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THE PRESENT AND FUTURE

STATE-OF-THE-ART REVIEW

Cardiac Pacemakers: Function, Troubleshooting, and Management

Part 1 of a 2-Part Series

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ABSTRACT

Advances in cardiac surgery toward the mid-20th century created a need for an artificial means of stimulating the heart muscle. Initially developed as large external devices, technological advances resulted in miniaturization of electronic circuitry and eventually the development of totally implantable devices. These advances continue to date, with the recent introduction of leadless pacemakers. In this first part of a 2-part review, we describe indications, implant-related complications, basic function/programming, common pacemaker-related issues, and remote monitoring, which are relevant to the practicing cardiologist. We provide an overview of magnetic resonance imaging and perioperative management among patients with cardiac pacemakers. (J Am Coll Cardiol 2017;69:189–210)

A BRIEF HISTORY OF CARDIAC PACING

Cardiac pacing, electrical stimulation to modify or create cardiac mechanical activity, began in the 1930s with Hyman's "artificial pacemaker" (his term), in which a hand crank created an electric current that drove a DC generator whose electrical impulses were directed to the patient's right atrium through a needle electrode placed intercostally. At that time, Hyman faced professional skepticism, litigation, and accusations of creating "an infernal machine that interferes with the will of God," and he never found a manufacturer for his machine (1).

After World War II, public perception changed and daring pioneers made great advances. Large, external, alternating current–powered pacemakers tethered to an extension cord gave way to battery-powered, transistorized, "wearable" pacemakers (**Central Illustration**). The birth of pacing was linked to cardiac surgery, which was burgeoning. In 1957, at the University of Minnesota, C. Walton Lillehei had performed over 300 open-heart operations on young adults and children with congenital defects. Dr. Lillehei and coworkers developed a myocardial wire for post-operative pacing. On October 31, 1957, a municipal power failure in Minneapolis lasted 3 h and led to the tragic death of a baby (2). The following day, Dr. Lillehei asked Earl Bakken, a hospital equipment engineer, to build a battery-powered device to prevent future tragedies. Bakken modified a circuit for an electronic metronome he had seen in the April 1956 issue of *Popular Electronics* that used transistors, which had been invented 10 years before. He modified the 2-transistor circuit so that the electrical pulses would pace the heart, rather than power a speaker. The device was immensely successful. He named the company he founded Medtronic. Other innovations would lead to the founding of other, now familiar manufacturers.

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ABBREVIATIONS AND ACRONYMS

CIED = cardiac implantable electronic device

CS = coronary sinus

PMT = pacemaker-mediated tachycardia

PVARP = post-ventricular atrial refractory period

RV = right ventricle

TARP = total atrial refractory period

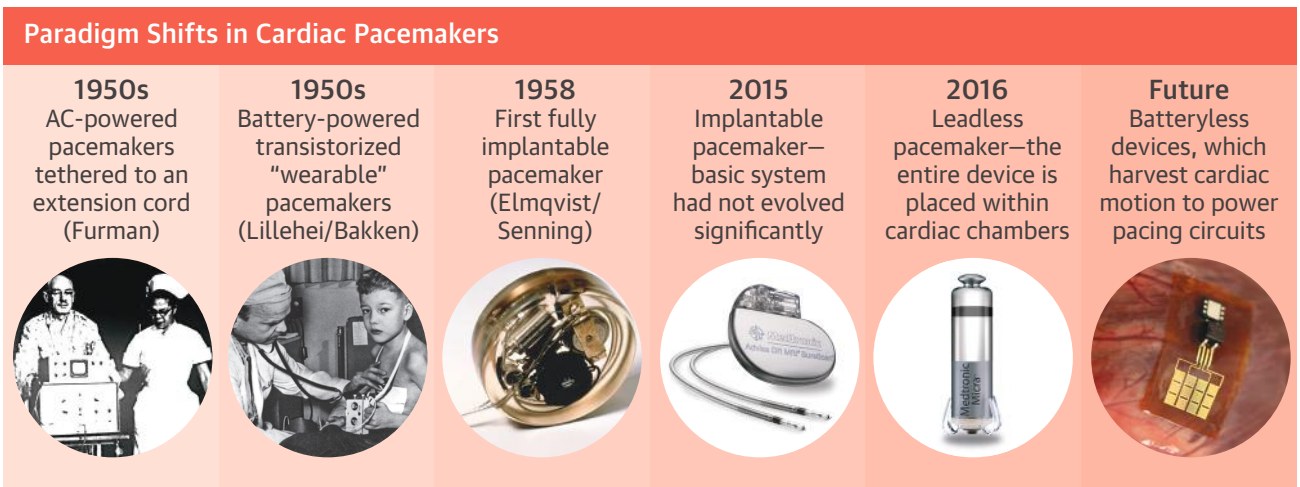
In 1960, Rune Elmqvist and Ake Senning of Stockholm placed the first fully implantable pacemaker in Arne Larsson. Larsson's wife had pleaded with Senning to use the experimental technology to help her desperately ill husband, who had complete heart block and frequent Stokes-Adams syncopal attacks. To avoid publicity, the initial implantation was performed at night, when the operating rooms were empty. The original system lasted 8 h. Arne Larsson ultimately went on to undergo over 20 pacemaker replacements, and he outlived both his surgeon and device engineer. He was an advocate for pacing until his death at 86 years of age in 2002 (3).

Although significant advances in pacing technology were developed over the next 50 years, including multichamber pacing, rate responsiveness, device size reduction, internet-based remote monitoring, and marked increases in battery longevity, the basic system paradigm of an extravascular pulse generator connected to 1 or more leads that traverse the venous system to contact myocardial tissue would not change for 50 years (**Central Illustration**). However, many pacemaker-related complications (infection, thrombosis, lead failure, and pneumothorax) are related to this basic construct, particularly the leads. This has led to a paradigm shift: the development of a leadless pacemaker, in which the entire device is placed within cardiac chambers. Batteryless devices, which harvest cardiac motion to power pacing circuits, are on the horizon as a coming paradigm shift. In this first part of the 2-part review of cardiac pacing, we explore the state-of-the-art: the basics of pacing physiology, pacing modes and indications, periprocedural management, complications, basic troubleshooting, perioperative management for nonpacemaker procedures, and cardiac magnetic resonance imaging (CMR) of patients with pacemakers. In part 2 of our review (4), we will examine recent advances and future directions, including resynchronization for heart failure, His bundle pacing, remote monitoring, and leadless and batteryless devices.

BASICS OF CARDIAC PACING

Normal cardiac activity begins in the sinus node, where cells with intrinsic automaticity act as pacemaker cells. Electrical wave fronts then spread across the atria to the atrioventricular (AV) node, which they pass through to enter the His-Purkinje system to rapidly spread to and depolarize the ventricles (**Figure 1**). When intrinsic cardiac automaticity or

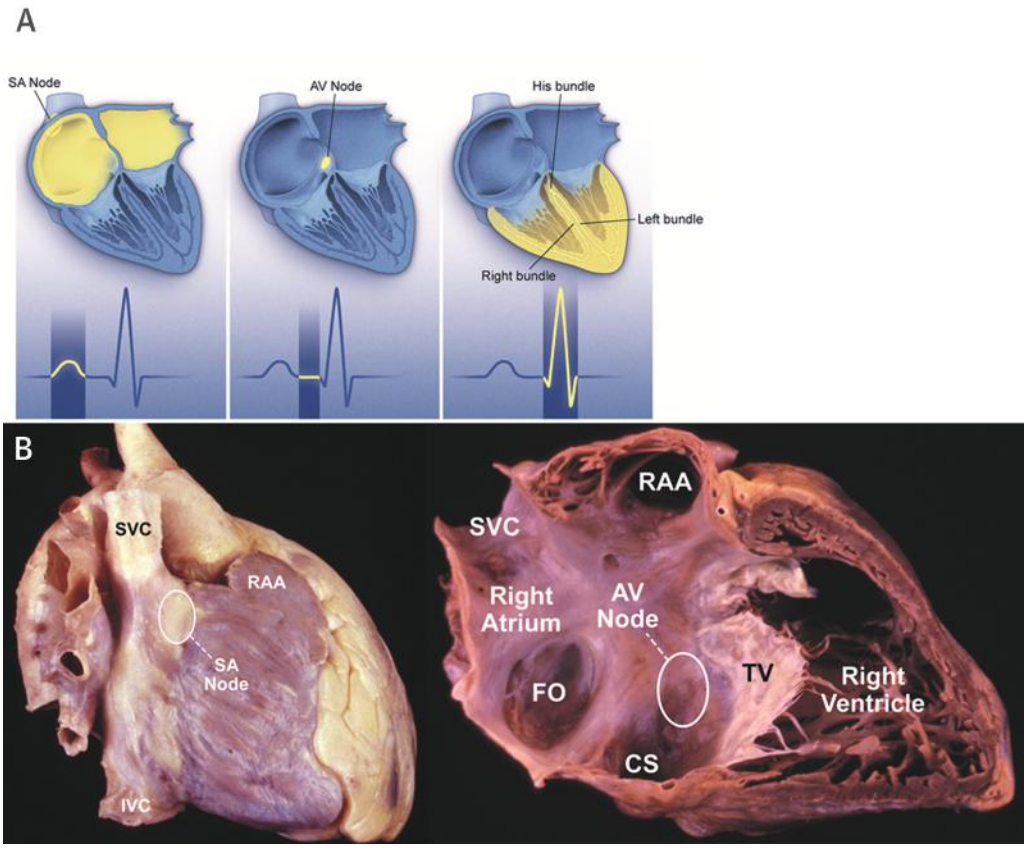
CENTRAL ILLUSTRATION An Overview of the History of Cardiac Pacing



Mulpuru, S.K. et al. J Am Coll Cardiol. 2017;69(2):189–210.

Historically, pacing developed using large, external, alternating current (AC)–powered devices, which subsequently evolved to "wearable" transistorized battery powered pacemakers—both comprise the era of external devices. A paradigm shift occurred with the introduction of the entirely implantable pacemaker, composed of an extravascular pulse generator connected to a transvenous lead in contact with the myocardium. This paradigm continues to this day. An emerging and rapidly developing new paradigm is that of leadless pacemakers, which are available for clinical use. Batteryless pacemakers that harvest cardiac mechanical motion to generate current, or that modify or add cells to introduce biological pacing activity, are under active investigation.

FIGURE 1 The Cardiac Conduction System

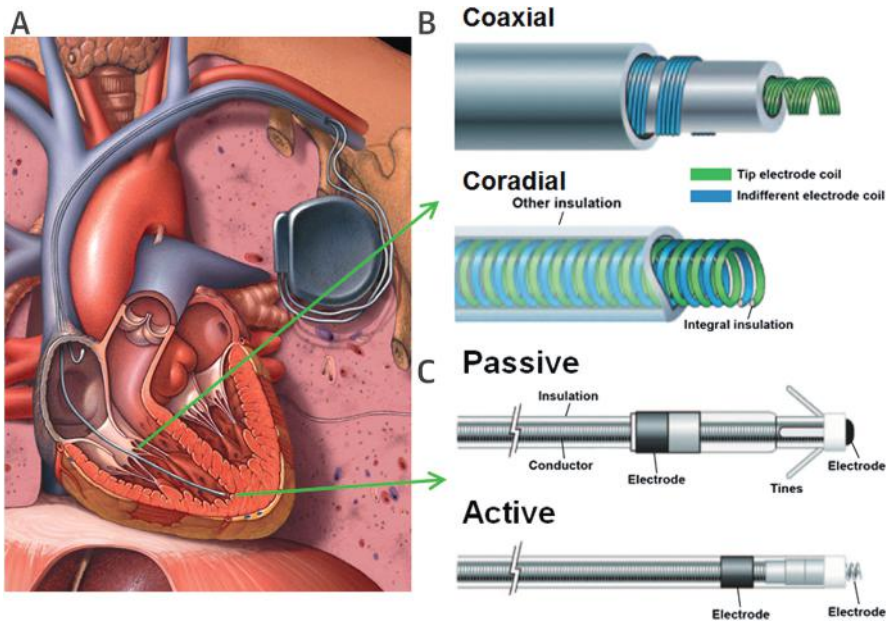


(A) Conduction begins in the SA node **(left)**. Impulse propagation through the atria gives rise to the P-wave **(bottom left)**. The impulse is then delayed in the AV node to allow blood to flow to the ventricles; wave front travel through the AV node is not seen on the surface electrocardiogram. The wave fronts then pass through the His-Purkinje system to rapidly activate the ventricular myocardium, giving rise to the large amplitude QRS complex. **(B)** An anatomic specimen showing the location of key conduction system elements. **(Left)** An external view of the heart with the region of the sinoatrial node in the epicardium at the juncture of the SVC and right atrium indicated. The structure itself is not visible to the naked eye. **(Right)** The right atrial and ventricular free walls have been removed to reveal the position of the AV node anterior to the CS and atrial to the TV, situated in Koch's triangle (bounded by the TV, CS, and tendon of Todaro; not shown). Reproduced with permission from Hayes et al. (40). AV = atrioventricular; CS = coronary sinus; FO = fossa ovalis; IVC = inferior vena cava; RAA = right atrial appendage; SA = sinoatrial; SVC = superior vena cava; TV = tricuspid valve.

conduction integrity fails, the electrical excitability of cardiac tissue allows a small, external electrical stimulus to drive myocytes to threshold, leading to depolarization of neighboring myocytes through energy-consuming biological processes and the consequent propagation of an electrical wave front, with near-simultaneous muscular contraction via excitation-contraction coupling. Pacemakers provide that external stimulus.

