AV block, 87
iliac vein approach, 153, 154
impact of diseased myocardium proximate to pacing electrodes, 66
impedance, 446t, 447, 457–63
impedance plethysmography, 562–3, 564
implant, hardware-related complications, preimplantation symptom recurrence, 227–32
implantable cardioverter-defibrillator detection, 344–56
implantable cardioverter-defibrillator follow-up, 590–6
implantable cardioverter-defibrillator sensing, 340–3, 341–5
implantable defibrillator troubleshooting, 445–68
implantation facility, 144, 145
implantation-related complications, 202–33
recurrence of preimplantation symptoms, 227–32
related directly to implant procedure, 202–27
importance of waveform, 27–32, 28
inaccurate template, 478
inactivated implantable cardioverter-defibrillator, 493–4
inadvertent left ventricular lead placement, 214, 215–16
indications for implantable cardioverter-defibrillator, 101–13
for lead extraction, 194–5
for permanent pacing, 82–101
for rate-adaptive pacing, 380–91
industrial environment, 563–65, 564, 565t
ineffective delivery therapy, 501–2
ineffective therapy, 474t, 492–502
infection, 194t, 218–21, 221–3, 224t
infective endocarditis, 87
inferior leads, 277
infiltrative diseases, 87
influence of pacing site, 64
initial electrocardiographic interpretation, 266–8, 267–8
insulation, 125–8, 127
integrated vs. bipolar sensing, 132–4, 133–5
interaction of detection zones, detection enhancements, 475–7
interactions between devices, 490–1, 599–601, 602t
interactions of antibradycardia pacing with tachycardia programming, 369
interrogation, 301, 302
interval stability, 471–2
intracardiac position, 526–8, 527–9
intracardiac signals, 470–71, 483–90
intra-device interactions, 499–501, 500
intravenous, 36
isoproterenol, 36, 409
jugular approach, 151–3, 153
lateral, posterolateral cardiac vein, 282–3
leads, 278, 520–42, 523, 538–42, 523, 538–42
damage, 218, 220
design, 8, 8–12, 9–11
diameter, 128, 128
dislodgment, 202–5, 203–4, 205
extraction, 194–9
failure, 447, 491–2, 492–3
fracture, insulation defect, 229, 230, 230–1
t
integrity, 403–6, 404, 404–5, 405–6
perforation, 207–9, 208–9
placement, 202–10
polarity, 128
stability, 174, 174–5
system, defibrillation, 32–5, 33–5
left, lateral leads, 276–7, 277
left atrial pacing, 71
left ventricular, biventricular pacing, 63–4, 64
left ventricular lead, 12
deployment, 172
left ventricular pacing ECG patterns, recognition, 279–81, 281
less common indications for pacing for hemodynamic improvement, 72–5
lifestyle, 610–12, 611t
limb-girdle dystrophy, see Erb’s dystrophy
lithotrity, 556, 566
long QT syndrome, 106–8, 107, 108t, 109, 109t
loose connector block connection, 218, 219
loss of circuit integrity, 219, 227–9, 228–9
low-energy cardioversion, 369
lower rate behavior, 263
LQTS, see long QT syndrome
LV threshold, 504–7, 506–8
Lyme disease, 87
magnetic resonance imaging, 556, 560–1
malignant disease, 87
managed ventricular pacing from Medtronic, 256–7
Marfan’s syndrome, 87
measured data, 302–3, 304–5
measurement of pacing, sensing thresholds, 180–3
mechanical, pulse generator, or interaction “failure” preventing delivery of therapy, 499–501
mechanism of improved efficacy with biphasic waveforms, 31
medical advisories, 608–10, 609–610
medications, 601–3, 602t
membrane stabilization, 31
metal detector, electromagnetic interference, 566
mexiletine, 36
MI, see myocardial infarction
microwave, electromagnetic interference, 566
