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Pacemaker and ICD Troubleshooting

Sorin Lazar, Henry Huang and Erik Wissner

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Abstract

Continuous advancements in technology and software algorithms for pacemakers and implantable cardioverter-defibrillators (ICDs) have improved functional reliability and broadened their diagnostic capabilities. At the same time, understanding management and troubleshooting of modern devices has become increasingly complex for the device implanter. This chapter provides an overview of the underlying physics and basic principles important to pacemaker and ICD function. The second part of this chapter outlines common device problems encountered in patients with pacemakers and ICDs and provides solutions and tips for troubleshooting.

Keywords: signal processing, filters, pacemaker troubleshooting, ICD troubleshooting

1. Introduction: Device physics

1.1. Signal processing

Signal processing refers to the science of analyzing time-varying physical processes [1]. Signal processing is divided into two categories: analog signal processing and digital signal processing. An analog signal is continuous in time and can take on a continuous range of amplitude values. A discrete-time signal is an independent time variable that is quantized so that only the value of the signal at the discrete instant in time is known. This can be illustrated in the following example: a continuous sinewave with peak amplitude of 1 and frequency of f is described in Eq. (1):

$$x(t) = \sin(2\pi ft) \quad (1)$$

where the frequency f is measured in Hertz (Hz).

By plotting Eq. (1) a continuous curve is obtained (**Figure 1a**). If the continuous waveform represents a continuous physical voltage sampling every t_s seconds, using an analog-to-digital converter

would result in a sinewave represented as a sequence of discrete values shown in **Figure 1(b)**. **Figure 1(b)** represents the digitization of the continuous signal in **Figure 1(a)**. Variable t in Eq. (1) and **Figure 1(a)** is a continuous and independent variable. Variable n in **Figure 1(b)** is a discrete and independent variable that can take only integer values. As a result, the index n identifies the elements of the digital signal in **Figure 1(b)**. All naturally occurring intracardiac signals are continuous and all signals stored by pacemakers and defibrillators are digital.

1.2. Filtering

Filters are used for two general purposes: (1) separation of signals that have been combined and (2) restoration of signals that have been distorted in some form. Signal separation is needed when if the signal is contaminated by interference, noise or other signals. As an example, filtering is used to separate nonphysiologic high-frequency pulmonary vein potentials recorded during catheter ablation of atrial fibrillation (AF) from physiologic signals. Signal restoration is used

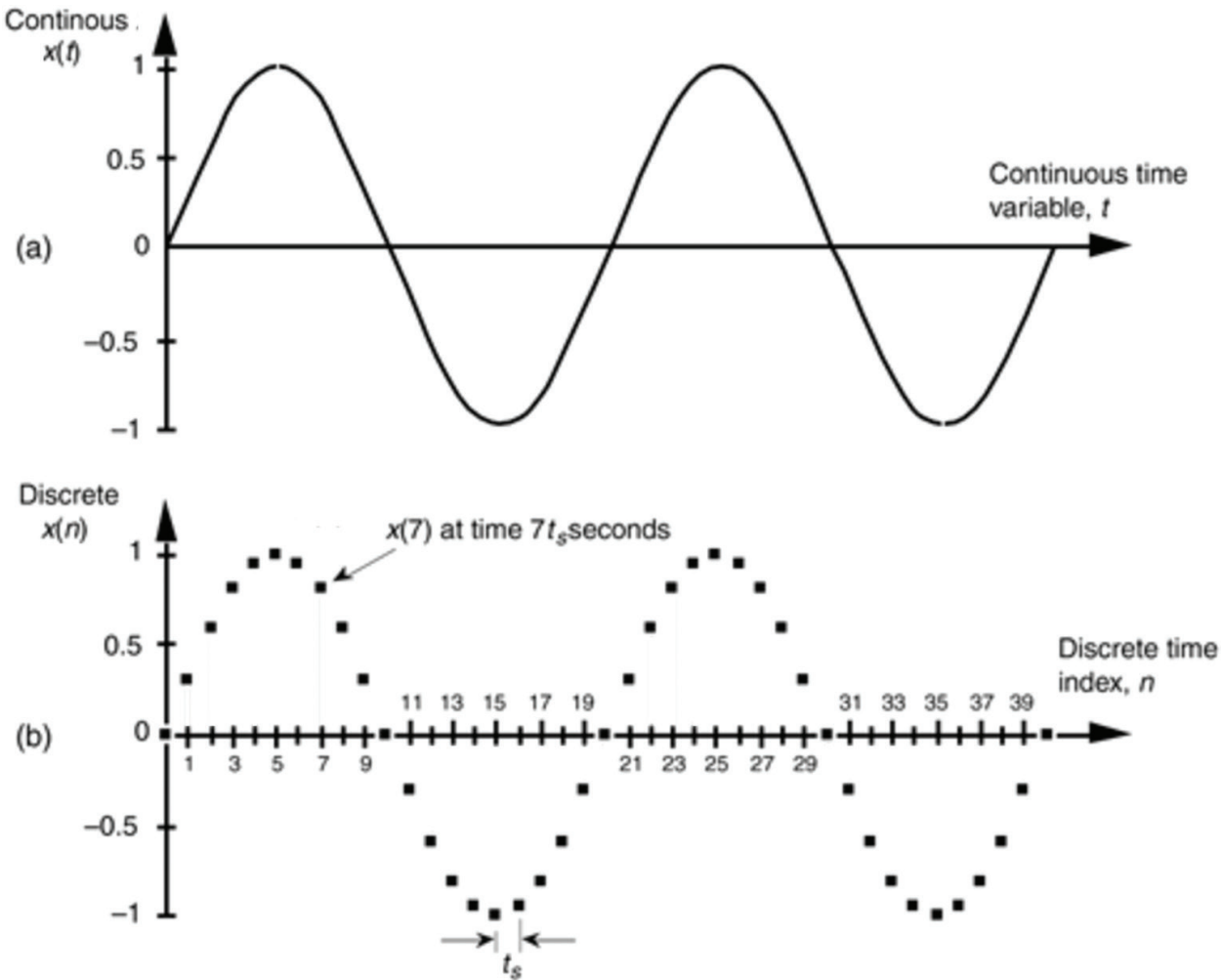


Figure 1. A time-domain sinewave: (a) continuous waveform representation and (b) discrete sample representation.

when a signal has been distorted in some form. For example, an audio recording obtained with poor equipment may be filtered to improve fidelity so the actual sound is better reproduced.

For raw signal data to be analyzed, information must be represented in either the time or frequency domain. The most commonly used filters applied in intracardiac devices are in the frequency domain. **Figure 2** summarizes the four most common basic frequency responses. These filters allow unaltered passing of some frequencies, while other frequencies are completely blocked. Those frequencies that pass through are called “passband,” while frequencies that are blocked are referred to as “stopband.” The band in-between is called the “transition band.” A very narrow transition band is called “fast roll-off.” “The cut-off frequency” is the frequency that separates the “passband” from the “transition band.” Analog filters use a cut-off frequency that is decreased to 0.707 from the original amplitude. Digital filters are less strict, and usually the cut-offs used are 99, 90, 70.7, and 50% of the original amplitude levels.

The example shown in **Figure 3** highlights the three parameters described above. An example of a “fast roll-off,” is shown in (a) and (b). A “passband ripple” example is shown in (c) and (d), and finally, “stopband attenuation” is shown in (e) and (f).

1.3. Chebyshev filters

Chebyshev filters are used to separate one band of frequencies from another. They are the most commonly used in cardiac electrophysiology applications. The primary attribute of Chebyshev filters is their speed. The Chebyshev response is a mathematical strategy for achieving a faster “roll-off” by allowing “ripple” in the frequency response.

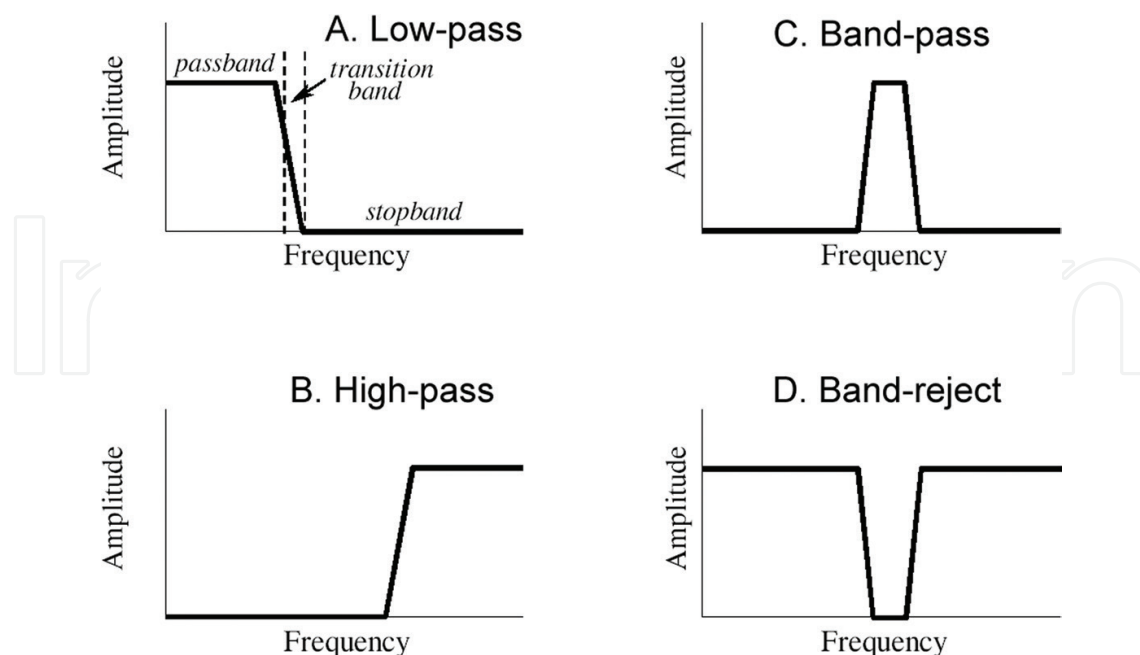


Figure 2. The four common frequency responses.