Pacemakers consist of a *pulse generator* or can, which contains the battery and electronics, and *leads*, which travel from the can to contact the myocardium, to deliver a depolarizing pulse and to sense intrinsic cardiac activity (**Figure 2**). Insulation materials separate the conductor cables and the lead tip electrodes. Depending on the relationship between the cables, the leads can be designed as coaxial (a tube within a tube) or coradial (side-by-side coils). The lead fixation to the myocardium may be active (with an electrically active helix at its tip for mechanical stability) or passive (electrically inert tines anchor the lead). Disruption of conductor elements and insulation materials results in either high impedance (fracture) or low impedance due to short-circuiting (insulation breach), respectively. Pacing occurs when a potential difference (voltage) is applied between the 2 electrodes. In bipolar pacing, the potential difference occurs between the lead tip (cathode)

FIGURE 2 Transvenous Pacemaker System and its Associated Components



(A) A pacemaker system primarily consists of a hermitically encased can containing the battery and all of the circuitry placed in the pectoral region. The can is connected to the myocardial tissue by a pacemaker lead. The leads contain conductor coils to the distal electrodes separated by insulation material. **(B)** The leads are of coaxial (coil within a coil) or coradial (side-by-side coils) design, depending on the arrangement of the conductor coils. **(C)** The lead tips are attached to the myocardium by a penetrating helix (active fixation) or by tines that embed in the myocardial trabeculations (passive fixation). Reproduced with permission from Hayes et al. (40).

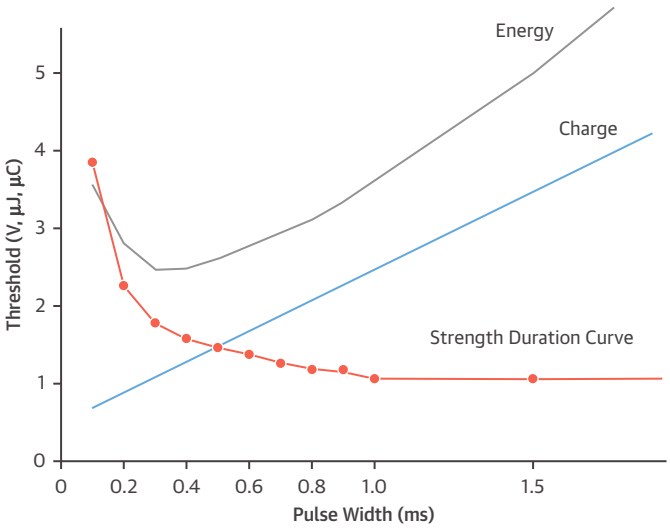
and a proximal ring (anode). With unipolar pacing, current is delivered between the lead tip and the pulse generator can. The minimum amount of energy required to depolarize myocardium is called the *stimulation threshold*. The delivered stimulus is described by 2 characteristics: its *amplitude* (measured in volts) and its *duration* (measured in milliseconds). The energy required to pace the myocardium depends on the programmed pulse width and on the voltage delivered between the electrodes. An exponential relationship (strength-duration curve) exists between the stimulation threshold and the pulse amplitude and duration (**Figure 3**). This is clinically relevant, in that optimizing the pulse width and amplitude can significantly affect current drain and battery longevity. Another clinical use for these parameters includes reprogramming to prevent extracardiac (e.g., phrenic) stimulation by lowering the pacing voltage to minimize the risk of far-field capture and increasing the pulse width to ensure cardiac stimulation. At implantation, a typical acceptable threshold is under 1.5 V with a pulse width of 0.5 ms, but this may vary, and higher values are accepted for coronary sinus leads. Pacemaker leads are built with steroid-eluting collars to prevent tissue-lead fibrosis and to mitigate the threshold rise over time that is otherwise seen.

The relationship among voltage, current, and resistance is defined by Ohm's Law ($V = IR$), where V = voltage, I = current, and R = resistance. Classically, Ohm's law uses impedance, which includes the effects of inductance and capacitance in opposing flow; however, for clinical purposes, these are generally negligible and resistance alone is used. Pacing lead conductors are designed to have a low internal resistance, to minimize wastage of energy as resistive heat. Because permanent pacemakers generate a constant voltage, the higher the pacing resistance (the load, resistance of current passage through tissue) the lower the current drain ($I = V/R$), and the lower the rate of battery depletion per each pacing pulse. Thus, lead tip electrodes are optimized to have a relatively high resistance (typically 400 to 1,200 Ω) to minimize current flow and preserve battery (1). Up to 50% of the current drain from the battery is used for pacing, whereas the other one-half is used for sensing and housekeeping functions (algorithms and storage of electrograms) (5).

Resistance is affected by many factors: lead-tissue interface, body position, and tissue edema, to name a few, but abrupt changes (>30%) may suggest lead malfunction.

Initial pacemakers functioned as pacing-only devices without the capability to sense intrinsic cardiac activity. Asynchronous pacing provided a minimum heart rate, but was complicated by atrial and ventricular dissociation, and by competition between pacing impulses and intrinsic cardiac activity. This led to the development of sensing and demand-pacing modes (discussed later). *Sensing* is the process whereby a pacemaker determines the timing of the cardiac depolarization of the chamber that a lead is in. To effectively sense, the pacemaker must optimally sense near-field depolarization signals, reject near-field repolarization signals (T-waves), and reject far-field signals (signals generated by tissues that the lead electrode is not in contact with) as well as non-physiological signals (electromagnetic interference generated by cell phones, and so on). Atrial channels are optimized to sense in the frequency range of 80 to 100 Hz, and ventricular channels in the 10- to 30-Hz range (6,7). Typical amplitude ranges for signals recorded from atrial and ventricular leads are 1.5 to 5 mV and 5 to 25 mV, respectively. Electrogram amplitudes below these values may lead to *undersensing*, the failure to detect cardiac depolarization, with possible inappropriate delivery of pacing pulses (**Figure 4**).

FIGURE 3 Relationships Among Chronic Ventricular Strength-Duration Curves From a Canine, Expressed as Potential, Charge, and Energy



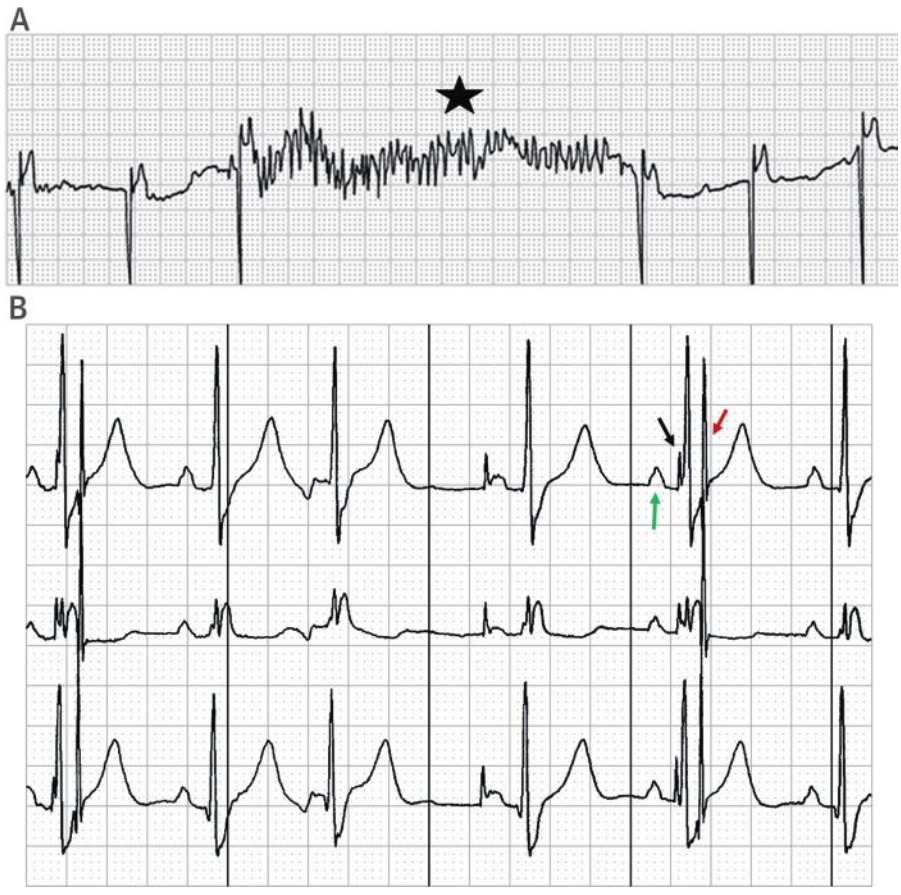
With longer pulse widths, less voltage is required to stimulate the heart. The energy requirements, which are a function of the programmed pulse width and voltage, are larger at very short and very long pulse widths. The energy expended is lower when the pulse width is kept constant at 0.4 to 0.5 ms, and the voltage is adjusted to have an adequate safety margin. Reprinted with permission from Stokes et al. (41).

PACING MODE

In response to a sensed intracardiac signal, a pacemaker may inhibit output, trigger output, or pace in a different chamber after a timed delay. This function is governed by the programmed pacing mode. The pacing mode is described with a 4- or 5-letter code (e.g., DDDR), in which the first position identifies the chamber paced (A for atrium, V for ventricle, D for dual/both), the second position indicates the chamber sensed, the third position denotes the device response to sensed events (I for inhibit, T for trigger, or D for dual [both]), the fourth position indicates whether rate response is on, and the fifth position (when used), indicates whether multisite pacing is employed in the atrium (A), ventricle (V), or both (D).

With regard to the device response (position 3 in the code), inhibition indicates that a sensed event inhibits pacing and initiates a new timing cycle. If the timing cycle (the length of which is determined by the programmed pacing rate) elapses before another event is sensed, then pacing will occur. This is most commonly used with single-chamber pacing, such as VVI or VVIR (also called *demand pacing*); the presence of an intrinsic depolarization above the pacing rate inhibits pacing. If the intrinsic rate drops below the programmed rate, pacing occurs. Rate response (discussed in the following text) detects physical activity, such as exercise, and functionally increases the lower rate (shortens the cycle length) for pacing. With triggered pacing, a sensed event may trigger pacing in the same chamber or, typically after a programmed delay, in the other chamber. A triggered mode alone is uncommonly used. With the dual mode (e.g., DDDR) both triggering and inhibition are used. In the DDD mode (**Figure 5**), inhibition occurs in the atrium if the intrinsic atrial rate exceeds the programmed lower rate. An atrioventricular clock is then started. In the absence of an intrinsic ventricular event, a ventricular pacing spike is triggered; a sensed intrinsic ventricular event inhibits pacing. In all pacing modes, a *lower rate limit* indicates the rate below which pacing occurs (this is the slowest heart rate that should be present, although some features and algorithms may permit programmable exceptions), and an *upper rate limit* indicates the fastest rate at which the pacemaker will pace, although intrinsic cardiac activity has no such limit. Common pacing modes and their clinical utility follow:

FIGURE 4 Sensing Abnormalities in Modern Pacemakers



(A) Myopotential oversensing **(star)** and inhibition of pacing in a patient with a pacemaker programmed to VVI mode. **(B)** Atrial undersensing on a tracing from an ambulatory monitor. This is best seen at the fifth ventricular complex. A P-wave precedes the intrinsic ventricular event, but this is not sensed **(green arrow)**, and an atrial pacing artifact **(black arrow)** occurs immediately before the intrinsic ventricular complex. The ventricular complex is sensed in the cross-talk sensing window. As a result, the ventricular pacing artifact **(red arrow)** is delivered early after the intrinsic ventricular event (i.e., ventricular safety pacing).