middle cardiac vein, 283, 283–4
minute-ventilation sensors, 384, 384–5
mitral regurgitation, 71
mode resetting (power-on reset, or electrical reset), 552, 555–6, 556
morbidity, mortality, effect of pacing mode on, 55, 55–62, 57t, 58–60
moricizine, 409
morphology, 351–3, 354–6, 458–9, 460–1, 469–71
morphology algorithm errors, 477–81, 478
MVP, see managed ventricular pacing
myocardial infarction, 502
myocardial stimulation, electrophysiology of, 2, 2–3
myocardial thermal damage, 557
myotonic muscular dystrophy, 87
N-acetylprocainamide, 36
neurally mediated reflex syncope, 83t, 92–4, 93–5
neurocardiogenic syncope, carotid sinus hypersensitivity, 124
neuromuscular disease, 87
new symptoms after pacemaker implantation, 443–5
secondary to pacemaker placement, 222–6
new technologies, 563
noise, pacemaker responses to, 551–69
non-INDUSTRIAL, home environments, 565–9, 566t
nonresponders, 100
onset, 462–5, 472
operative evaluation of pacing systems, 409t, 414–15
optimal ventricular pacing sites, 59–62, 59t, 61t–62t, 63
optimizing programming, 370–5, 371t
optimizing site of pacing, 66
oral, 36
P-wave oversensing, 487–8
pacemaker clinical follow-up visit, 581–90, 584t
follow-up, 576–81
Index

pacemaker (continued)
  inhibition, 557
  leads, 524, 524–38, 525–7
  nomenclature, 14–15, 14t
  programming, 300–33, 301t
  radiography, 517–49
  responses to noise, 551–69
  selection, 121–24, 122, 122t
  syndrome, 227
  troubleshooting, 401–12, 401t
  pacemaker–ICD interactions, 499
  pacing
    after cardiac transplantation, 100–1
    in congestive heart failure, 62–6
    in hypertrophic obstructive cardiomyopathy, 72–3, 73
    mode, 235–66
    effect on morbidity, mortality, 55, 55–62, 57t, 58–60
    overview, 3–8
    parameters, 457–65
    problems, 503–4, 505t, 506
    sensing threshold evaluation, 406–10, 408–9, 409t
    sensing thresholds, measurement of, 180–3
  pain, 211–14, 213
  patient concerns during follow up, 606–12
  patient history, physical examination, 445–51, 447
  patient-related factors, 501
  PEA sensor, see peak endocardial acceleration sensor
  peak endocardial acceleration sensor, 385, 385–6, 386
  pectoral myopotential interference, 481
  pediatric patients, 186–8, 188–90
  pericarditis, 209–10
  permanent damage to pulse generator, 557
  peroneal muscular atrophy, Charcot–Marie–Tooth disease, 87
  personnel, 573
  phantom shocks, 492
  phase duration, tilt, 30, 30
  phrenic nerve stimulation, 507–8, 509
  piezoelectric crystal, 382, 382–3
  pneumothorax, 205–7, 206–7, 502
  polarity
    biphasic waveforms, 30
    programmability, 323, 325–6
    polyarteritis nodosa, 87
  portions of pacemaker timing cycles, 243–7
  postimplant order set, 192–3
  postoperative or traumatic, 87
  power line, electromagnetic interference, 566
  PR Logic, 363–6, 365
  PR pattern, 364–5
  premature battery depletion, 455–7
  premature beats, biventricular timing cycles, 265–6
  primary preventions, 102–13, 103, 103t
  proarrhythmia, alteration of defibrillation threshold, 502
  probabilistic nature of defibrillation, 502
  procainamide, 36, 87, 409
  programmable parameters, 301t, 303–11, 394–6, 396–7
  programmable waveforms, 135
  programmed parameters, 302, 303
  programmers, 300
  programming, 300–79, 392, 392–6, 393–5
  assessment, 333–5, 336–9
  defibrillator programming, algorithms, 340–67
  diagnostics set-up, 333–5, 336–9