Figure 4 shows three ripple values, 0, 0.5, and 20%, for a low-pass Chebyshev filter. If the ripple decreases (good) the roll-off becomes less sharp (bad). The Chebyshev filter design is an optimal balance between these two parameters. If the ripple is 0%, the filter is called “Butterworth filter.” A ripple of 0.5% is often a good choice for digital filters. This matches the typical precision and accuracy of the analog electronics that the signal has passed through.

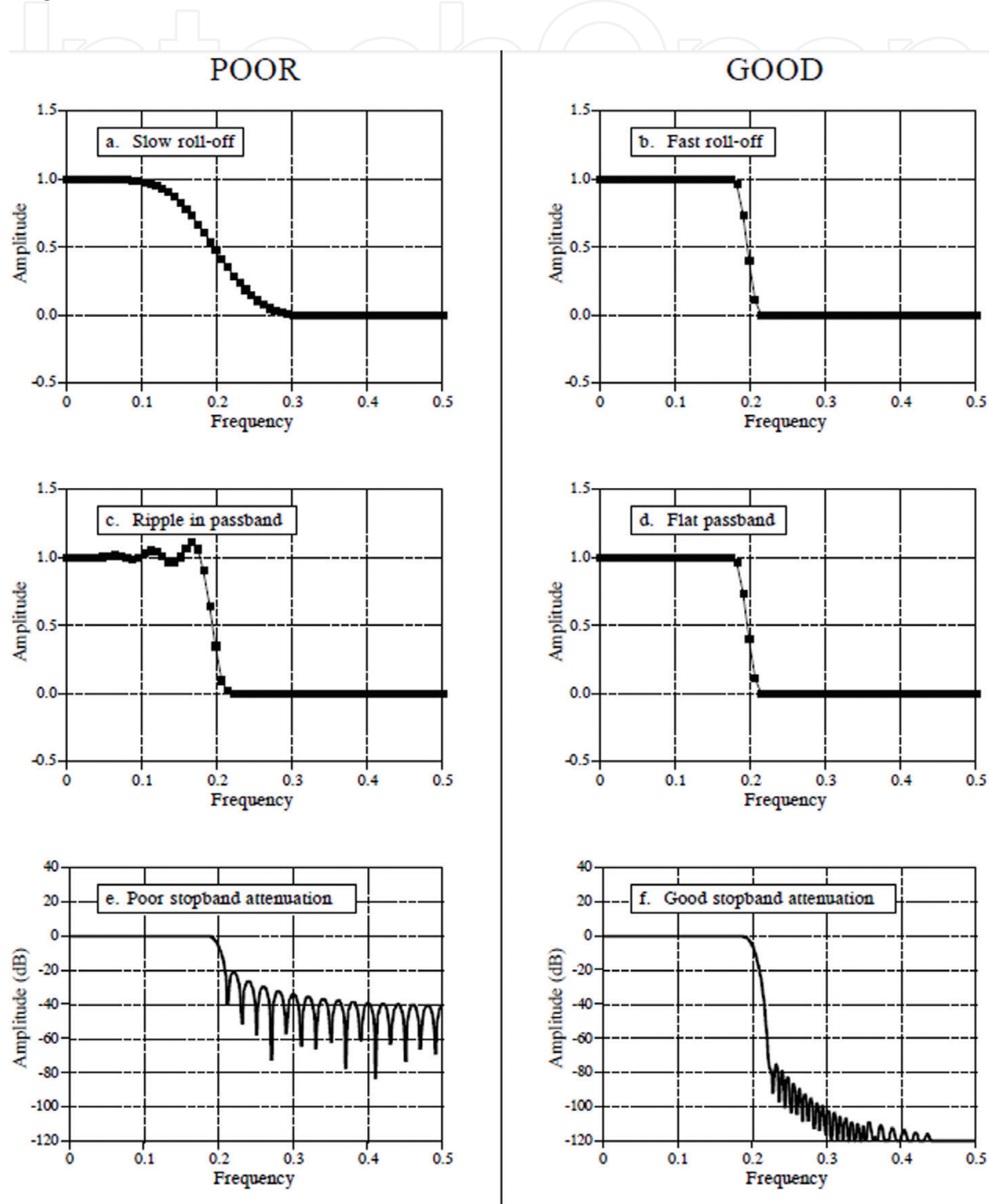


Figure 3. The three parameters important for evaluating frequency domain performance: (1) roll-off sharpness, (a) and (b); (2) passband ripple, (c) and (d); and (3) stopband attenuation, (e) and (f).

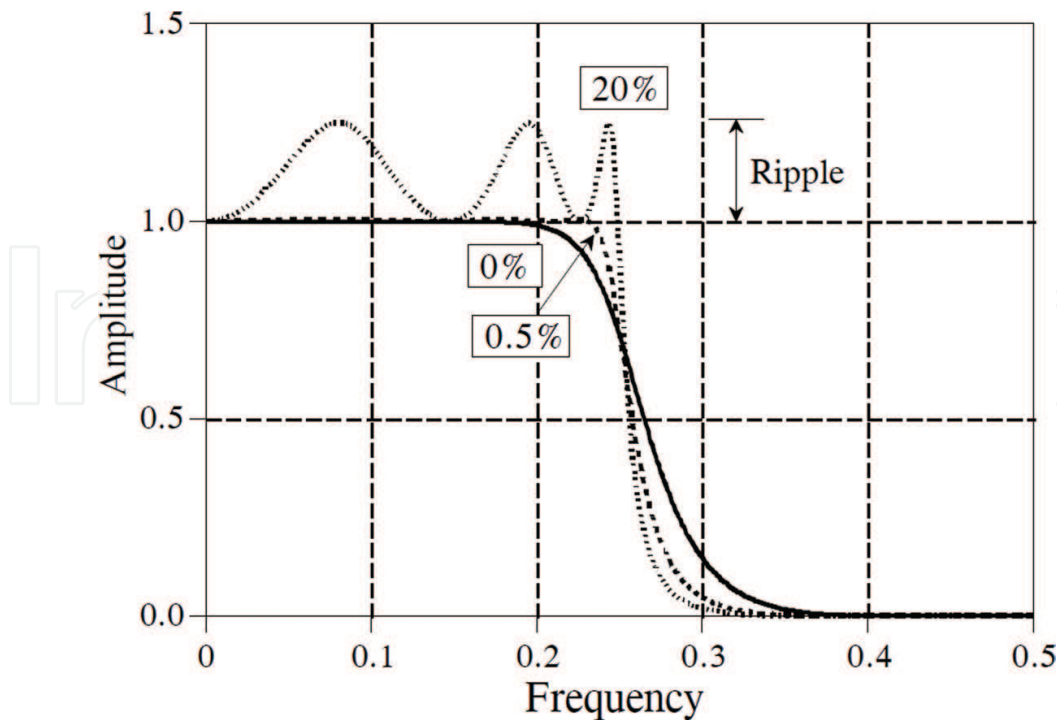


Figure 4. The Chebyshev response: Chebyshev filters achieve a faster roll-off by allowing ripple in the passband. When the ripple is set to 0%, it is called a maximally flat or Butterworth filter.

1.4. Unipolar versus bipolar recordings

All electrical circuits must have a cathode (negative pole) and an anode (positive pole) [2]. In general, there are two types of electrical circuits used in pacing systems depending on the location of the anode. In a unipolar system, as shown in **Figure 5(a)**, the metal can of the pacemaker is used as the anode (+), and the distal electrode of pacemaker lead as the cathode (-). In a unipolar system, the pacing lead has only one electrical pole. **Figure 5(b)** shows the other type, a BIPOLAR system where both the anode (+) and cathode (-) are located on the same pacing lead. In all pacing systems, the distal pole that is in direct contact with cardiac tissue is negative. All currently available ICDs are bipolar, however, based on the lead utilized, the system may be dedicated bipolar or integrated bipolar. In a dedicated bipolar design, the anode is separate from the shock coil. In an integrated design, the distal shock coil also serves as the anode for pacing and sensing. An integrated design allows for more simple lead construction, as the distal shock coil serves two purposes. A dedicated bipolar system may provide more reliable sensing than an integrated design with a shared coil, as the anode is not affected by the high-voltage shock. Unipolar pacing systems have the advantage of a simpler and more reliable single-coil lead construction. It is also much easier to appreciate the pacing artifact of a unipolar system due to the anatomic distance between lead and pulse generator with parts of the electrical circuit closer to the skin surface. In some instances, sensing and capture thresholds are better than those obtained from a bipolar system, although lower lead impedance may result in higher current drain from the battery. In order to reduce the risk of pacemaker stimulation of the pectoralis muscle and/or device oversensing of electrical signals generated by the pectoralis muscle, many of the older pacemaker models incorporated a layer of protective coating on the device side facing the muscular tissue.