DDD. Standard dual-chamber pacing is used when the sinus mode is intact, but AV conduction impaired. Sinus activity is sensed and will trigger ventricular pacing following a programmed AV delay (p-synchronous pacing).

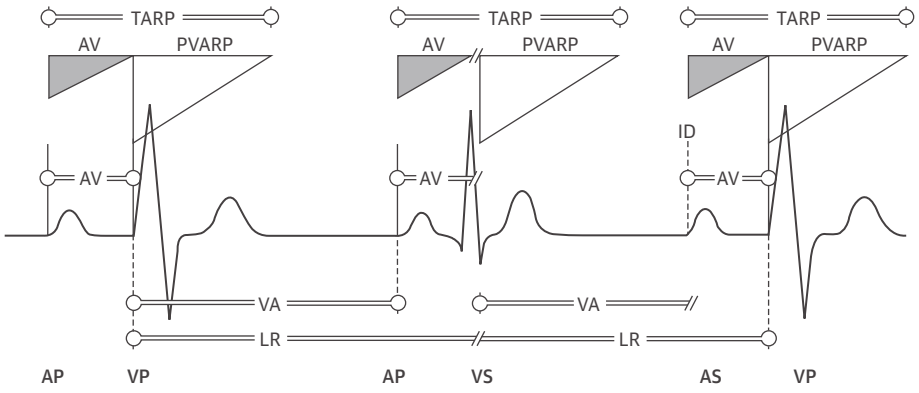
DDDR. Rate response is added when sinus and AV nodal function are both abnormal; the rate responsive feature provides chronotropic response. Most modern devices use sensors to determine the rate responsiveness to physiological demands.

VVI AND VVIR. Ventricular-only pacing is used in patients with chronic atrial fibrillation, or infrequent pauses or bradycardias. The potential for tracking atrial arrhythmias is eliminated. Rate response provides chronotropic support when needed. Single-chamber pacemakers with leads in the ventricle can deliver these modes.

AAIR. This mode is reserved for isolated sinus node dysfunction with intact AV nodal conduction. It avoids ventricular pacing and, when delivered by a single-chamber pacemaker, eliminates the need for a lead that crosses the tricuspid valve.

VOO/DOO. Asynchronous modes are programmed to avoid recognition of electrical activity, most commonly electrocautery, CMR signals, or other electromagnetic interference (EMI). These modes prevent sensing of extrinsic electrical activity, which may be "misinterpreted" as native cardiac events, inhibiting pacing, or lead to rapid ventricular pacing up to the upper rate limit if sensing occurs on the

FIGURE 5 Dual-Chamber Pacemaker Timing Cycles



The timing cycle in DDD consists of a lower rate (LR) limit, an atrioventricular (AV) interval, a ventricular refractory period, a post-ventricular atrial refractory period (PVARP), and an upper rate limit. If intrinsic atrial and ventricular activity occur before the LR limit times out, both channels are inhibited and no pacing occurs. In the absence of intrinsic atrial and ventricular activity, AV sequential pacing occurs (first cycle). If no atrial activity is sensed before the ventriculoatrial (VA) interval is completed, an atrial pacing artifact is delivered, which initiates the AV interval. If intrinsic ventricular activity occurs before the termination of the AV interval, the ventricular output from the pacemaker is inhibited (i.e., atrial pacing with intrinsic conduction [second cycle]). If a P-wave is sensed before the VA interval is completed, output from the atrial channel is inhibited. The AV interval is initiated, and if no ventricular activity is sensed before the AV interval terminates, a ventricular pacing artifact is delivered (i.e., P-synchronous ventricular pacing [third cycle]). Reproduced with permission from Hayes et al. (40).
AP = atrial pace; AS = atrial-sensed event; ID = intrinsic deflection; TARP = total atrial refractory period (which includes the AV interval and PVARP); VP = ventricular pace; VS = ventricular-sensed event.

atrial lead. In current clinical practice, these modes are only used temporarily to prevent oversensing.

BASIC PROGRAMMABLE FEATURES

Several basic programmable features are important for arrhythmia management.

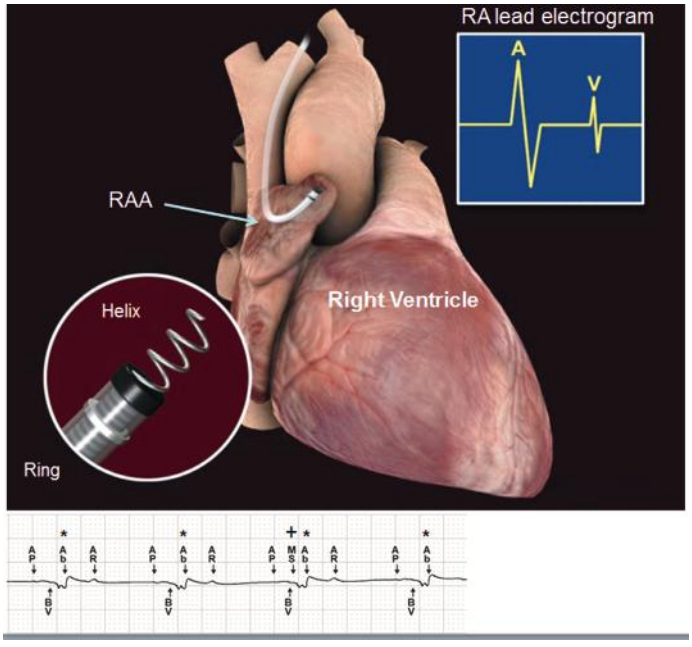
MODE SWITCHING. In dual-chamber systems, an atrial tachyarrhythmia sensed in DDD mode can lead to ventricular pacing at rates up to the upper rate limit because atrial events are tracked in the ventricle. Mode-switching algorithms identify the presence of an atrial tachyarrhythmia and switch to a nontracking mode (VVI, DVI, or DDI, with or without rate response). Although the specific algorithms vary, the presence of more atrial events than ventricular events at a rate above a programmable value triggers a mode switch, with the pacing rate then driven by the programmed lower rate limit or the sensor-indicated rate. Following a mode-switch event, atrial activity is scanned so that a tracking mode resumes upon arrhythmia termination. Mode-switching events generate internet-based alerts when available (discussed in part 2 [4]), and are reported upon device interrogation. Mode-switch events indicate the presence of atrial arrhythmias and potential need for anticoagulation. Inappropriate mode switching can occur with far-field R-wave oversensing (**Figure 6**).

AVOIDING VENTRICULAR PACING. Isolated right ventricular (RV) pacing activates the interventricular septum before the left ventricular (LV) lateral wall, seen as a left bundle branch block pattern on the electrocardiogram (ECG) due to propagation of the electrical wave front away from the sternum. This results in LV dyssynchrony and mismatched timing between chamber walls, with deleterious effects on LV function and adverse clinical outcomes, including

TABLE 1 Recommendations for Permanent Pacing in Sinus Node Dysfunction

Class I: Permanent Pacemaker Implantation Is Indicated for
1. Documented symptomatic bradycardia, including frequent sinus pauses that produce symptoms. 2. Symptomatic chronotropic incompetence. 3. Symptomatic sinus bradycardia that results from required drug therapy for medical conditions.
Class IIa: Permanent Pacemaker Implantation Is Reasonable for
1. Sinus node dysfunction with heart rates <40 beats/min when a clear association between significant symptoms consistent with bradycardia and the actual presence of bradycardia has not been documented. 2. Syncope of unexplained origin when clinically significant abnormalities of sinus node function are discovered or provoked in electrophysiological studies.
Class IIb: Permanent Pacemaker Implantation May Be Considered in
1. Minimally symptomatic patients with chronic heart rate <40 beats/min while awake.
Class III: Permanent Pacemaker Implantation Is Not Indicated in
1. Asymptomatic patients. 2. Patients for whom the symptoms suggestive of bradycardia have been clearly documented to occur in the absence of bradycardia. 3. Patients with symptomatic bradycardia due to nonessential drug therapy.

FIGURE 6 Far-Field R-Wave Oversensing, a Cause of Inappropriate Atrial Fibrillation Detection by a Biventricular Pacemaker



(Top) The pacing lead is commonly positioned in the right atrial appendage (RAA), which hangs over the right ventricle. The spacing between the distal helix and ring electrode determines the size of the antenna of the pacemaker lead. The large signal generated by the right ventricle may be oversensed by the atrial lead (V deflection**, inset**), leading to overcounting of atrial events and inappropriate detection. **(Bottom)** An example of a device tracing in which the far-field ventricular signals were sensed on the atrial channel **(*)**, and an inappropriate mode switch (MS) (+) occurs. Also present are paced atrial impulses (AP), atrial events sensed during the blanking period (Ab), and atrial events sensed in the PVARP period (AR). Reproduced with permission from Seet et al. (42). BV = biventricular pacing; PVARP = post-ventricular atrial refractory period; RA = right atrium.

TABLE 2 Recommendations for Pacing in Acquired Atrioventricular Conduction Abnormalities

Class I: Permanent Pacemaker Implantation Is Indicated for
1. Third- or advanced-second-degree AV block: a) If associated with symptoms or ventricular arrhythmias. b) In awake, asymptomatic patients: i. In sinus rhythm, with documented asystole ≥3.0 s or any escape rate <40 beats/min, or originating from below the AV node. ii. With AF and bradycardia with 1 or more pauses >5 s. iii. With neuromuscular diseases such as myotonic muscular dystrophy, Kearns-Sayre syndrome, Erb dystrophy (limb-girdle muscular dystrophy), and peroneal muscular atrophy, with or without symptoms. iv. That is precipitated by exercise, and in the absence of myocardial ischemia. v. With average awake ventricular rates of 40 beats/min or faster if cardiomegaly or LV dysfunction is present or if the site of block is below the AV node. 2. Recurrent syncope, reproduced by CSM induction of ventricular asystole >3 s.
Class IIa: Permanent Pacemaker Implantation Is Reasonable for
1. Persistent third-degree AV block with an escape rate >40 beats/min in asymptomatic adult patients without cardiomegaly. 2. Asymptomatic second-degree AV block at intra- or infra-His levels found at electrophysiological study (HV >100 ms). 3. First- or second-degree AV block with symptoms similar to those of pacemaker syndrome or hemodynamic compromise. 4. Syncope not demonstrated to be due to AV block when other likely causes have been excluded. 5. Symptomatic neurocardiogenic syncope associated with bradycardia documented spontaneously or at the time of tilt-table testing.
Class IIb: Permanent Pacemaker Implantation May Be Considered for
1. Neuromuscular diseases such as myotonic muscular dystrophy, Erb dystrophy (limb-girdle muscular dystrophy), and peroneal muscular atrophy with any degree of AV block (including first-degree AV block), with or without symptoms, because of unpredictable progression of AV conduction disease. 2. AV block in the setting of drug use and/or drug toxicity when the block is expected to recur even after the drug is withdrawn.
Class III: Permanent Pacemaker Implantation Is Not Indicated in
1. Asymptomatic first-degree or fascicular block AV block; and type I second-degree AV block at the supra-His (AV node) level. 2. AV block that is expected to resolve and is unlikely to recur (e.g., drug toxicity, Lyme disease, or transient increases in vagal tone or during hypoxia in sleep apnea syndrome in the absence of symptoms). 3. Asymptomatic hypersensitive cardioinhibitory response to carotid sinus stimulation. 4. Situational vasovagal syncope in which avoidance behavior is effective and preferred.