  hysteresis, 272, 311–14, 312–16, 338
  optimizing, 370–5, 371t
  output, 314–21, 316, 318–21
  pacemaker programming, 300–33, 301t
  programmers, 300
  rate-adaptive parameters, 333, 334–5
  resynchronization algorithms, 508–9
  during routine follow-up, 335–40, 340
  unexpected, 335
  ventricular therapies, 367–9
  programming sequence, 584–9
  progressive depolarization, 18, 20
  progressive external ophthalmoplegia, Kearns–Sayre syndrome, 87
  promoting continuous biventricular pacing, 138
  propafenone, 36, 409
  propranolol, 36
  pseudomalfunctions, 424–43, 426–7
  pulse generator assessment, 590–3, 593–4
  pulse generator pocket, 144–6, 145–6, 210–11, 211–13
  pulse generator replacement, 191, 191–2
  pulse generators, 12–14, 406, 407, 518–20, 519–21, 521, 522
  pure atrioventricular block, 123–4
  pure sinus node dysfunction, 122
  QRS duration, 99–100
  QRS vector fusion, 67, 67–8, 68–9
  QT interval, 72
  quinidine, 36
  R wave, pacing threshold, 463–5
  radar, electromagnetic interference, 566
  radiation, electromagnetic interference, 566
  radiofrequency ablation, 556
  radiographic assessment, 589–90
  radiography, 449–51, 451–5, 452–4, 517–49, 596
  rate-adaptive atrioventricular timing, 398
  rate-adaptive pacing, 380–400
  cardiac resynchronization devices, 396–8
  indications for, 380–91
  sensor applications for hemodynamic management, 391–2, 392
rate-adaptive pacing with cardiac resynchronization devices, 396–8
rate-adaptive parameter programming, 584–9, 588–9
rate-modulated asynchronous pacing, 239
rate-modulated pacing, 238–9
rate programmability, 309, 309–11, 310–11
rate-related aberrancy, 481
rate smoothing, 274, 274–5, 275
rate-variable or rate-adaptive atrioventricular interval, 247, 247–8
real-time telemetry, 454–7, 458–9, 274–5, 275
recalls, 608–10, 609–t610
refractory periods, 323–30
biventricular pacing, 266
shortening, 31
regularity, 366
relationship between defibrillation threshold, dose-response curve, 23–4, 24
remote access monitoring, 603–5, 604, 605t, 606
reprogramming, 557
requirements for device follow-up clinic, 573–6
resources for lead performance, survival data, 129–32, 130–2
response to magnet application, 268–9, 269–71
response to therapy, 459, 468, 472–3
resynchronization, 234–99
resynchronization devices, leads, 138–9, 139–40
retrieval of previous data, records, 581
reversion to fallback or noise reversion mode or electrical reset, 557
rheumatoid arthritis, 87
rhythm ID, 363
right bundle branch block, 100
right ventricular apical anterior interventricular site, 284–5
lateral venous stimulation, 285
right ventricular impedance-based sensor, 386, 386–7
right ventricular pacing sites, ECG recognition, 278–9, 279–80
right ventricular sites along with left ventricular stimulation, 285
rightward chest leads, 277–8
RV–LV pacing offset, vector of pacing, 139–40
sarcoidosis, 87
scapuloperoneal syndrome, 87
scleroderma, 87
secondary prevention, 101–2, 101t
securing permanent leads, 175–6, 176
sensing, 6–8, 7
sensitivity programmability, 320, 321–3, 322–4
sensor applications for hemodynamic management, 391–2, 392
sensors available for rate-adaptive pacing, 380–5, 381, 381t, 382, 382t
severity of CHF, 100
sheath removal, 175
shock dose, measuring, 31, 31–2
management, 474–5
single-chamber defibrillators, 344–7, 346–9, 350t
single-chamber discriminators, 497–9
single-chamber pacemakers, 267–8, 270–2, 271, 323–6