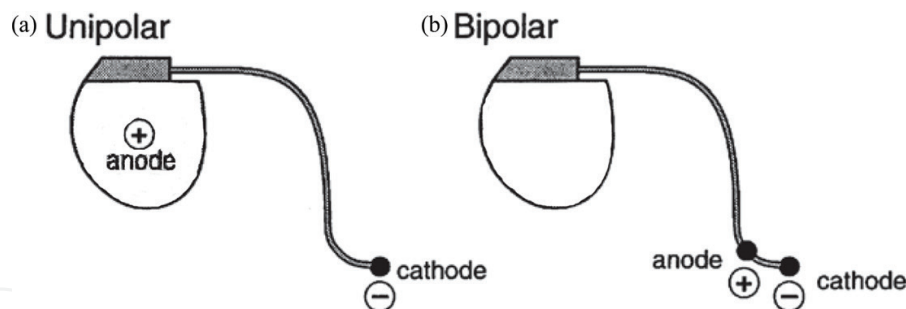


Figure 5. (a) Unipolar pacing system: the lead tip is the cathode and the pacemaker is the anode. (b) Bipolar pacing system: the lead tip is the cathode and the proximal ring is the anode. The pacemaker is not part of the circuit.

Bipolar pacing systems offer several advantages that have made this polarity choice increasingly popular, especially as dual-chamber pacing has become more prevalent in clinical practice. Because the distance between the individual electrodes is small (short antenna) and since both are located deep within the body, bipolar devices are less susceptible than unipolar systems to electrical interference caused by skeletal muscle activity or electromagnetic interference (EMI). Also, higher output settings required for unipolar pacing may result in stimulation of the pocket around the pacemaker. This problem is virtually unknown in normally functioning bipolar systems. One downside to using bipolar pacing is that the pacing artifact is very small and often difficult to discern on the surface electrocardiogram. This makes determination of proper function and malfunction more difficult. For this reason, it is not uncommon to see a pacemaker programmed to unipolar pacing but bipolar sensing.

1.5. Sensing

Sensing is the ability of the device to detect the intrinsic cardiac activity [3]. This is measured in millivolts (mV). The larger the signal in mV, the easier it is for the device to sense the event as well as to discriminate normal intrinsic from spurious electrical signals. Setting the sensitivity of a pacemaker is often confusing. When programming this value, it must be understood that the value programmed is the smallest amplitude signal that will be sensed (**Figure 6**). There is an inverse relation between sensing and sensitivity. The higher the sensing value, the lower the sensitivity to detect the intrinsic electrical signal. Thus, a setting of 8 mV requires at least an 8 mV electrical signal for the pacemaker to detect. A 2 mV setting will allow any signal above 2 mV to be sensed by the pacemaker.

1.6. Slew rate

Measurement of the intrinsic electrical signal for sensing is not simple, as the pacemaker does not use the entire electrical signal that is present. This “raw” electrical signal is filtered to eliminate a majority of noncardiac signals as well as portions of the cardiac signals that are not needed. Because filtering allows only certain frequencies to pass through to the sensing circuit, the final “filtered” signal may be substantially different from the original signal

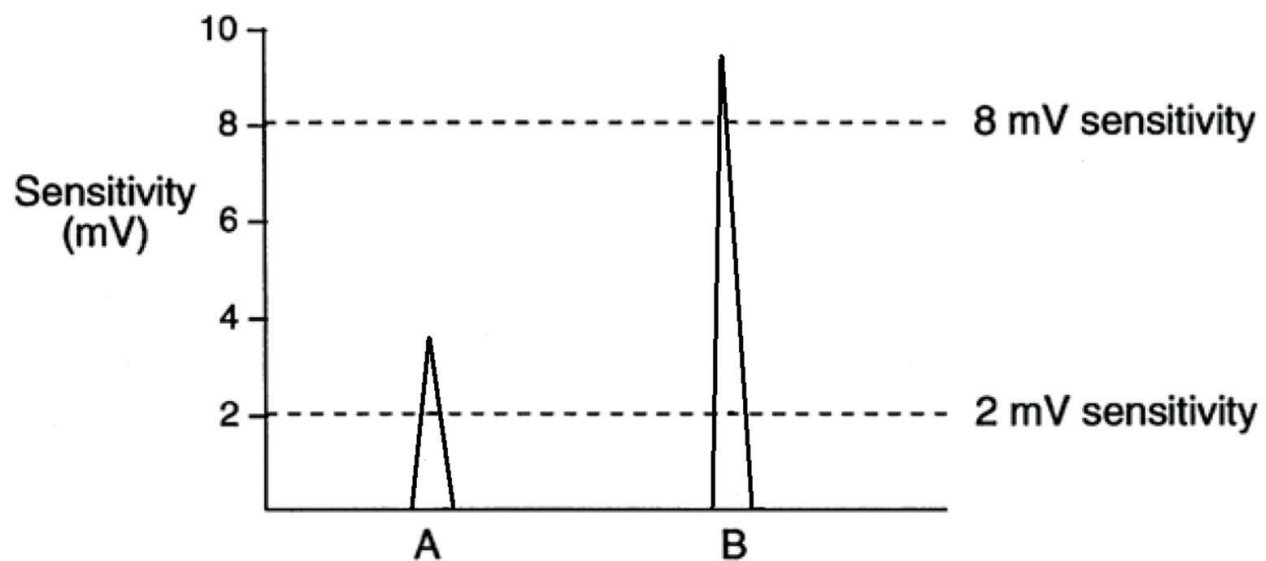


Figure 6. Concept of sensitivity. Electrogram A is 3 mV in size and electrogram B is 10 mV in size. At sensitivity of 2 mV, both electrical signals have sufficient amplitude to be sensed. At setting of 8 mV, only the larger signal will be sensed.

(**Figure 7**). One way of measuring the quality of a sensed signal is to look at the slew rate. The slew rate refers to the slope of the intrinsic signal (**Figure 8**) and is measured in volts/second. High slew rates (>1.0 V/s in the ventricle and >0.5 V/s in the atrium) are desirable for consistent sensing.



Figure 7. Raw and filtered electrograms sensed by a pacemaker. The top tracing is the filtered signal used by the pacemaker for sensing. Filtering of the raw signal is necessary to prevent (over)sensing of the T-wave, far-field signals, and myopotentials.

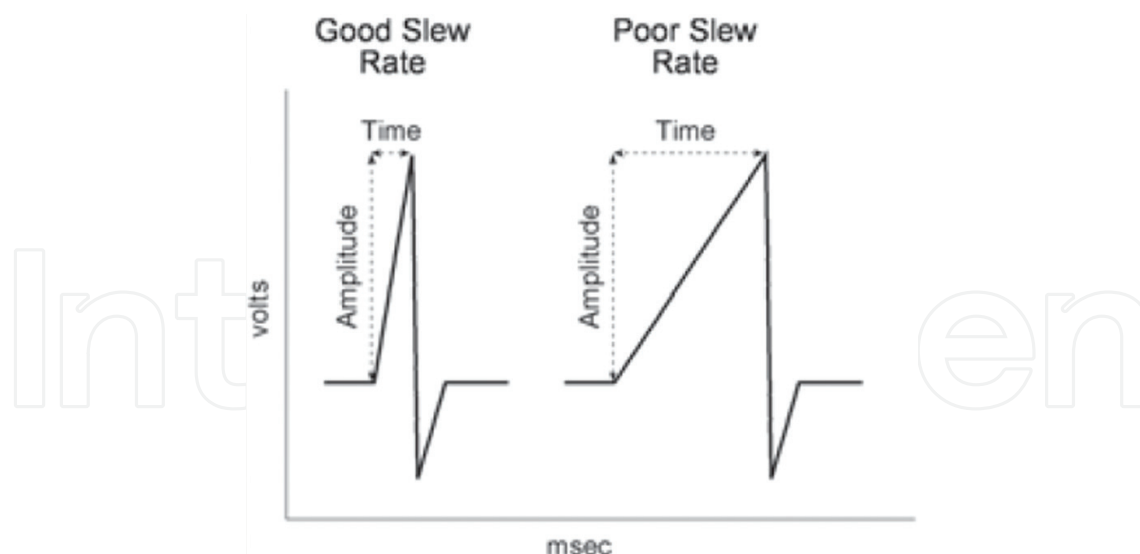


Figure 8. Slew rate measured in volt/second. The more rapid the voltage increase, the sharper the electrogram, the higher the slew rate.

1.7. Automaticity

As pacing systems become increasingly sophisticated, programming optimal pacing and sensing parameters for individual patients also becomes more difficult [4]. In addition, biological systems by nature are constantly changing, making settings that are appropriate at one point in time inappropriate at other times. Some pacemakers now have algorithms to automatically adjust one or more parameters. Automaticity is commonly applied to the rate response sensor function and sensitivity. There are different sensitivity adjusting algorithms for pacemakers and ICDs. For pacemakers, these algorithms assess the size of the sensed signal, and then attempt to provide a safety margin by adjusting the sensitivity. This tends to result in a less sensitive setting for the ventricle, as much of the time ventricular signals are very large. Lowering the susceptibility to EMI, it may in turn cause occasional undersensing of ectopic intrinsic beats. For a bipolar pacing system, the nominal (“out of the box”) sensitivity settings are usually acceptable and rarely result in under- or oversensing. Some pacemakers utilize an automatic gain feature similar to that of ICDs, which differs from the automatic adjustment feature currently in use in that the programmed sensing values remain unaffected (**Figure 9**).