AF = atrial fibrillation; AV = atrioventricular; CSM = carotid sinus massage; HV = His-ventricle; LV = left ventricular.

heart failure and mortality (8). Several studies have reported RV pacing-induced cardiomyopathy rates of up to 20% with frequent RV pacing among patients with preserved ejection fraction. Male sex, wide native QRS complexes, and frequent RV pacing (>20%) are reported predictors of RV pacing-associated cardiomyopathy (9). For this reason, algorithms to avoid or minimize RV pacing have been developed for dual-chamber pacemakers. The fundamental concept is adjustment of timing intervals to deliver atrial pacing whenever required, but to extend the AV interval as long as possible to permit intrinsic conduction and thus eliminate the need for ventricular pacing. Implementations include use of an atrial pacing mode (AAI) with an automatic switch to ventricular pacing (DDD) if AV conduction fails, and AV interval prolongation to supraphysiological values with shortening if intrinsic conduction does not manifest. These algorithms effectively decrease the percentage of ventricular pacing in patients without permanent complete AV block (10,11). The benefit of RV pacing avoidance algorithms on development of atrial fibrillation, heart failure, and mortality is less clear from clinical trials. Moreover, very long AV delays (>400 ms) result in atrial contraction occurring during early diastole, resulting in cannon A waves and adverse hemodynamics, referred to as "pseudopacemaker syndrome." RV pacing avoidance

TABLE 3 Pacemaker-Related Complications

Complication	Reported Frequency (%)
Pneumothorax	0.9–1.2 (22)
Cardiac perforation	<1 (44)
Hemothorax	<1
Significant pocket hematoma requiring	3.5 (19)
Surgery	
Interruption of anticoagulation	
Prolongation of hospital stay	
Lead dislodgement	
Right-sided leads	1.8 (22)
LV leads	5.7 (22)
Venous thrombosis and obstruction	1–3 (45)
CIED device infection	1.0–1.3 (46)
Mechanical lead complications	<1

CIED = cardiac implantable electronic device; LV = left ventricular.

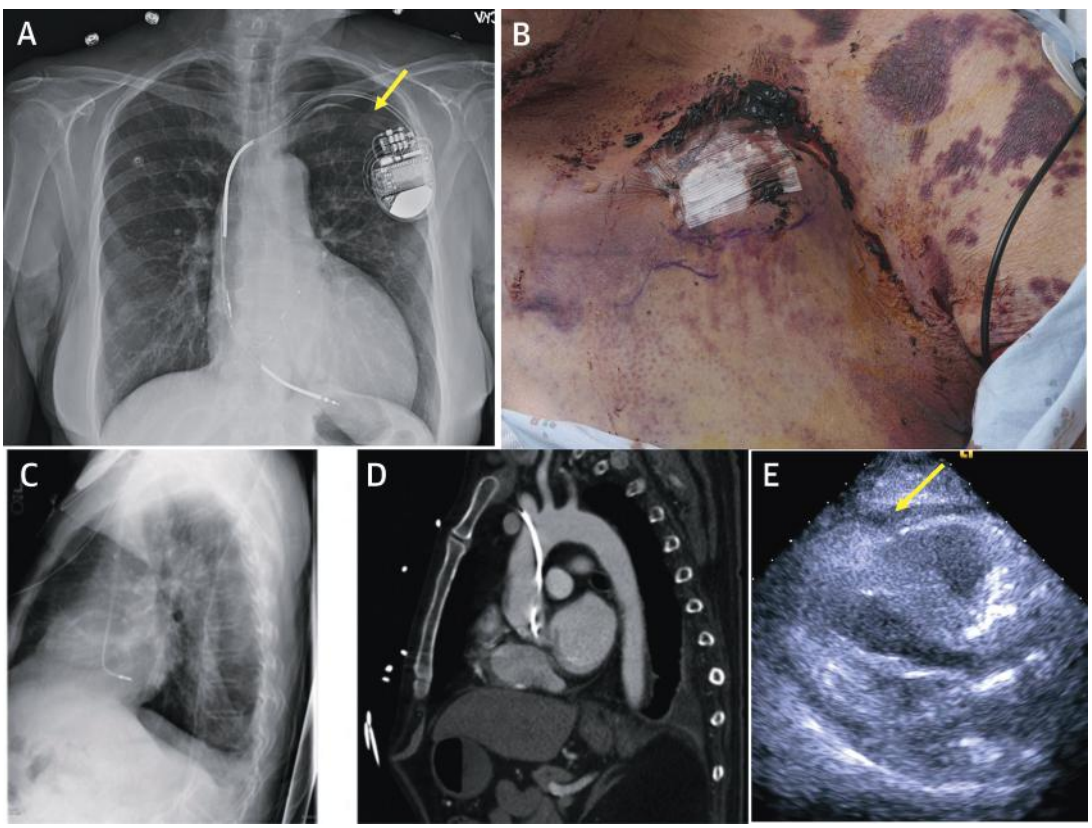
algorithms can lead to pacing below the programmed lower rate. Pause-dependent cases of ventricular proarrhythmia were reported in patients with RV pacing avoidance algorithms (12–14).

In contrast to standard dual-chamber pacemakers, biventricular pacemakers aim to maximize ventricular pacing to deliver the highest possible dose of cardiac resynchronization therapy (CRT, discussed in part 2 [4]). Algorithms that shorten the AV interval when intrinsic conduction or PVCs are sensed have been developed for CRT systems.

RATE RESPONSE

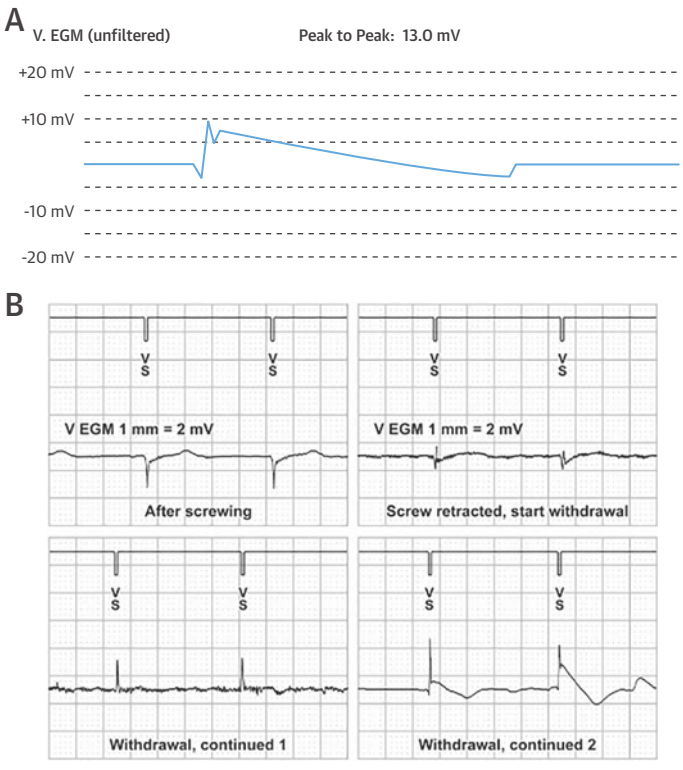
Rate response (also called rate-adaptive pacing) refers to an increase in the pacing rate in response to physical, mental, or emotional exertion. Rate-adaptive pacing improves exercise capacity in patients with chronotropic incompetence. The most

FIGURE 7 Immediate Complications Associated With Pacemaker Implantation



(A) Chest x-ray after device placement showing pneumothorax. The **arrow** highlights the border of the collapsed lung. **(B)** Hematoma associated with blood drainage from the pocket. Chest x-ray **(C)** and computed tomography (CT) image **(D)** of arterial placement of a pacing lead. Note that the lead is pointing posteriorly on a lateral chest x-ray while traversing through the ascending aorta on the CT scan. **(E)** Acute pericardial effusion **(arrow)** with hemodynamic collapse in a patient after right ventricular lead implantation. Also see Online Video 1.

FIGURE 8 Implant Predictors of Lead Stability



(A) Typical electrogram (EGM) changes at 100 mm/s sweep speed. The EGM widening and ST-segment elevation seen here are good long-term predictors of lead fixation. **(B)** EGMs in a patient who developed cardiac perforation. Negative current of injury (ST-segment) changes to a positive pattern as the pacemaker lead is pulled back into the right ventricle. Reprinted with permission from van Gelder et al. (21). VS = ventricular-sensed event.

common sensor used to drive rate response is the accelerometer, which rapidly detects physical motion. Limitations include a need for upper extremity motion to drive the heart rate (so that walking with limited arm motion on a treadmill leads to a poor rate response) and abrupt deceleration after a period of moderate-intensity exercise when the metabolic demands are still high, although programmable functionality can mitigate these limitations. Physiological sensors have used minute ventilation, cardiac contractility, blood temperature, or volume changes to drive rate response. Although more specific, they introduce greater latency and are often used with accelerometers to provide a blended response. Minute ventilation sensors transmit a low-energy current from the lead tip to the pulse generator to measure the variations in pulmonary impedance that occur with respiration. They may not be suitable for patients with tachypnea related to pulmonary disease, and may inappropriately increase the heart rate when patients are connected to ECG monitors that inject small currents into the body, which results in sensor error.

INDICATIONS FOR PACING

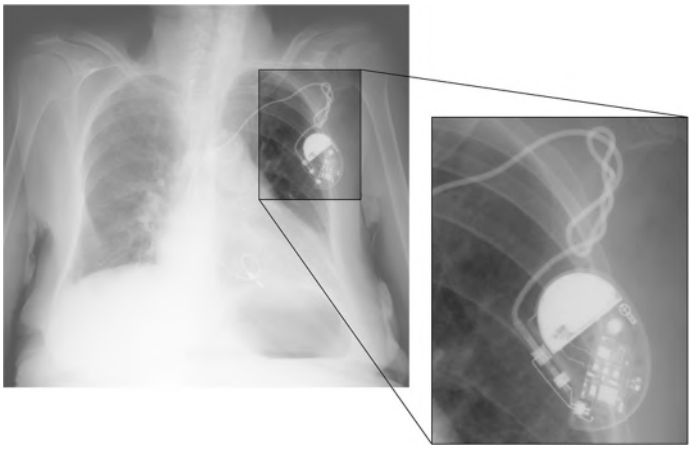
Diseases of the sinus node, AV node, or His-Purkinje system due to aging, fibrosis, inflammation, infarction, or other conditions disrupt cardiac electrical signaling. In general, when symptomatic bradycardias ensue, pacing is indicated. An important consideration is whether the bradyarrhythmia is reversible, in which case a temporary pacemaker is preferred. Examples include Lyme disease or inferior myocardial ischemia, which can present with alarming bradycardia, but often with spontaneous recovery within 1 week. Treatable causes of bradycardia should be considered, and include medications (beta-blockers, calcium-channel blockers, most antiarrhythmic drugs, ivabradine, and others), obstructive sleep apnea (especially during apnea), infections (Lyme disease, Chagas disease, Legionnaires' disease, psittacosis, Q fever, typhoid fever, typhus, among others), and metabolic conditions (hypothyroidism, anorexia nervosa, hypothermia, and hypoxia). In younger patients, hypervagotonia in association with athleticism or vasovagal spells may lead to slow heart rates. When pacing is warranted, the type of pacemaker (atrial, ventricular, dual-chamber, or biventricular) is determined by the nature of the conduction system defect (sinus node, AV node, or intraventricular conduction delay, such as a left bundle branch block).