single-chamber rate-modulated pacing, 238–9, 239
single-chamber triggered-mode pacing, 237–8, 238
sinus node dysfunction, 90–2, 91
size, longevity, 134–5
skin adherence, 218
sorin, 139, 249, 251, 257, 385, 402, 521, 557–8, 583
sotolol, 36, 409
space, 573, 573–5
specific device, lead features influencing selection, 136–40
SSIR, see rate-modulated pacing
stability, 353, 357, 358–60
onset errors, 481–2
stimulation threshold, 3, 3–5, 4–5
stimulus–T or QT, sensing pacemaker, 387, 387
stored episode data, 459, 468–73
strategies to minimize shocks, 603
subclavian approach, 150–1
sudden bradycardia response algorithms, 272, 311–14, 312–16, 338
supraventricular arrhythmias, 462–3, 475, 475–7
surface electrocardiography, 469, 470–1, 473
SVT–VT discriminators, 497
symptomatic bradycardia, 122
syphilis, 87
systemic lupus erythematosus, 87
T-wave oversensing, 448, 487, 488–90, 489–90
tachyarrhythmias, 94–6, 95t–96t
tachycardias with 1, 1 AV relationship, 460–1, 466, 468, 472
with atrial rate greater than ventricular rate, 466–7, 472
telemetered data, 455–7
temperature sensors, 387–8
therapeutic radiation, 87, 568–9, 569
threshold, dose-response curve, 22, 23
thrombosis, 214–18, 215, 217–18
timing components of ventricular avoidance pacing algorithms, 255–7, 257
timing cycles with algorithms responding to sudden bradycardia, 260, 261
implantable cardioverter-defibrillators, 266–75
unique to biventricular pacing, 260–1, 261
transcutaneous electrical nerve stimulation, 561–2
transtelephonic monitoring, 576, 577–9
transtelephonic monitoring sequence, 579–81, 583, 583t
transvenous atrial leads, 528–31, 530–1
transvenous ICD leads, 540, 540–2
transvenous ventricular leads, 517, 531–8, 532–8
troubleshooting, 401–516
cardiac resynchronization devices, 454, 502–9
diagnostic features, 412–15, 413–14
focused, 415–45
frequent, recurrent shocks, 473–92, 474t
implantable defibrillator troubleshooting, 445–68
tuberculosis, 87
twiddler’s syndrome, 222, 226
ultrasound, electromagnetic interference, 566
underdetection of ventricular arrhythmias, 465, 495–9, 498
undersensing, 436–41, 439–41, 441t, 442–3, 494–5, 494t, 495–7
unexpected device failure, 414
univentricular sensing, biventricular pacing, 263–4, 264–5
upper limit of vulnerability, 17, 17–18, 18–19, 27
upper rate behavior, 274, 274–5
upper rate behavior in biventricular pacing, 264–5, 266
V anodal capture, 504–7, 506–8
V–V optimization, see ventricular timing optimization
V–V timing, 398
variation in stimulation threshold, 5, 5–6, 7
varying pacing vector, 66
VDD, see atrial synchronous (P-tracking/P-synchronous) pacing
venous approaches, 147–53
ventricular arrhythmias, 474–5, 474t–475t
undersensing, 494–5, 494t, 495–7
ventricular asynchronous pacing, 235, 235–40, 236
ventricular inhibited pacing, 236, 236–7
ventricular lead placement, 153–61, 155–6, 158, 158–62
ventricular oversensing, 482–90, 486–8
ventricular pacing, 596, 598
avoidance algorithms, 272, 311–14, 312–16, 338
ventricular rate regularization, 63, 72, 275
ventricular sensing problems, 502–3, 504, 505t
ventricular therapies, 367–9
ventricular timing optimization, 66–72
verapamil, 36
vibration sensor, see piezoelectric crystal
viral myocarditis, 87
virtual electrode depolarization, 18–19, 20–1
VOO, see ventricular asynchronous pacing
VVI, see ventricular inhibited pacing
VVT, see single-chamber triggered-mode pacing
waveform theory, clinical practice, 32–5