Sensing the intrinsic heart rate is very important as this is the primary method for the ICD to detect the presence of a tachycardia. Typically, true bipolar and integrated bipolar configurations are used, while unipolar pacing and sensing have no role in ICD programming. True bipolar sensing in ICDs uses the same methodology as for conventional pacing leads. The dedicated pacing and sensing cathode and anode are located toward the distal tip of the lead within the ventricle (**Figure 10a**) and kept separate from the high-voltage shocking circuit. In an integrated bipolar configuration, the lead tip serves as the cathode and the distal shocking coil as the anode (**Figure 10b**) allowing for simpler lead design. However, since the shock coil doubles as the sensing coil, suboptimal sensing may result immediately after a shock has been delivered. Normal sensing resumes shortly after shock delivery once physiologic cardiac depolarization has returned. Some devices use true bipolar sensing and integrated bipolar pacing to overcome this limitation. The use of standard sensing protocols applying fixed

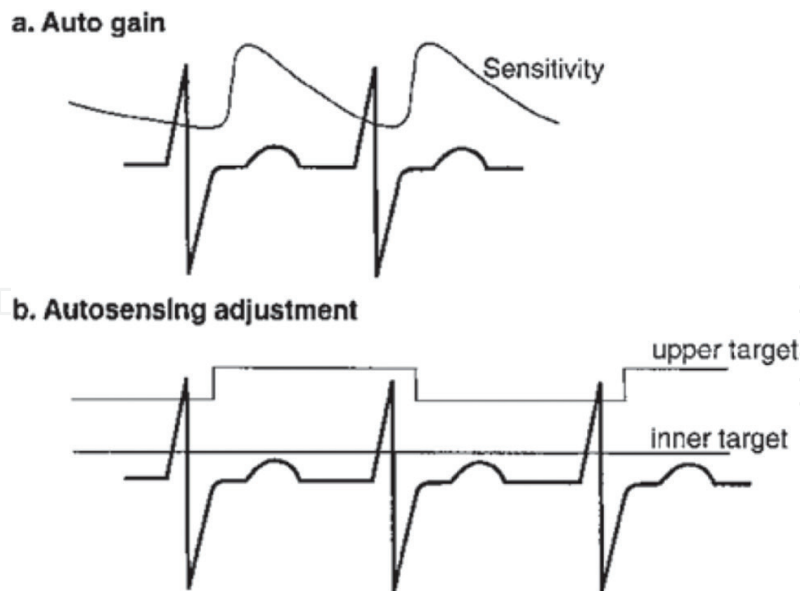


Figure 9. (a) Auto gain is a method to automate the sensing function. Sensitivity increases with the length of the sensing interval. Once a signal is sensed, the sensitivity abruptly decreases to avoid oversensing of the evoked response and the T-wave. (b) Autosensing adjustment is a method to automatically set the sensitivity. An inner and upper target is set for sensing. When a beat is sensed on both the inner and upper targets, the upper target is moved further out (made less sensitive) until sensing no longer occurs. The upper target is then moved back. In this way, the device can determine the signal amplitude and set the overall sensitivity of the device appropriately.

sensitivity values works well for pacemakers but not for ICDs because of the need to reliably sense and differentiate between varying clinically relevant rhythms such as sinus rhythm, premature ventricular (PVC) beats, and ventricular fibrillation where extreme variation in amplitude of intracardiac electrograms may occur. A fixed sensitivity level would be unable to adapt to these changes. Most ICDs use some variation of automatic sensitivity control, allowing for a “most sensitive value” to be programmed either as an absolute number or as a general term such as “least” or “most” sensitive. After a sensed event, the device reverts to higher sensing threshold in order to prevent T-wave oversensing or inadvertent oversensing of noncardiac

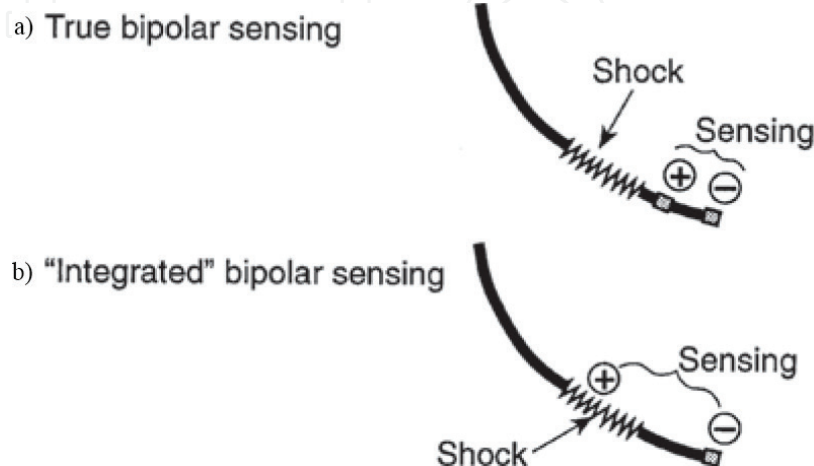


Figure 10. (a) True bipolar sensing occurs between the lead tip (cathode) and proximal ring (anode) independent of the defibrillation coil. (b) Integrated bipolar sensing utilizes the lead tip as the cathode and the defibrillation coil as the sensing anode. Sensing is more with a true bipolar configuration.

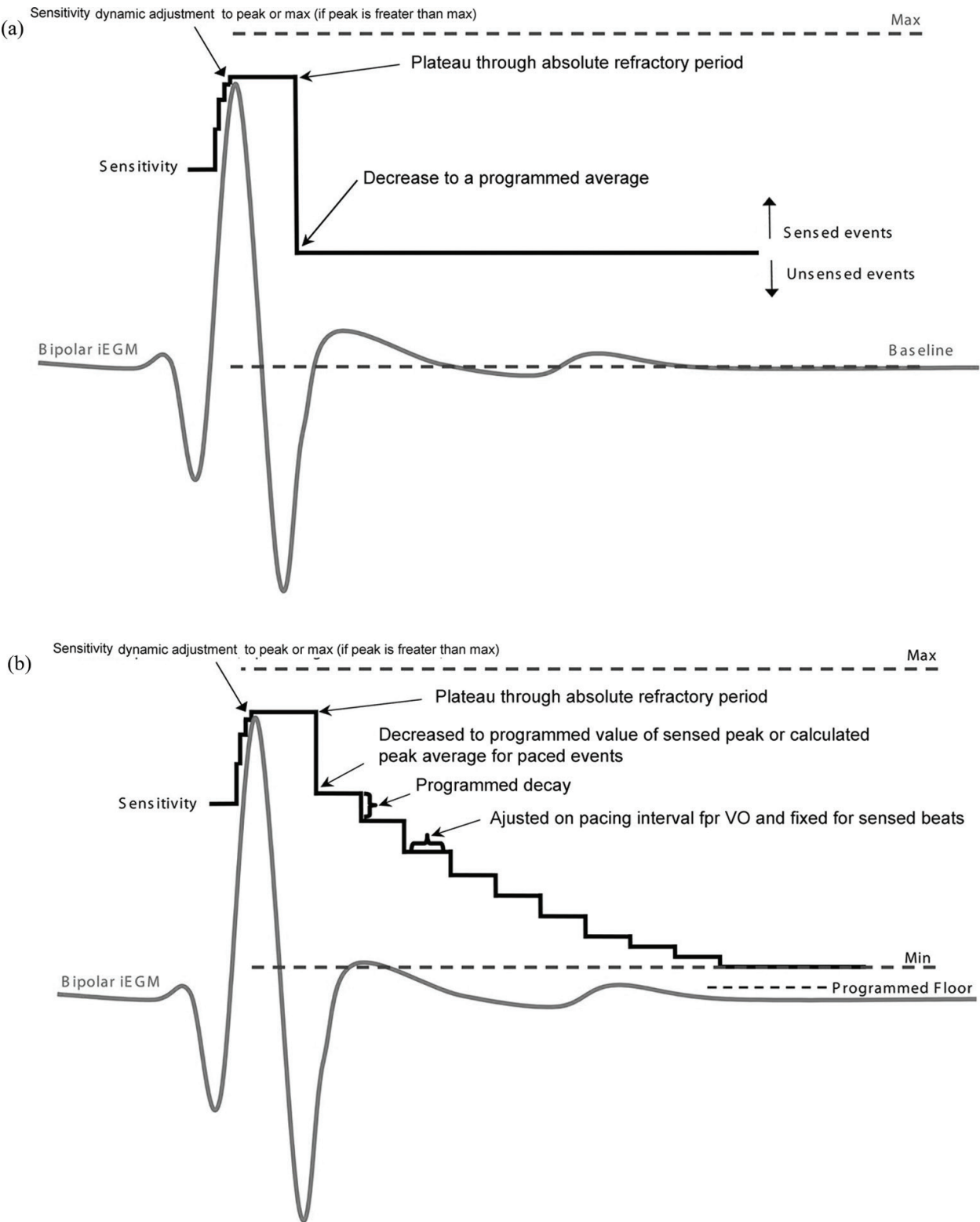


Figure 11. (a) A typical example of a bipolar right ventricular intracardiac electrogram from a pacemaker. The electrogram peak value is determined and the sensitivity is decreased to a fixed programmed value or a programmed average percentage of the peak value. (b) An example of a bipolar right ventricular intracardiac electrogram from a defibrillator. The electrogram peak value is determined and stored. Then a stepwise decay algorithm is used to the maximum programmed sensitivity. Sensitivity algorithms differ between device manufacturers.

events. The longer the period the device monitors for a sensed event, the more sensitive it becomes. This function provides the ability to determine if the patient has gone into fine ventricular fibrillation that might otherwise be missed if the device was set at less sensitive settings. **Figure 11** shows a comparison of the sensing algorithms between pacemakers and ICDs.

2. Device troubleshooting

2.1. Pacemaker troubleshooting

The first step in evaluating pacemaker malfunction is to determine whether the function of the device is truly abnormal [5]. If a pacemaker malfunction has been confirmed, the next step consists of a detailed patient history. The history may provide important clues to the likely diagnosis. If the problem or complaint occurs shortly after device implant, lead dislodgment, loose set screws, misalignment of the lead within the connector block or poor lead placement should be suspected as potential causes [2]. In the acute period, battery depletion or lead fracture is highly unlikely. Conversely, a patient presenting with an older device is more likely to experience lead failure or battery depletion rather than lead dislodgment or connection issues.