SINUS NODE DYSFUNCTION. *Class I* indications for permanent pacemaker implantation are present when it is clear that sinus bradycardia is responsible for symptoms. The bradycardia may manifest as pauses or chronotropic incompetence. The latter is defined as a failure to achieve 70% of the age-predicted maximum heart rate or 100 beats/min during maximal exertion. *Class II* indications are present when a sinus node abnormality is the likely cause of the symptoms, but correlation is difficult. Sinus bradycardia <40 beats/min in a patient with symptoms suggestive of bradycardia constitutes a class II indication for pacing when an association between bradycardia and symptoms cannot be definitively demonstrated. Similarly, unexplained syncope in a patient with evidence of sinus node dysfunction is a class II indication. In many patients, medications cause sinus bradycardia; if the medications (such as calcium-channel blockers or beta-blockers) are necessary, then permanent pacemaker implantation is reasonable (Table 1).

ACQUIRED ATRIOVENTRICULAR CONDUCTION DISEASE. Class I indications are present in patients with severe AV conduction disease who are at risk for catastrophic events, such as syncope, falls, or sudden death. With these conditions, conduction disease is commonly infranodal, and is often characterized by a wide QRS bradycardia. High-risk conditions include symptomatic Mobitz type I (Wenckebach) or type II second-degree AV block, advanced AV block (block of 2 or more consecutive P waves), or complete (third-degree) AV block. Patients with asymptomatic complete heart block or advanced AV block also warrant pacing, despite being asymptomatic (Table 2).

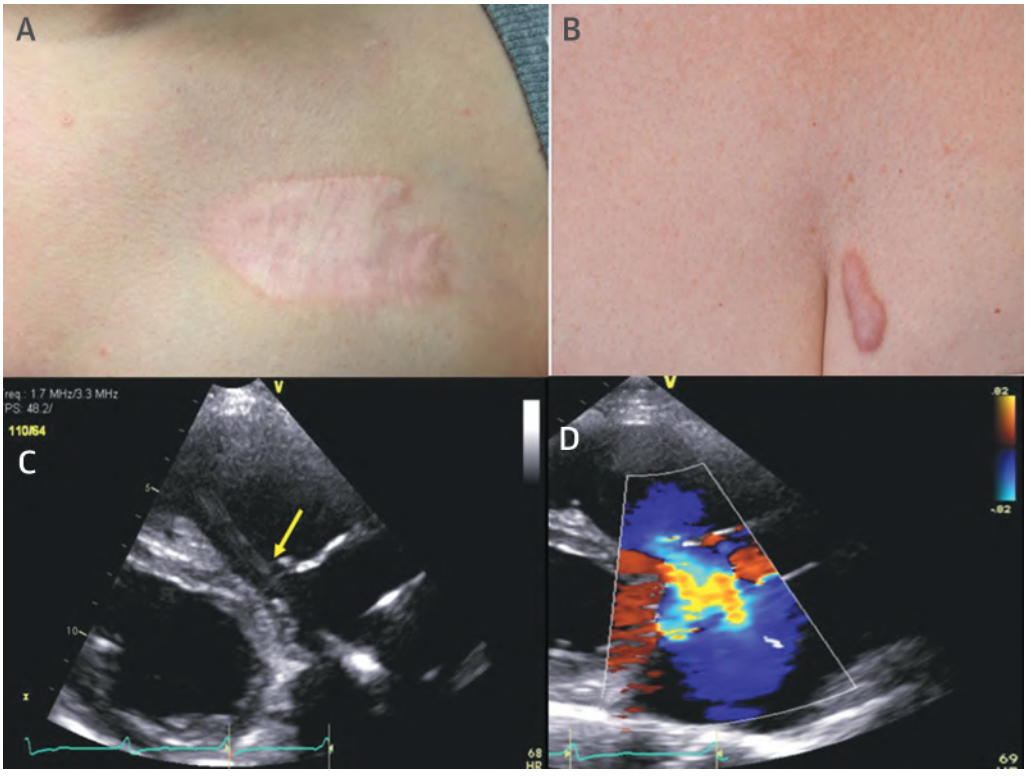
NEUROCARDIOGENIC SYNCOPE. Neurally-mediated syncope is a difficult syndrome for the clinician to diagnose and treat. Patients with confirmed carotid sinus hypersensitivity (demonstrated by a pause of 3 s or more, with carotid sinus pressure reproducing symptoms) or other neurally-mediated cardioinhibitory pauses may benefit from permanent pacemaker implant. Although pacing may be

FIGURE 9 X-Ray of a Patient With Twiddler Syndrome Who Developed Loss of Capture and High Impedance on the Ventricular Lead



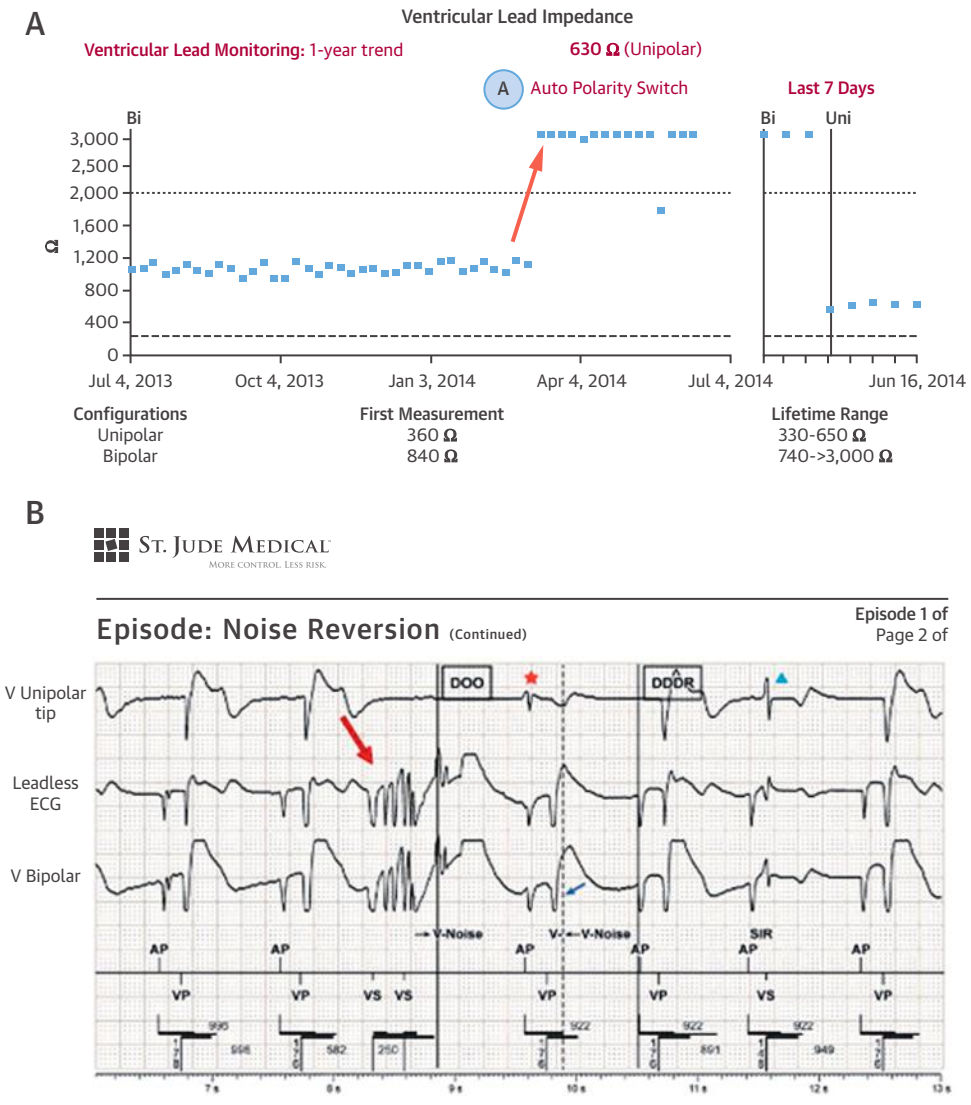
Note that several loops on the ventricular lead can put strain on the lead, leading to fracture and high impedance.

FIGURE 10 Intermediate-Term Complications



(A) Hypertrophic scar after pacemaker implantation. **(B)** Keloid formation at the site of device surgery. **(C and D)** Echocardiographic images of the right ventricular lead through the tricuspid valve **(arrow)** associated with severe tricuspid regurgitation.

FIGURE 11 Electrical Diagnosis of Lead Failure

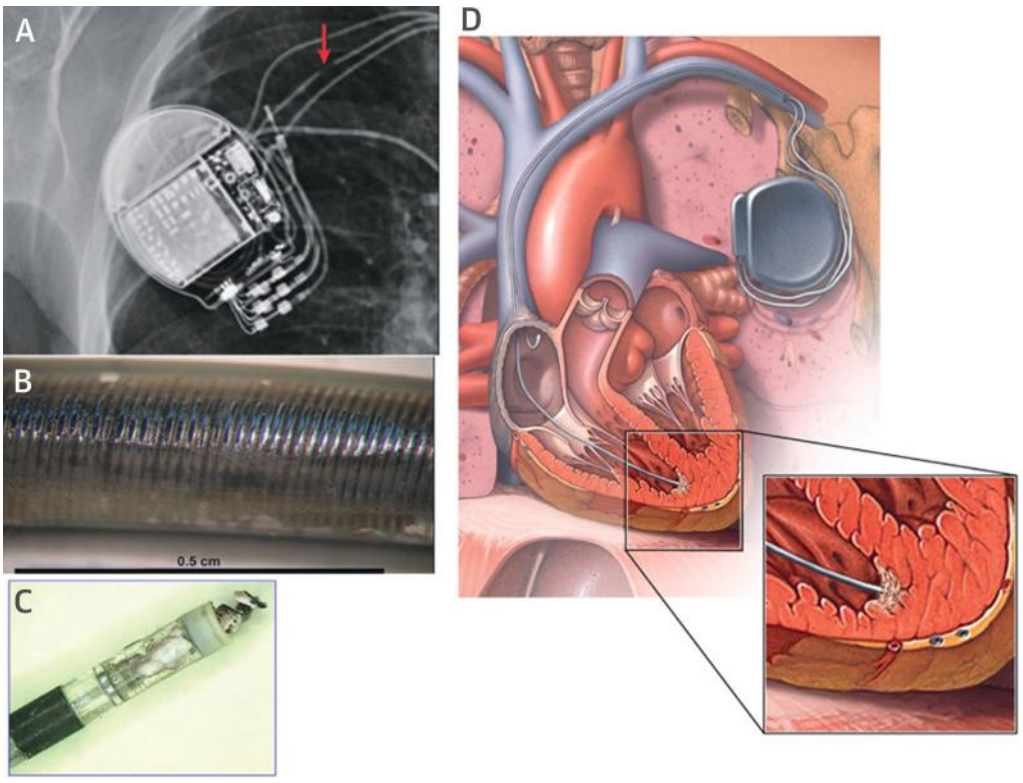


(A) Abrupt increase in ventricular lead impedance, consistent with conductor fracture. **(B)** Electrograms obtained from the patient's pacemaker. Intermittent, short-duration, high-frequency signals with nonphysiological intervals **(red arrow)** in a patient with an abrupt change in lead impedance universally points to lead fracture. The **red star** shows obligatory under sensing during DOO mode and functional noncapture **(blue arrow)**. After reversion to DDDR mode, appropriate sensing **(blue triangle)** is noted. Modern pacemakers can detect noise and switch to a synchronous mode. Most often the fractures are not radiologically detectable because they are result of very small conductor filar disruptions. ECG = electrogram; SIR = sensor indicated rate; other abbreviations as in Figure 5.

effective in patients with an isolated cardioinhibitory response, often a vasodepressor response coexists, limiting the utility of pacing. Up to 20% of patients with carotid hypersensitivity continue to experience syncopal spells during a 5-year follow-up after pacemaker implantation, and the syncope may related to the vasodepressor component of the syndrome (15). In patients with situational syncope (cough, micturition, and so on), trigger avoidance is emphasized.

NEUROMUSCULAR DISEASES. Many progressive neuromuscular diseases, such as the muscular dystrophies, are of special concern, given the unpredictable disease course and their predilection for cardiac muscle and fibrosis in and around the

FIGURE 12 Long-Term Complications Associated With Pacemakers



(A) Lead fracture on a chest x-ray. Lead fracture is associated with high impedance due to structural discontinuity **(arrow)** of the lead. **(B)** Lead insulation break, which is typically associated with low impedance. Reprinted with permission from Kutarski et al. (43). **(C)** Deposition of crystals in the lead, which may be associated with high thresholds and impedances (functional in nature). Reprinted with permission from Marshall et al. (30). **(D)** Development of fibrosis at the lead myocardial interface, resulting in high thresholds, and impaired sensing, resulting in exit block.