A tachycardia driven by the pacemaker presents a more difficult situation. In most cases, application of a magnet or reprogramming the device will terminate the abnormal rhythm. In rare cases, the pacemaker will not respond to these simple measures, and urgent surgical intervention may be required for “runaway pacemaker” (**Figure 12**). This uncommon malfunction is caused by a major component failure in the pacing circuit. The vast majority of rapid ventricular-paced rhythms in Dual pacing, Dual sensing, Dual action (DDD) or Ventricular pacing, Dual sensing, Dual action (VDD) devices are due to tracking of atrial fibrillation or atrial flutter. The pacemaker will attempt to track the rapid atrial rate to the programmed upper rate limit (URL) if mode switching is not enabled or fails to respond appropriately. Placing a magnet over the device will drop the pacing rate to the magnet rate of the device until a nontracking mode such as DDI or Ventricular pacing, Ventricular sensing, Inhibited (VVI) is programmed. Sensor-driven devices may cause rapid pacing as well. After the patient is stabilized, a history is obtained, and the initial device data collected, the 12-lead surface Electrocardiogram (ECG) should be evaluated. An approach to determine the general function of the pacing system is detailed below:

- i. Pacing
 - a. Spike present
 1. Verify appropriate rate interval
 2. Verify appropriate depolarization response
 - a. capture
 - b. pseudo-fusion
 - c. fusion

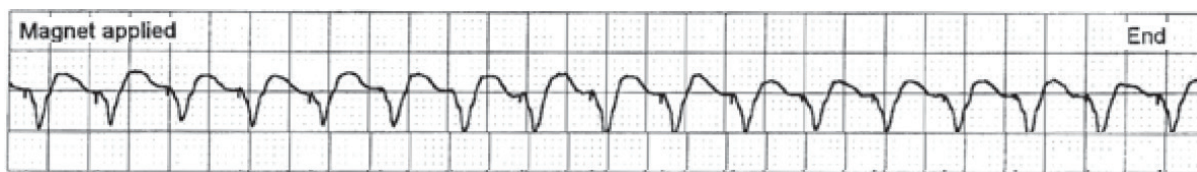


Figure 12. Runaway pacemaker: this strip shows VVI pacing at 180 bpm (the runaway protect limit on this device). The pacemaker was programmed to the DDD mode with an upper rate limit of 120 bpm. Therapeutic radiation delivered to the pacemaker in a patient with breast cancer resulted in circuit failure and rapid pacing. Even magnet application did not slow the pacing rate.

b. Spike absent

1. Apply magnet (magnet function must be enabled)

(Note: a ventricular pacemaker spike falling in the absolute refractory period of the myocardium will NOT result in capture.)

2. Observe for pace artifact and capture on 12-lead surface ECG.

ii. Sensing

- a. Patient must have a nonpaced rhythm

- b. Appropriate escape interval

- iii. Compare function to known technical information, watch for end of service indicators and other variations

Oversensing is readily diagnosed by placing a magnet over the device. If the pacemaker was not pacing or paced too slow due to oversensing, pacing will resume once the magnet is in place. If there is no pacing with the magnet on, then either the pacemaker is not putting out a pulse or the pulse is not reaching the heart.

Causes for true pacemaker failure are noted below:

- Depletion of the device battery [6]
- Defibrillation close to or on top of the device
- Device in the radiation field [7]
- Devices on recall or alert with known modes of failure [8]
- Electrocautery use close to or on the device
- Random component failure
- Direct mechanical trauma to the device

2.1.1. Noncapture

This potentially life-threatening problem is identified by the presence of pacemaker pulse artifact without capture in the appropriate chamber following the impulse. Causes of noncapture are listed below [2]:

- Exit block (high capture threshold) or inappropriate programming resulting in insufficient output or pulse width
- Malfunction or inappropriate programming of automatic capture output algorithms
- Lead dislodgment
- Lead fracture
- Lead insulation failure
- Loose lead-to-pacemaker connection
- Low battery output
- Severe metabolic imbalance
- Threshold rise due to drug effect
- “Pseudo-noncapture” (pacing during the myocardial refractory period due to undersensing of the preceding complex)

2.1.1.1. Corrective action

Increase pacemaker output if possible. Where appropriate, revise or replace lead or pacemaker, correct metabolic imbalances. For pseudo-noncapture adjust the sensitivity to a more sensitive setting, or revise the lead if sensing is very poor. Program to unipolar polarity.

2.1.2. Undersensing

Undersensing is recognized by the presence of a pulse artifact that occurs after an intrinsic event, but fails to reset the escape interval. The pacing output may or may not capture depending on the timing during the cardiac cycle. Causes of undersensing (thus “overpacing”) are listed below [9]:

- Poor lead position with poor R-wave or P-wave amplitude
- Lead dislodgment
- Lead fracture
- Lead insulation failure
- Lead perforation of the myocardium
- Severe metabolic disturbance
- Defibrillation near pacemaker
- Myocardial infarction of tissue near electrode
- Ectopic beats of low intracardiac amplitude

- Dual pacing, Ventricular sensing, Inhibited (DVI)-committed function
- Safety pacing
- Inappropriate programming
- Magnet application

2.1.2.1. *Corrective action*

Increase pacemaker sensitivity. Where appropriate, revise or replace the lead. Try reprogramming polarity. If the problem is very infrequent then careful observation may be acceptable.

2.1.3. *Oversensing*

A diagram highlighting the different components of a single-chamber pacemaker and ICD is shown in **Figure 13**. In a single-chamber system, oversensing is recognized by inappropriate inhibition of the pacemaker. Oversensing may result in total inhibition of output or prolongation of the escape interval. Myopotentials are a common cause of oversensing, which is seen predominately in unipolar pacemakers and usually results from sensing noncardiac muscle activity (e.g. pectoralis muscle or abdominal rectus muscles). Myopotentials are triggered by arm movements such as arm lifting in patients with prepectoral implants, or moving from a lying to sitting position in patients with abdominal implants. Oversensing may also occur if the ventricular lead falsely senses the T-wave. Despite an increase in environmental EMI, pacemakers prove fairly resistant to this type of interference because of continuous design improvements. Sensing intrinsic or extraneous EMI results in the device falsely detecting a cardiac event. The pacing output will be inhibited as long as these interfering signals continue. A dual-chamber system may track electrical signals such as myopotentials in either the atrium, ventricle or both. The atrial channel is usually set to a more sensitive value than the ventricular channel. Tracking may result from oversensing of electrical signals on the atrial channel, inhibiting atrial output, while these signals are too small to be sensed by the ventricular channel. Each time oversensing occurs on the atrial channel an atrioventricular interval (AVI) is triggered, resulting in ventricular output at a rate up to the programmed upper rate limit (URL). Causes of oversensing are listed below [9]:

- Myopotentials
- Electromagnetic interference
- T-wave sensing
- Far-field R-wave sensing (atrial lead)
- Lead insulation failure
- Lead dislodgement

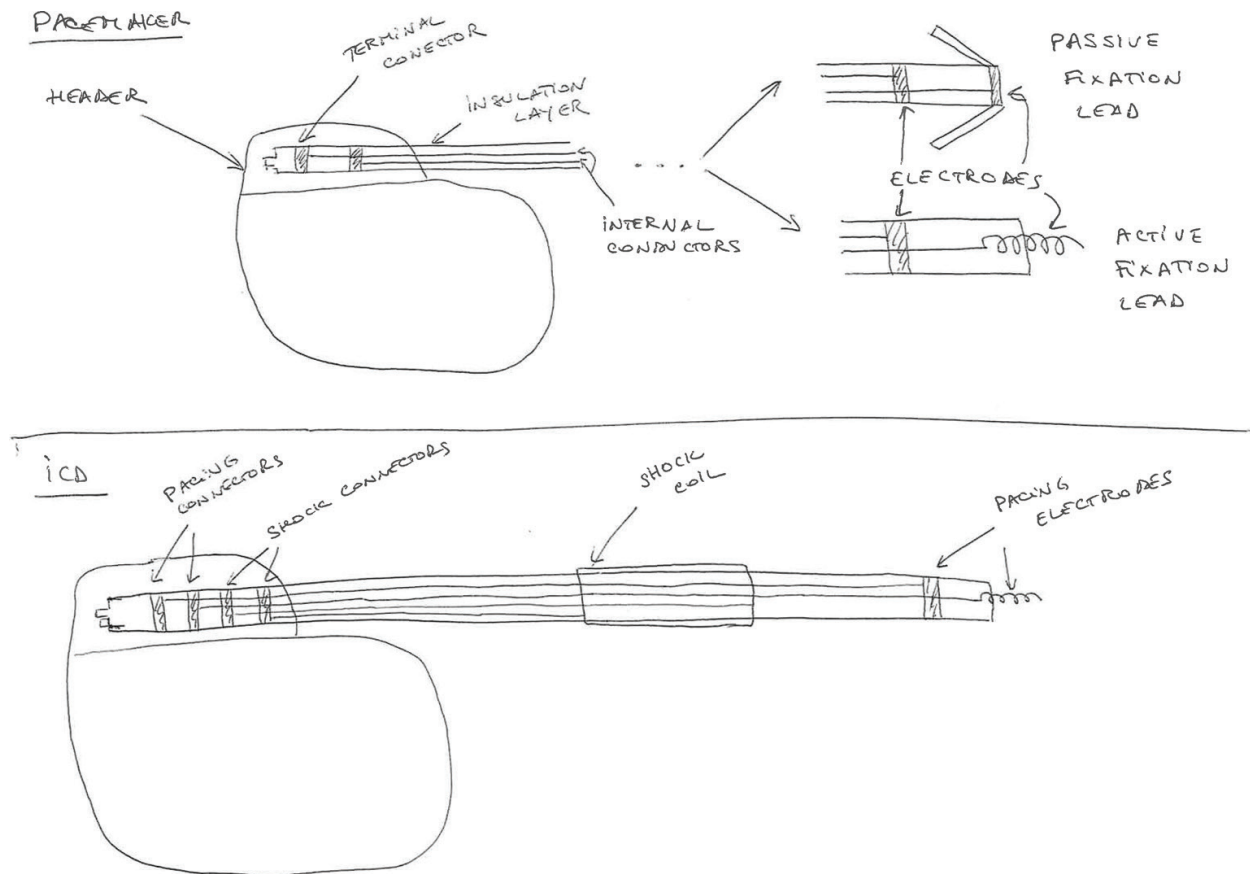


Figure 13. Basic components of a single-chamber pacemaker (top) and single-chamber ICD (bottom) (DF4 lead model shown).