His-Purkinje system. A class I indication for pacing is present when second- or third-degree AV block is seen, irrespective of symptoms.

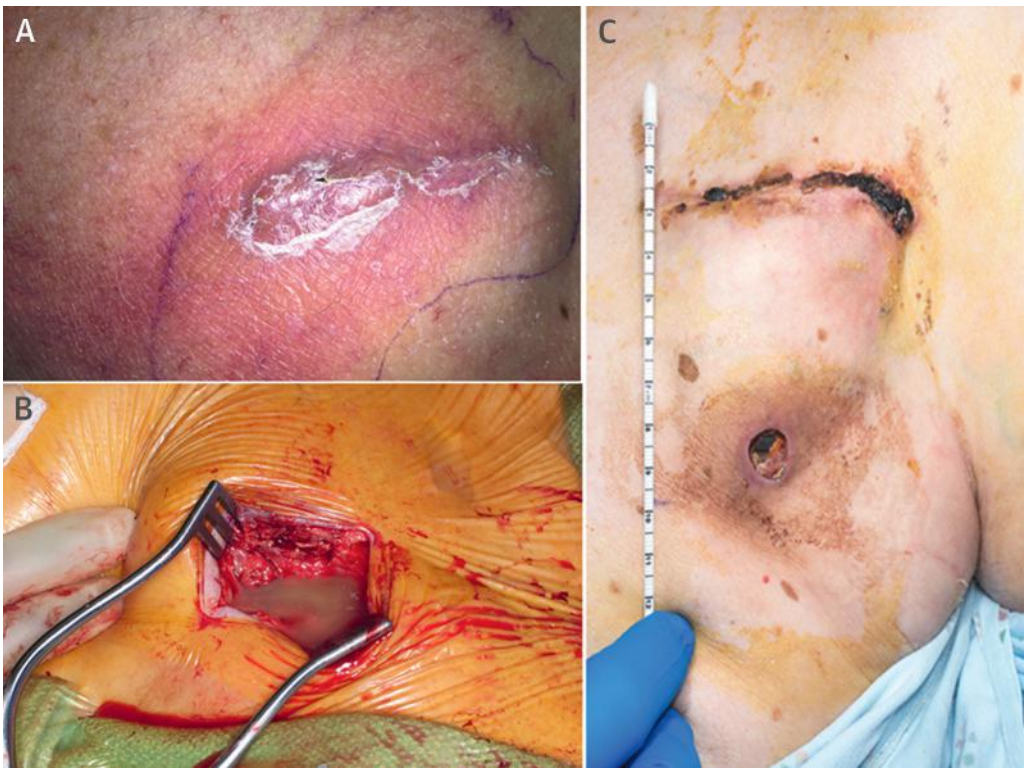
Congestive heart failure. Patients with depressed ventricular function, a wide QRS interval, and symptomatic heart failure benefit from cardiac resynchronization pacing, with reductions in heart failure and mortality, discussed in detail in part 2 (4).

IMPLANT-RELATED COMPLICATIONS

Complication rates range from <1% to 6% with current implant tools and techniques. These are broadly divided into immediate/procedure-related, intermediate-term, and long-term or late complications (Table 3). Their prompt recognition permits timely management.

IMMEDIATE PROCEDURE-RELATED COMPLICATIONS. Transvenous lead placement requires venous puncture in the pre-pectoral region. Due to the proximity of the apex of the lung to vascular targets, pneumothorax (Figure 7A) and hemothorax occur in up to 1% of cases. During implantation, the risk is lowered when vascular access using anatomic landmarks is replaced by vein visualization with cephalic vein cut down (16), contrast venography (17), or ultrasound guidance (18). Arterial puncture and inadvertent placement of pacing leads in the arterial system (Figures 7C and 7D) are avoided by advancing the guidewire into the inferior vena cava before advancing sheaths to deliver the leads. Inadvertent placement of the RV lead in the LV through a patent foramen ovale or the middle cardiac vein is avoided during implant by advancing the pacing lead across the pulmonary valve, and then withdrawing it to allow it to drop into the RV cavity. Post-operatively, if lead position is uncertain, the lateral chest x-ray, oblique imaging (right anterior oblique, left anterior

FIGURE 13 Signs of Pocket Infection



(A) Redness and skin changes in a patient 2 weeks after device surgery. **(B)** Purulence within the pocket. **(C)** Erosion in a patient who subsequently developed systemic signs of infection after pacemaker surgery.

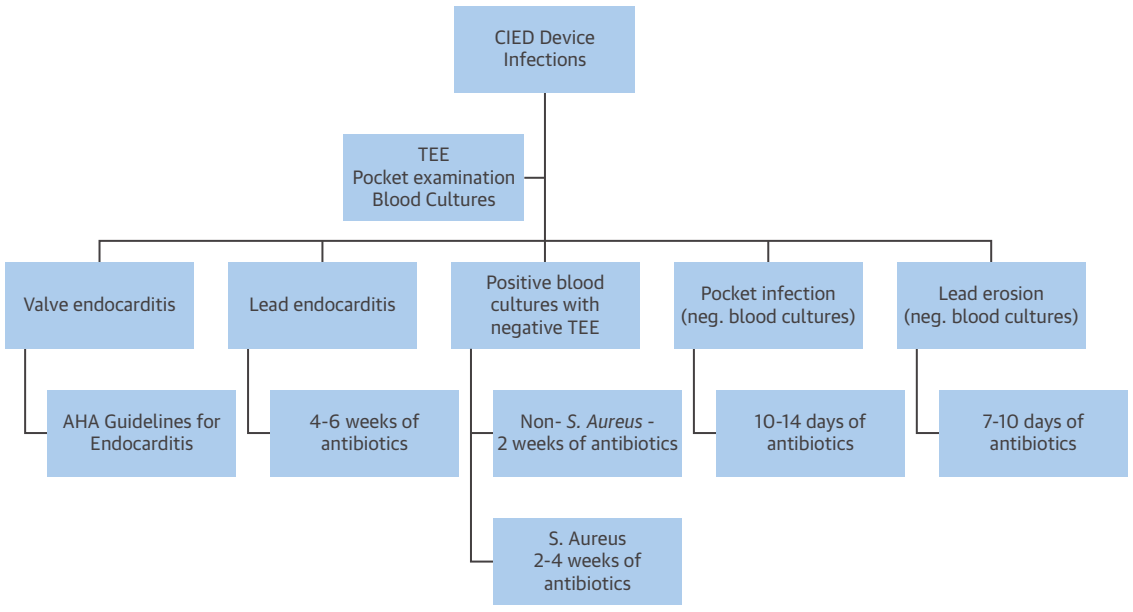
oblique), echocardiography, and computed tomography scan may be helpful. Right bundle branch morphology during pacing suggests a left-sided lead. Intracavitary LV leads may result in thromboembolism, and are generally repositioned, or if discovered late after implant, anticoagulation is instituted.

Pocket hematoma requiring intervention is infrequent (3.5%), and occurs more commonly in patients who are taking combinations of anticoagulant and antiplatelet drugs (**Figure 7B**). Uninterrupted anticoagulation with warfarin lowers the rate of hematoma formation compared with perioperative heparin bridging (19). The optimal use of periprocedural novel oral anticoagulants remains unresolved, and when possible, they are discontinued 2 to 3 days before pacemaker placement (20). Pericarditis and cardiac tamponade are mechanical complications associated with cardiac perforation (**Figure 7E**, Online Video 1). Indications for repositioning a lead when microperforation is suspected include refractory pericarditic pain, persistent effusion, or unacceptable pacing or sensing function. Passive fixation leads have a lower risk of perforation, but are used less frequently than active fixation leads due to the greater ease of site selection and ease of extraction with the latter. Use of soft stylets and recognition of negative current of injury (21) on the electrogram recording enables implanters to minimize the risk of cardiac perforation (**Figure 8**). Micro- and macrodislodgements are uncommon after device implant. The risk of dislodgement is higher for coronary sinus pacing leads (22) and passive fixation leads.

INTERMEDIATE-TERM COMPLICATIONS. Acute hematoma increases the risk of pocket and systemic infection. Device movement and subsequent lead dislodgements are infrequently seen in current practice. Manipulation of the pacemaker pocket can put undue strain on the pacemaker lead, resulting in lead malfunction (**Figure 9**) (23).

Excess scar at the incision site is associated with unfavorable cosmetic result, pain, and discomfort. Hypertrophic scar (**Figure 10A**) and keloid formation (**Figure 10B**) are due to excess interstitial tissue

FIGURE 14 Summary of CIED Device Infection Management



The duration of antibiotic therapy depends on imaging (TEE), cultures, microbiology and clinical presentation. AHA = American Heart Association; CIED = cardiac implantable electronic device; neg. = negative; TEE = transesophageal echocardiography.

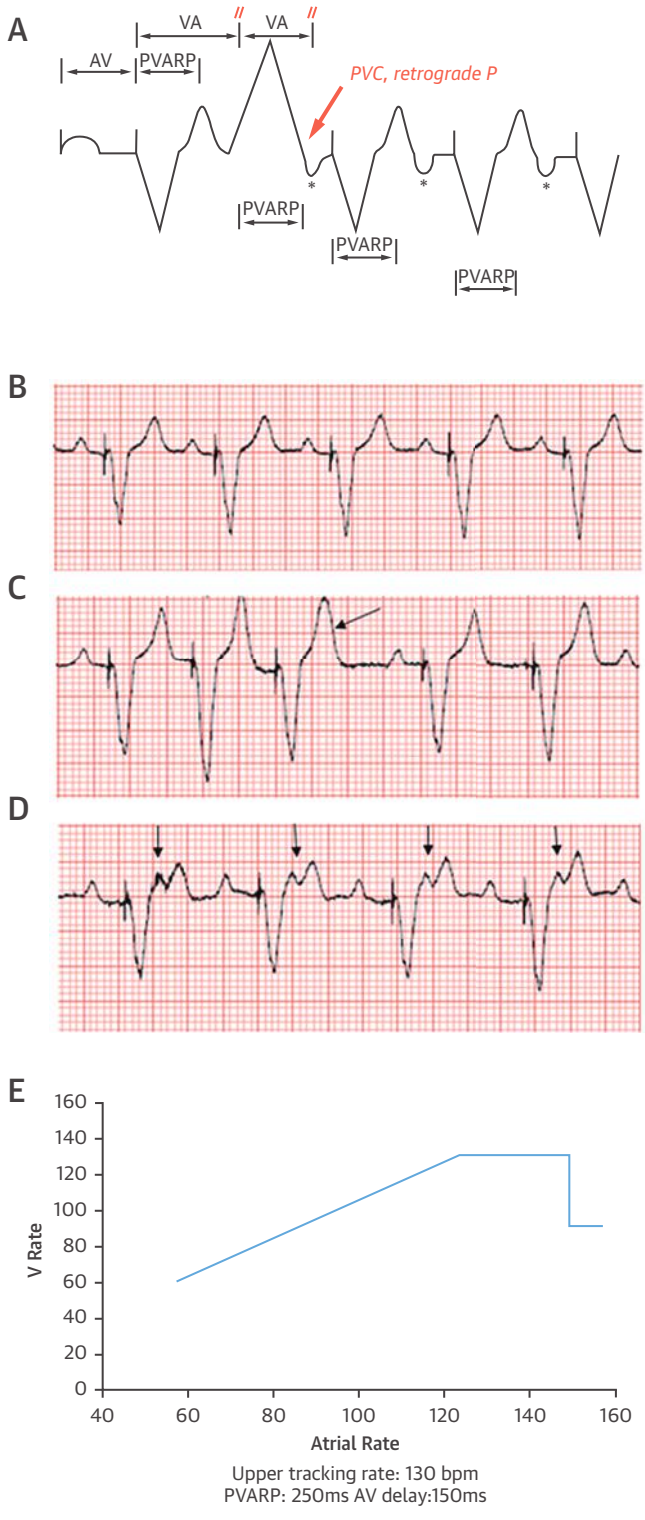
formation during the healing process. The latter entity has a raised surface and is frequently associated with itching and photosensitivity. The risk of keloid and hypertrophic scar formation is reduced by use of monofilament suture, good surgical technique, and avoidance of excess tension on the suture line. Keloids near the suture line are treated with intralesional steroid injection, silicone sheeting, and laser phototherapy.

Pacemaker lead placement through the tricuspid valve (TV) is infrequently associated with leaflet perforation and impingement of leaflet motion, resulting in valve dysfunction (**Figures 10C and 10D**) (24). When this leads to chronic fibrotic changes in the TV, tethering of the leaflet often ensues. Complications associated with TV dysfunction are avoided by using soft stylets and by crossing the valve at a lower plane, thereby avoiding chordae on the septal aspect of the tricuspid valve. Pacemaker leads often accumulate adherent fibrous strands composed of fibrotic material or thrombi. Recurrent emboli of mobile echo-dense structures increase the risk of pulmonary hypertension and, in patients with a patent foramen ovale, possibly stroke (25).

Other complications may include pocket pain or arm swelling. Pocket pain is infrequently reported after device implant. Pocket pain in the absence of device infection occurs when the device is placed in the subcuticular plane, leading to the stimulation of pain corpuscles (26). Occasionally ventricular pacing is associated with uncomfortable sensation or frank pain (27,28). In the absence of clinically significant coronary artery disease, treatment is often conservative, using ventricular pacing avoidance algorithms and occasionally lead repositioning to an area where pacing-related discomfort is minimal. The presence of multiple leads in the venous system or endothelial injury during implantation can predispose patients to development of venous thrombosis and stenosis, which can manifest as unilateral edema or superior vena cava syndrome.

LATE COMPLICATIONS. Because it is subject to repetitive mechanical stress with each cardiac cycle and with shoulder girdle movement in the hostile environment of the human body, the lead is the most common pacemaker system component to fail. Conductor fracture typically results in nonphysiological noise caused by the lead itself (high-frequency, saturated electrograms generated by intermittent contact between disrupted conductor elements, called filars) and can be associated with high lead impedance (**Figures 11** and **12A**). An insulation break results in low impedance and

FIGURE 15 Pacemaker-Mediated Tachycardia and Upper Rate Behavior



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oversensing of signals generated by surrounding structures (e.g., muscles) as conductors are exposed (**Figure 12B**) (29). Acute venous entry angle, medial venous access near the costoclavicular ligament, sharp turns in the pocket, young age, subpectoral placement of device, tight sutures, and silicone insulation are risk factors associated with lead fracture and insulation break. Occasionally, pacing thresholds and impedance rise, typically gradually over months, without any detectable fracture. This results from the development of scar tissue at the electrode myocardial interface (exit block) or the deposition of calcium hydroxyapatite crystals (30) at the lead-tissue interface, which can grow into the lead (**Figures 12C and 12D**) (30). Steroid elution in current-generation pacing leads has virtually eliminated the risk of exit block. Gradual impedance may be misinterpreted as a lead failure, but it is an important distinction; gradual impedance rise requires no action in an otherwise functional lead, whereas lead failure may require surgical intervention.

Pacemaker infections can involve the pocket, associated endovascular leads, and the valves. The risk is higher for subsequent generator changes than for the initial implant procedure. Diabetes, heart failure, renal failure, corticosteroid use, postoperative hematoma, lack of antibiotic prophylaxis, oral anticoagulation, previous cardiac implantable electronic device (CIED) infection, generator change,

FIGURE 15 Continued

(A) Pacemaker-mediated tachycardia. The first beat illustrates AV sequential pacing, followed by a PVC beat. The PVC starts a new VA interval. Any AV decoupling event (most frequently PVCs) can retrogradely conduct to the atrium. An atrial event is sensed if it falls outside of the PVARP. The VA interval expires and a new AV interval is triggered. Ventricular pacing occurs at the end of the AV interval setting up a circuitous series of events termed pacemaker-mediated tachycardia. **(B to E)** Upper-rate behavior in a patient with a dual-chamber pacemaker. The patient was programmed to an upper tracking rate of 130 beats/min. 1:1 AV conduction occurs at baseline and up to 130 beats/min **(B)**. With an increase in sinus rate above 130 beats/min, some of the P waves fall in the PVARP period **(arrows)** and are not tracked **(C)**. Progressive PR prolongation is seen before blocked beats (pacemaker Wenckebach). When the sinus rate reaches the TARP (AV interval + PVARP), every other P-wave falls in the PVARP and is not tracked, resulting in an effective ventricular rate of 75 beats/min **(D and E)**, and an abrupt drop in heart rate (2:1 block rate in this patient, around 150 beats/min). AV = atrioventricular interval; PVC = premature ventricular beat; other abbreviations as in **Figure 5**.

and use of a temporary pacemaker are known risk factors for CIED infection (31). Timing of CIED infection depends on the virulence of the organism. It most commonly occurs within a few weeks, but may present up to a year after device surgery. Early reintervention is the strongest risk factor for CIED infection. Pocket re-entry should be avoided with needle aspiration or surgery, unless the swelling is very tense and painful. Redness, purulence, erosion, discharge of gelatinous material, chronic pocket pain, and nonhealing incision site with or without thinning are signs of pocket infection (**Figure 13**). Systemic blood stream infections and endocarditis of the lead or the valves are often associated with fever, chills, positive blood cultures, and the presence of intracardiac vegetations. Appropriate referral to a specialized center managing device infections, complete device removal, and targeted antibiotic therapy are associated with better outcomes (32). The duration of antibiotic therapy is summarized in **Figure 14**.

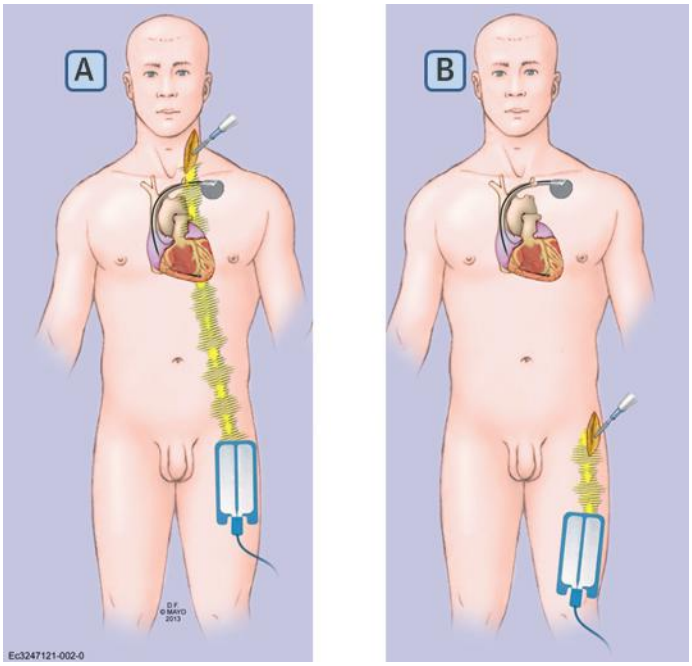
BASIC PACEMAKER TROUBLESHOOTING

BATTERY. Malfunction may occur with battery depletion. *Elective replacement indicator* denotes that 90 days of reliable function remain, whereas *end of life* indicates a battery depleted to the point of unpredictable function. Recalls are uncommon in pacemakers, in contrast to implantable cardioverter-defibrillators, and most often involve software updates to remedy the unintended consequences of the problem.

LEAD DIAGNOSTICS. Pacemakers can be programmed to pace and to sense in a bipolar mode (lead tip to lead ring) or a unipolar mode (lead tip to pulse generator). Bipolar sensing is preferred due to its smaller sensing "antenna," which minimizes oversensing of extracardiac signals. Bipolar pacing is favored to minimize pectoral muscle stimulation. However, when bipolar thresholds are elevated or sensing is impaired (e.g., due to a damaged conductor to the ring electrode), unipolar pacing or sensing may be attractive. The programmed sensitivity is the smallest signal that will be detected as a cardiac event; thus, a setting of 0.5 mV is twice as sensitive as 1.0 mV. In general, a sensing safety margin of 2 is preferred (sensitivity set to 0.5 mV if atrial electrogram is 1.0 mV). Sensitivity is adjusted to improve oversensing or undersensing. Some devices can automatically adjust sensitivity on the basis of the measured amplitude of sensed events.

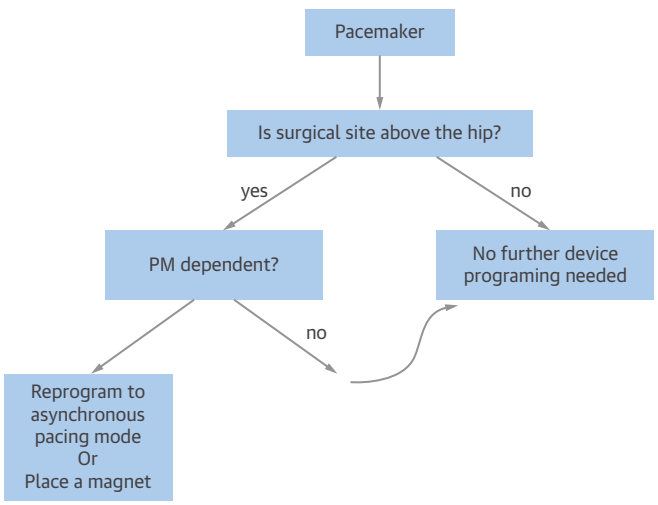
Two commonly encountered device-related arrhythmias are pacemaker-mediated tachycardia and

FIGURE 16 Role of Current Pathway and Pacemaker Interference



(A) High risk of interference: pre-operative reprogramming or magnet use required.
(B) Low risk of electromagnetic interference.

FIGURE 17 Algorithm for Pre-Operative Pacemaker Reprograming



Devices are programmed to asynchronous mode when surgical procedures involving electrocautery are performed above the hip in pacemaker-dependent patients.
PM = pacemaker.

TABLE 4 Potential Adverse Effects of CMR in Patients With Pacemaker

Adverse Effect on Exposure to External Electromagnetic Fields	Mechanism	Incidence and Risk Factors	Prevention
Mechanical movement of device or dislocation of lead	Effect of static magnetic field on ferromagnetic components of device and lead. Risk is proportional to: 1) strength of magnetic field (1.5-T vs. 3-T); 2) amount of ferromagnetic material in lead and device; 3) distance of CMR bore from device; and 4) stability of device and lead (time from implant).	• Movement of device or lead is very rare. • Current leads have almost no ferromagnetic material. • Torsional force from 1.5-T scanner typically not significant (47). • Delay CMR in fresh implants to allow lead stabilization.	• CMR-conditional devices and leads minimize/eliminate ferromagnetic material. • Avoid 3-T CMR due to lack of clinical experience.
Current induction and heating of tissue at lead interface	Radiofrequency and pulsed gradient magnetic fields can induce current in the lead, and the body can complete the circuit. Radiofrequency pulses can heat tissue. Device and leads can amplify this (the "antenna" effect) (48). Tissue edema and necrosis at the lead tip can occur. Lead parameters may change including impedance change, reduced sensing, and elevated threshold.	• The SAR describes the CMR radiofrequency energy deposition in tissues. • Risk of tissue heating depends on lead length and lead configuration, such as loops (48). • Epicardial, fractured, and abandoned leads are more likely to cause local heating (49). • Risk is higher with thoracic CMR. • Changes in lead impedance, sensing, and threshold have been reported immediately after CMR and in the long term. Changes that warrant device reprogramming or revision are rare (38,50–52). Others have reported no changes to lead parameters (53,54). • Temporary changes in battery voltage can also be seen and often recover over time (50,54). • Elevation in cardiac enzymes, typically of small magnitude, has been reported (39).	• Minimize SAR while still ensuring image quality; input from magnetic resonance physicist should be considered. • Avoid CMR in patients with epicardial, fractured, and abandoned leads. • Appropriate selection of CMR landmark. • Assessment of lead immediately after CMR to detect changes that may need intervention.
Electromagnetic effects			
EMI leading to inhibition of pacing or tacking at a faster rate	RF pulses in magnetic fields are sensed as true cardiac signals.	• Magnet mode and pauses in dependent patients are rarely reported (53,55).	• Newer devices are better shielded from EMI. • Program asynchronous pacing in pacemaker-dependent patients. • Program demand pacing in nondependent patients. • Continuous monitoring during scan.
Induction of arrhythmias	A strong pulsed gradient field results in myocardial electrical stimulation causing arrhythmias.	• Demonstrated mostly in animal experiments. • Current generated under clinical circumstances is too low to capture myocardium (56).	• Continuous monitoring of rhythm by personnel trained in cardiac resuscitation. • Limit lead length and looped leads (56).
Change in Reed switch behavior	Static magnetic field can activate the Reed switch. Consequence: asynchronous pacing detection/therapy.	• Reported incidence variable from 0%–38% (38,54,57). • Determined by distance from bore of the magnet.	• Some CMR-conditional devices use Hall sensor, which has more predictable behavior.
Power on reset causes the device to revert to default factory settings; in some devices, this can be VVI mode	Combination of static and dynamic RF pulses that induce currents in various pacemaker components in an electromagnetic field.	• Incidence 0.7%–3.5% (38,39). • More likely in devices released before 2002 (39).	• Discontinue scan if noted during monitoring. • Avoid CMR in dependent patients with devices from before 2002.