- Lead fracture (**Image 1**)
- Loose fixation screw (**Image 2**)
- Crosstalk

2.1.3.1. Corrective action

Decrease the sensitivity of the device. For far-field or T-wave oversensing, prolongation of the refractory period will also correct the problem. The sensing polarity may be reprogrammed to bipolar if the option is available and the patient has a bipolar lead. In some instances, surgical intervention may be indicated to repair the lead, replace the lead or upgrade to a bipolar system. Loose set screw almost always needs corrective surgery as well as lead dislodgement. Lead fracture, if not complete, can be managed by changing the programming from a bipolar lead to a unipolar, using the functioning lead channel.

2.1.4. Diaphragmatic pacing and extracardiac stimulation

Diaphragmatic stimulation may result from inadvertent right phrenic nerve pacing by the right atrial lead or by direct stimulation of the diaphragm or chest wall muscle by the ventricular lead. Extracardiac stimulation occurs due to poor lead placement and/or high output pacing. Lead

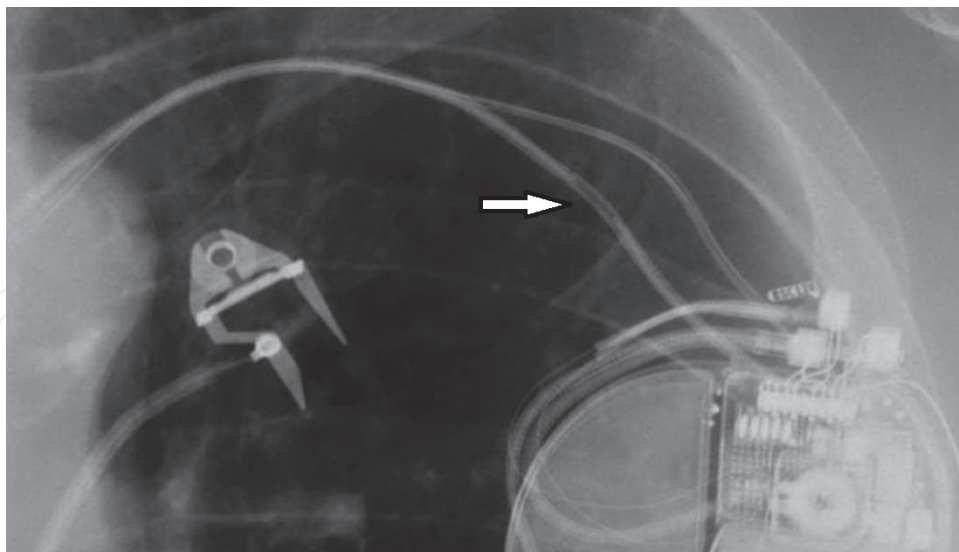


Image 1. Chest radiography demonstrating a lead fracture (arrow) resulting in high lead impedance.

perforation through the myocardial wall may cause extracardiac stimulation [10]. Unipolar pacemakers and leads with failed outer insulation may trigger stimulation of tissue adjacent to the site of the exposed conductor coil.

2.1.4.1. Corrective action

Decrease output if possible while maintaining an adequate safety margin for cardiac capture. Revision of the culprit lead may be required. Reprogram to bipolar polarity if unipolar.

2.1.5. Pacemaker syndrome

Pacemaker syndrome can occur in patients in sinus rhythm who receive a VVI pacing system or in patients with a dual-chamber device if the atrial lead fails to properly capture or sense

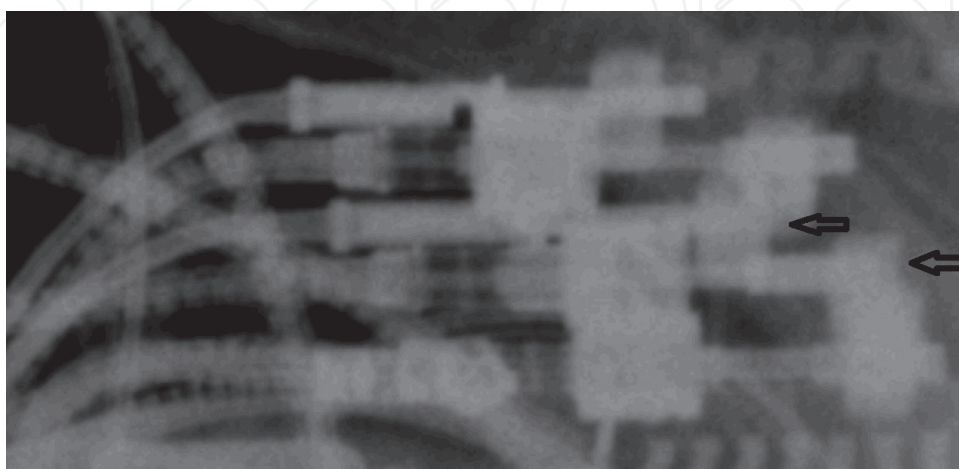


Image 2. Chest radiography showing two loose set screws (black arrows) resulting in lead noise and high impedance. After pocket revision and proper reconnection of both lead device and lead parameters normalized.

[11]. Ventricular pacing asynchronous to atrial contraction will limit the atrial contribution to ventricular filling. The resultant decline in cardiac output may cause patient fatigue and discomfort whenever the pacemaker is pacing. The classic example of pacemaker syndrome is caused by retrograde AV-nodal conduction. When the ventricle is paced and contracts, the depolarization impulse travels in a retrograde manner up the His bundle through the AV node towards the atrium. The atrium then contracts while the mitral and tricuspid valves are closed due to simultaneous ventricular contraction. The late atrial contraction causes blood to flow retrograde into the venous system resulting in “cannon A-waves,” dyspnea, hypotension, fatigue or even syncope. The surface ECG can give important clues to the correct diagnosis. In many cases, a retrograde inverted P-wave is seen embedded in the T-wave, as a sign of ineffective (as well as detrimental) atrial contraction. Patients with diastolic dysfunction, pericardial disease or loss of ventricular compliance due to hypertension, ischemic heart disease, ventricular hypertrophy or age are more likely to experience pacemaker syndrome.

2.1.5.1. Corrective action

For VVI devices, reduce the pacing rate or program a hysteresis rate to allow more time in sinus rhythm. If device reprogramming fails to resolve the problem, upgrade to a dual-chamber pacemaker is indicated. A malfunctioning atrial lead in a dual-chamber system may either be reprogrammed or require surgical correction.

2.1.6. Pacemaker-mediated tachycardia (PMT)

Pacemaker-mediated tachycardia (PMT), also referred to as endless-loop tachycardia or ELT, is an abnormal state caused by the presence of an accessory conducting pathway (the pacemaker) [12]. The mechanism of tachycardia is similar to that seen in patients presenting with the Wolff-Parkinson-White Syndrome. PMT often begins with a premature ventricular beat that is either spontaneous or pacemaker induced (**Figure 14**). The electrical impulse traverses retrograde through the His bundle and AV node to the atrium. The pacemaker will sense the retrograde P-wave if it falls outside of the postventricular atrial refractory period (PVARP). This will trigger an AVI after which the pacemaker will pace the ventricle. This cycle will repeat itself until one of the following occurs: (1) the retrograde P-wave blocks at the level of the AV node, (2) the retrograde P-wave falls within PVARP, (3) a magnet is applied to the pacemaker (disabling sensing) or (4) the device is reprogrammed to a longer PVARP or AV interval. The patient may use standard vagal maneuvers to induce transient AV block, thereby terminating the tachycardia. Though not commonly used for this purpose, adenosine (or any other AV-nodal blocking agent) may be given to terminate the tachycardia. PMT may initiate or restarted if a ventricular-sensed beat precedes an atrial beat. This includes a PVC, premature junctional beat, loss of atrial sensing or capture, and myopotential tracking or inhibition in the atrium. Appropriate programming of the PVARP will prevent PMT such that any retrograde P-wave will fall within this interval and therefore not be sensed by the atrial channel. However, some patients have markedly prolonged AV-nodal conduction and the long PVARP that is necessary to prevent PMT may severely limit the maximum tracking rate of the device due to the resulting long total atrial refractory period (TARP). Most modern pacemakers offer PMT prevention, yet still allow programming a short PVARP. One option automatically extends the PVARP for one cardiac cycle whenever a sensed R-wave is