CMR = cardiac magnetic resonance; EMI = electromagnetic interference; RF = radiofrequency; SAR = specific absorption rate.

upper rate behavior resulting in high or low ventricular rates.

1. *Pacemaker-mediated tachycardia (PMT).* Ventricular events (premature ectopic beats or paced beats), when conducted retrograde to the atria, set up a circuitous cycle of ventricular pacing at the upper tracking rate (**Figure 15A**). The retrograde wave front (ventriculoatrial conduction) is sensed on the atrial channel, which then triggers ventricular pacing at the end of the programmed AV interval. Each ventricular-paced beat conducts retrogradely, setting up a circuitous pacemaker-mediated tachycardia. Atrioventricular decoupling due to retrograde ventriculoatrial conduction results in an unfavorable hemodynamic profile, and is associated

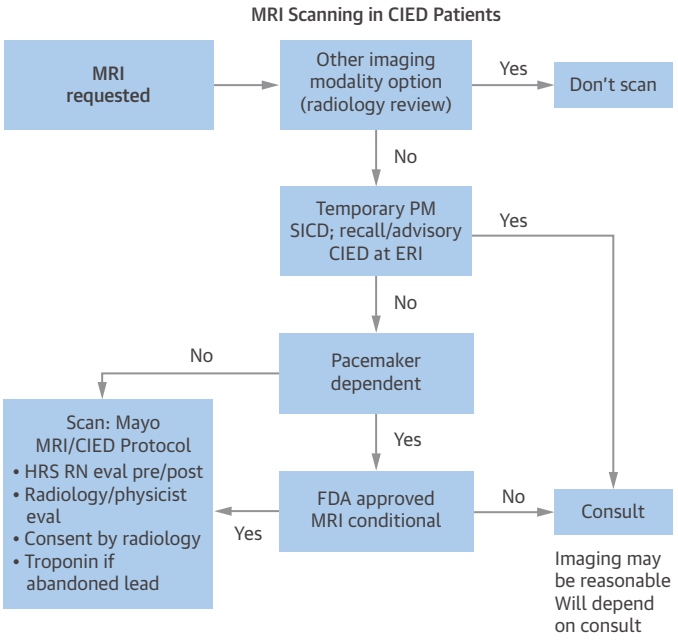
with a sensation of palpitations and fullness in the neck due to canon A waves. PMT is terminated by magnet application (which results in asynchronous pacing and prevents tracking of the retrograde P-wave, thereby breaking the cycle) or by use of PMT algorithms. Algorithms recognize ventricular pacing at the upper tracking rate, and they either extend the post-ventricular atrial refractory period (PVARP) or pace the atrium simultaneously with ventricular pacing for 1 cycle. PVARP extension results in functional atrial undersensing, whereas atrial pacing makes the atria refractory to the retrograde impulse.

2. *Upper rate behavior.* In dual-chamber pacing mode, the upper rate is determined by the total atrial refractory period (TARP). The TARP is a combination of AV delay and the PVARP. If properly programmed, the device tracks in a 1:1 fashion until reaching the programmed upper rate. As the atrial rate increases, ventricular pacing cannot violate the upper rate limit, resulting in progressively longer AV intervals. This is referred to as pacemaker Wenckebach. However, if the TARP is excessively long, abrupt 2:1 block may develop with a sudden slowing of the ventricular rate, which may cause symptoms. This occurs because when the atrial rate hits the TARP, every other event falls in the PVARP and is not tracked. Effectively, the ventricular pacing rate is one-half of the maximum tracking rate. Undesirable upper rate behavior is avoided by meticulous attention to intervals, by programming to allow a Wenckebach interval between the maximum tracking rate and the 2:1 block rate, and by allowing rate-adaptive shortening of the AV and PVARP intervals (**Figures 15B to 15E**).
3. *Inappropriate mode switches.* Occasionally, far field events or frequent ectopic beats result in high atrial counters and inappropriate detection and classification of events as atrial fibrillation. It is imperative to evaluate electrograms before therapeutic interventions for management of atrial arrhythmias.

PERIOPERATIVE MANAGEMENT OF PACEMAKERS

Electrosurgery is the application of a 100-kHz to 4-MHz electric current to cut or coagulate biological tissue. With bipolar electrosurgery, both the active and return electrodes are affixed to the cautery pen placed at the surgical site, maintaining a local current pathway; the more commonly used monopolar electrosurgery delivers current from an active electrode in the surgical field to a larger dispersive electrode usually placed on the patient's thigh or back. If electrosurgical current enters a pacemaker lead electrode, it may result in EMI. EMI sensed on the ventricular channel is misinterpreted by the pacemaker as intrinsic cardiac events that inhibit pacing, potentially resulting in bradycardia or asystole. When sensed on the atrial lead, EMI may result in tracking with rapid ventricular pacing up to the programmed upper rate limit, or inappropriate mode switch. Electrosurgery-related EMI may also cause a power-on reset (abrupt reversion to nominal mode and pacing parameters), pulse generator damage, atrial and ventricular arrhythmia, or tissue injury at the lead-tissue interface, although these are uncommon (33,34). The risk of electrosurgical EMI depends on the surgical site and dispersive pad location, with the highest risk for surgery of the heart and chest, followed by the head and neck, shoulder/upper extremity, and abdomen-pelvis. In our experience, pacemaker EMI is nonexistent with hip and lower extremity surgery when the dispersive pad is applied to the lower extremities (**Figures 16A and 16B**).

FIGURE 18 Algorithm for Cardiac Magnetic Resonance in Patients With Legacy, Nonconditional Pacemakers



Collaborative working relationships involving heart rhythm professions, radiology service providers, and physicists are required for an efficient and safe imaging program in patients with pacemakers. CIED = cardiac implantable electronic device; ERI = elective replacement indicator; eval = evaluation; FDA = Food and Drug Administration; HRS = heart rhythm services; MRI = magnetic resonance imaging; PM = pacemaker; RN = registered nurse; SICD = subcutaneous implantable cardioverter defibrillator.

Professional society guidelines recommend the following steps when surgery is needed in a patient with a pacemaker: 1) use bipolar electrocautery when possible; 2) use short-duration cautery bursts <5 s, allowing for >5 s between bursts when using monopolar electrocautery; and 3) apply pads to direct the current pathway away from the pacemaker and leads (35). When surgery is performed below the umbilicus in pacemaker-dependent patients, or when significant pacing is anticipated during the procedure, the dispersive pad is placed on a lower extremity and pacemaker reprogramming is unnecessary (**Figure 17**). When the surgical site is above the umbilicus, pre-operative pacemaker interrogation is performed, and the device is reprogrammed to an asynchronous mode for the surgery (VOO, AOO, or DOO); magnet application (which causes the pacemaker to enter an asynchronous mode while the magnet is applied) is an alternative. Pre-operative device reprogramming to an asynchronous mode (VOO, AOO, or DOO) is a *must* for pacemaker-dependent patients (**Figure 17**) to prevent device inhibition. After surgery, the device is interrogated to ensure appropriate function and reprogrammed to its original settings.

CMR OF PATIENTS WITH PACEMAKERS

CMR has become ubiquitous, so that 75% of patients with pacemakers will likely need a scan at some time following implantation (36). In the 1980s, adverse events, including death, occurred when patients with pacemakers were (usually unknowingly) scanned, as pacemaker leads could act as antennae in the CMR environment, with induced currents during imaging leading to risk of arrhythmia induction, capture threshold changes, and device damage (**Table 4**). For this reason, governmental organizations and professional society guidelines considered CMR scanning to be contraindicated in patients with pacemakers. Manufacturers subsequently developed CMR-conditional pacemakers, which are specifically designed to be safe in the CMR environment. However, the vast majority of implanted patients have legacy, non-CMR-conditional pacemakers.

A growing body of evidence has indicated the safety of CMR in patients with legacy pacemakers implanted after the early 1990s, when imaging is performed in a program established by integrated teams that include radiologists, cardiologists, specialized nurses, and physicists, with continuous monitoring during the scan. Pacemakers made since the 1990s include pass-through filters and other technology to make them more resistant to EMI. In several large series, including the Mayo Clinic series that now exceeds 1,000 scans, imaging of patients with legacy pacemakers has been safely performed (**Figure 18**) (37,38). The greatest potential risk is that of power-on reset in pacemaker-dependent patients, which may change the programmed mode from asynchronous to synchronous, permitting oversensing of CMR signals and inhibition of pacing output. This was seen in a limited number of older devices from a specific manufacturer (39). Nearly all patients with legacy systems can be safely imaged at centers with dedicated programs. Although pacemaker pulse generator artifacts (particularly when the can is low on the chest wall) may lead to artifact obscuring the image, a large case series observed that the clinical question was answered in over 98% of nonthoracic scans (38), and newer CMR processing algorithms facilitate thoracic imaging.

CMR-CONDITIONAL DEVICE AND LEADS. All major device manufacturers have developed leads and pacemakers, cardiac resynchronization devices, and implantable cardioverter-defibrillators that are magnetic resonance–conditional. CMR-*conditional* refers to devices that have no known hazards or risks under specific magnetic resonance conditions. It is notable that there are no "CMR-safe" devices, which refers to devices that have no known hazards or risks under any condition. CMR-conditional devices have minimal ferromagnetic material, altered filtering, and redesigned lead conductors to minimize current induction and heating of tissue. There are specific device and CMR conditions under which such devices can be considered safe. Device requirements include: CMR-conditional device paired with CMR-conditional leads, and absence of abandoned, fractured, or epicardial leads. The device must be implanted in the pectoral region. Magnetic resonance scanning requirements are technical, and are designed to limit energy deposition on CIEDs. These include: static magnetic field of 1.5-T, maximum specific absorption rate (SAR) value of 2 W/kg, maximum gradient slew rate of 200 T/m/s, horizontal closed bore magnet, and supine or dorsal positioning of patients (not on their sides). Although several devices are approved for whole-body scanning, some have more limited approval for scanning parts other than the thorax. Continuous monitoring during the scan (ECG, hemodynamics, and symptoms) by an advanced cardiac life support-trained provider, and interrogation before and after scanning are also required.

To summarize, CMR can be safely performed in patients with magnetic resonance–conditional devices

and at centers with dedicated programs, in nearly all patients with legacy, non-CMR conditional devices.

CONCLUSIONS

Advances in pacemakers have led to their widespread use to treat patients with bradycardias and congestive heart failure. A basic understanding of their function, troubleshooting, and management at the time of surgery or imaging enhances patient care. In part 2 of this review (4), we explore recent innovations and future directions.

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APPENDIX For a supplemental video and legend, please see the online version of this article.