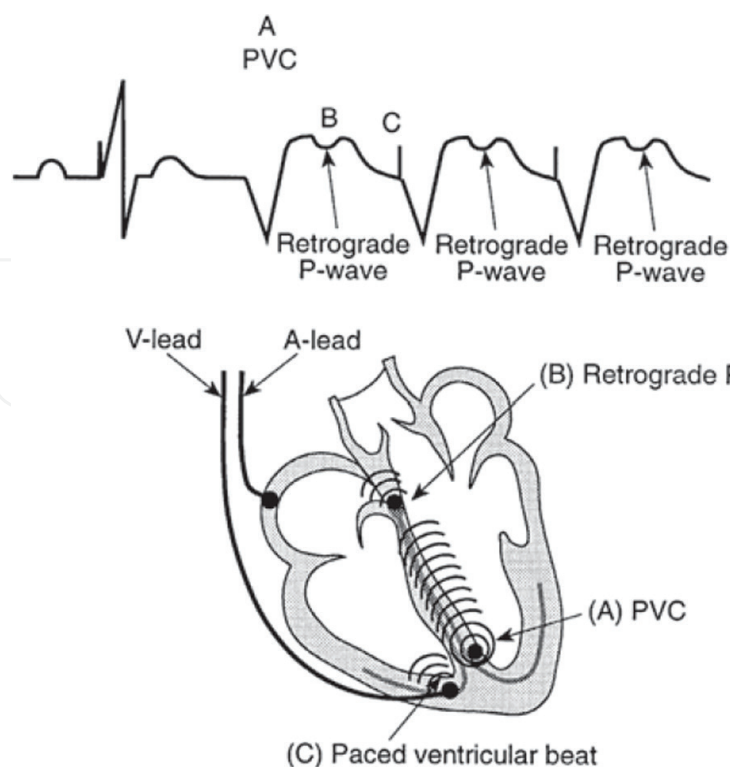


Figure 14. Pacemaker-mediated tachycardia (PMT): a PVC occurs (A) causing the ventricle to contract. The electrical impulse conducts retrograde through the AV node (B) resulting in atrial contraction. The retrograde P-wave is sensed by the pacemaker, which initiates an AV interval (AVI). At the end of the AVI, a pacing stimulus is delivered to the ventricle (C) and the cycle continues.

not preceded by a paced or sensed P-wave (assuming a PVC). An alternative method is to disable atrial sensing after a PVC is detected. This is also known as “DVI on PVC” since no atrial sensing takes place for one cardiac cycle. When first introduced by Pacesetter, it was known as “DDX” (some of these older devices are still in use today). The newest prevention algorithm will force an atrial output when sensing a PVC. Pacing the atrium at the time of a PVC will result in collision of anterograde and retrograde beats in the AV node, thus preventing the onset of PMT. Finally, most current devices provide an automatic termination algorithm if PMT is suspected. When the pacemaker reaches the upper rate (or a separately programmable PMT detection rate) for a specified number of beats, the PVARP is extended for a single cycle or alternatively a DVI cycle is introduced or atrial pacing is delivered, terminating PMT if present.

Figure 15 shows two different scenarios when atrial tachycardia (AT) can be misdiagnosed as pacemaker-mediated tachycardia (PMT).

1. An AT at a rate below the upper tracking limit is sensed on the atrial channel triggering an impulse on the ventricular channel.
2. Pacing in the atrial channel can suppress or terminate AT.
3. Prolonging the PVARP may lead to 2:1 atrioventricular conduction without interrupting the AT.

4. In true PMT, ventricular activation causes retrograde atrial activation which is sensed on the atrial channel triggering ventricular depolarization.
5. Pacing in the atrium during PMT interrupts the tachycardia but cannot differentiate PMT from AT (see [2])
6. Prolonging the PVARP will result in termination of PMT with resumption of sinus rhythm, while an underlying AT will conduct to the ventricle at exactly half the tachycardia rate.

In conclusion, prolonging the PVARP is a better method to differentiate AT from PMT and effectively terminate PMT.

2.1.7. Crosstalk

This is a potentially dangerous or lethal problem in patients who are pacemaker dependent [2]. Crosstalk occurs if the ventricular sensing amplifier misinterprets the atrial pacing impulse for an intrinsic ventricular beat. Ventricular output is inhibited and in patients without a ventricular escape rhythm, asystole will occur. On the surface, ECG crosstalk results in paced atrial P-waves without subsequent ventricular output. As a characteristic finding, the atrial pacing interval is equal to the atrial escape interval (AEI), rather than AVI and AEI combined. The shortened pacing interval results because the AVI is terminated prematurely due to the ventricular circuit falsely identifying the atrial pacing pulse for an intrinsic ventricular beat resetting the pacemaker to the next cycle. However, in a device using atrial-based timing, the AVI will be allowed to complete before the next AEI ensues, thus maintaining

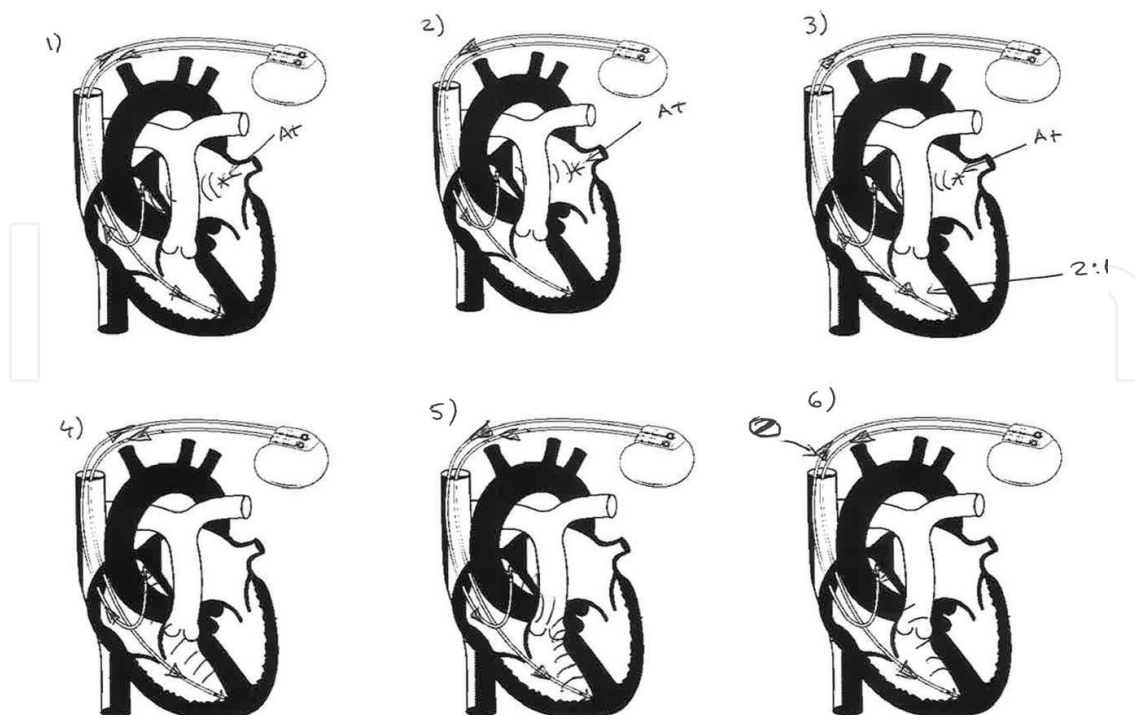


Figure 15. Differentiating atrial tachycardia (AT) from pacemaker-mediated tachycardia (PMT)—see the text for details.

the programmed pacing rate. Crosstalk is more likely to occur if high atrial output pacing is combined with very sensitive settings on the ventricular channel.

Most modern pacemakers are very resistant to crosstalk and certain features can prevent or reduce the effect of crosstalk. “safety pacing,” also known as “ventricular safety standby” or “nonphysiologic AV-delay” ensures a brief period of ventricular sensing during the early postatrial output period. This special sensing interval immediately following the ventricular blanking period is known as the “crosstalk-sensing window” (CTW). An event falling into the CTW may be the result of crosstalk or of true ventricular origin. If the ventricular lead senses an event during the CTW, a ventricular pacing output is committed at a short AV-delay (usually 100–120 ms), providing ventricular rate support should crosstalk be present. In the presence of a PVC or other intrinsic beat, use of a short AV-delay ensures that the ventricular output is not delivered during the relative refractory period (vulnerable period) of the T-wave (**Figure 11a**). While this feature will avoid the detrimental effects of crosstalk, the underlying cause needs to be identified and corrected as soon as possible.

Figure 16 represents an example of crosstalk: the atrial impulse delivered by the pacemaker is sensed on the ventricular channel resulting in inhibition of a ventricular output. In summary, the management of crosstalk includes:

1. Decreasing sensitivity of the ventricular channel
2. Decreasing output of the atrial channel
3. Activating ventricular safety pacing
4. Increasing the ventricular blanking period
5. Decreasing atrial pulse width
6. If the cause of crosstalk is insulation failure, implantation of a new atrial lead is warranted.

2.2. Defibrillator troubleshooting

Failure of the ICD to deliver a shock during ventricular tachycardia or ventricular fibrillation may result in presyncope, syncope or death. Conversely, inappropriate shock therapy causes patient discomfort, increases health care expenditure due to device clinic visits and/or hospitalization and heightens mortality [13]. Since all commercially available ICDs provide anti-bradycardia pacing their use is subject to the same potential problems as regular pacemakers. In addition, the dedicated bipolar ICD lead is used for tachycardia detection and treatment. In an “integrated bipolar” system, one of the shocking electrodes has the added function of a sensing and pacing anode. The ICD gathers information on the low-voltage impedance of the pacing system and the high-voltage impedance during shock delivery. The ability to evaluate the low- and high-voltage components separately can help the physician localize the site of lead failure. Adding a separate pacing/sensing lead may be a simple solution when only the low-voltage conductor is affected. However, failure of the high-voltage component often requires lead extraction and replacement. In certain circumstances, a second shocking coil may be added without removing the malfunctioning lead. The approach to the patient with suspected ICD malfunction is essentially the same

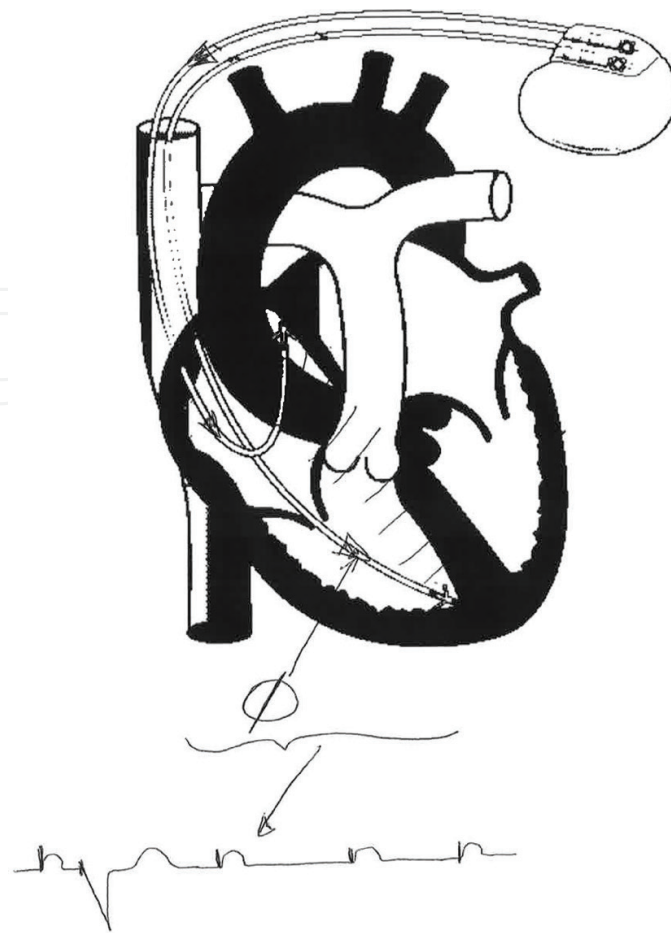


Figure 16. Crosstalk: atrial activation is sensed on the ventricular channel resulting in inhibition of ventricular pacing.

as the approach described for the pacemaker patient. Gathering patient data, understanding the indication for device implant, ICD interrogation, and evaluating the circumstances surrounding the incident in question are all essential. A common clinical scenario is the need to assess whether an ICD shock was appropriately delivered. For ICDs with limited diagnostic capability, elucidating the history surrounding the shock is crucial. The delivery of an appropriate ICD shock is often preceded by palpitations, lightheadedness, dyspnea or syncope. However, even in the absence of aforementioned symptoms, an appropriate ICD shock may have been delivered. Symptomatic hypotension may not ensue if the patient is in a sitting or supine position. Alternatively, the patient may simply not recollect the event due to insufficient brain perfusion or the patient was asleep at the time of the arrhythmic event. Indeed, nocturnal myoclonus is frequently misinterpreted by the patient's spouse as a device discharge. Inappropriate ICD shocks most commonly occur in the setting of AF. In the setting of AF with a ventricular response rate that exceeds the detection rate of the device, the ICD will charge and deliver one or repetitive shocks. Occasionally, the shock will convert AF to sinus rhythm. Dual-coil ICD leads with the proximal coil located within the right atrium are more likely to convert AF to sinus rhythm than single-coil leads. Importantly, these shocks are not the result of device malfunction, but rather due to an undesirable patient-device interaction. The specific categories of device malfunction are noted below.

2.2.1. Failure to shock or deliver anti-tachycardia pacing

Failure of the ICD to deliver anti-tachycardia therapy may be lethal. The reasons for failure to shock are listed as follows [14]:

1. Undersensing

- a. Lead malposition
- b. Lead dislodgment
- c. Lead perforation
- d. Lead fracture
- e. Lead insulation failure
- f. Lead-to-device connector problem
- g. Sensitivity set too low (i.e. insensitive)
- h. Poor electrogram amplitude due to change in myocardial substrate
- i. Myocardial infarction
- j. Drug therapy
- k. Metabolic imbalance
- l. "Fine" ventricular fibrillation

2. Primary circuit failure

3. Battery failure

4. Shock therapy turned off (by programming or magnet)

5. Magnet placed over the device

6. Strong magnetic field present

7. Detection rate set too high

8. Failure to meet additional detection criteria

- a. Rate stability
- b. Sudden onset
- c. Morphology criteria

9. Slowing of tachycardia below detection rate

- a. Substrate changes
- b. Metabolic changes

- c. Electrolyte changes
- d. Drug therapy changes

10. Interaction with permanent pacemaker

Lead failure or programming the rate detection zone too high is the most common reason for failure of the ICD to deliver therapy. The cause for lead failure may be identified on fluoroscopy. As older transvenous ICD leads are substantially thicker than conventional pacing leads, they are exposed to higher forces below the clavicle when using a subclavian vein access. Lead fracture typically affects one of the inner conductors of a coaxial or triaxial lead. Sometimes an intact outer conductor shielding a fractured inner conductor complicates proper diagnosis on fluoroscopy. Fractures can result in two broken ends remaining in intermittent contact. Several fluoroscopic projections may be required to visualize conductor failure and a slightly over-penetrated fluoroscopic image with settings similar to a dedicated thoracic spine view should be used. Fractures and insulation failures are more likely to occur after 1 or more years. If undersensing develops within 30 days of ICD implant, lead malposition, lead dislodgment or lead perforation need to be considered. Rarely, a loose connection between a connector pin and a connector block is the cause for ICD failure. Although ICDs are generally very reliable, a number of alerts have been reported for different models. Circuit failure, software lock-up, and other problems do occur infrequently and proper device interrogation will usually not be possible if any of these situations are present. In some cases, a “system reset” may be able to resolve the problem. In other cases, a software patch downloaded to the device will correct the problem.

Patient noncompliance with routine device clinic follow-up may result in ICD failure due to battery depletion. The ICD may become nonfunctional or lack sufficient power to charge the capacitors to the required voltage for discharge. Most ICDs restrict the time allowed for the capacitor to charge. Should the battery reserve be too low or the capacitor be defective (a common problem in earlier devices), the charge time may exceed the maximum time allowed and the ICD will not deliver a shock.

Occasionally, the rate detection zone is set too high. This may result from inappropriate programming or more commonly initiation of antiarrhythmic drug therapy such as amiodarone or sotalol. Antiarrhythmic drugs may cause slowing of the ventricular tachycardia cycle length below the programmed detection rate [15]. Significant metabolic or electrolyte abnormalities can affect the tachycardia cycle length, but may also alter the signal amplitude resulting in undersensing or failure to detect. Use of additional detection criteria to enhance specificity may delay or prevent appropriate ICD therapy and should be applied cautiously. Tissue injury due to myocardial infarction may lead to significant changes in the intracardiac electrogram and failure to sense.

Asynchronous pacing can be seen if bradycardia backup-pacing is turned on. In the past, many patients requiring pacing support underwent additional pacemaker implantation to prevent early ICD battery depletion from frequent pacing. This is usually of no clinical consequence

unless the ICD senses the pacing output delivered by the pacemaker. In a worst-case scenario, the pacemaker may misinterpret ventricular fibrillation for asystole and attempt to pace fibrillating myocardium. If the ICD were falsely interpret the pacing impulse from the pacemaker for a regular Waveforms of ventricular depolarization (QRS) complex, device therapy may be withheld indefinitely. For this reason, special care is exercised if a pacemaker patient undergoes additional ICD implantation or a dedicated pacemaker is indicated in an ICD patient. Be aware that, albeit less likely, oversensing of the atrial pacemaker impulse by the ICD may lead to similar grave consequences.

2.2.1.1. *Corrective action*

Defibrillator lead-related problems virtually always require surgical correction. Most physicians argue that lead failure requires lead removal due to the large size of the lead and potential interaction with a newly placed lead. A recently implanted ICD lead that has dislodged or demonstrated poor sensing performance may be repositioned if lead integrity can be verified. Immediate device replacement is indicated in the case of battery depletion or if a nonfunctional ICD fails software reset. Simple reprogramming of the ICD will resolve problems related to inappropriate tachycardia detection zones or if too many specificity criteria are applied to diagnose ventricular tachycardia causing delay or failure to deliver appropriate therapy. Interaction with a permanent pacemaker may be eliminated by reprogramming the pacemaker output and pulse width to lower values. Only a bipolar pacemaker should be implanted if an ICD is already present. Furthermore, the pacemaker should be a dedicated bipolar device or allow bipolar pacing as the “power-on-reset” polarity. The latter will prevent reset to unipolar polarity and guarantee pacing in the bipolar mode if power is temporarily interrupted. Since current ICDs integrate full-featured pacing capabilities, a separate pacemaker is rarely indicated. Noise due to lead fracture can cause oversensing with inhibition of output. Acute management includes changing to a unipolar configuration or sensing from a wider antenna, for example, lead tip to right ventricle (RV) coil, until the lead can be replaced.

2.2.2. *Failure to convert ventricular arrhythmia*

Despite proper detection and appropriate ICD therapy, some arrhythmic episodes may fail to convert to sinus rhythm with potentially lethal consequences for the patient. Below is a list of problems that may prevent restoration of sinus rhythm despite appropriate ICD therapy [15]:

- High defibrillation threshold
- Poor cardiac substrate (fibrosis, scar, etc.)
- Acute myocardial infarction
- Metabolic abnormality
- Electrolyte abnormality
- Drug therapy
- Drug proarrhythmia
- High-voltage lead fracture

- High-voltage lead insulation failure
- High-voltage lead migration
- Inappropriate device programming
- Low (inadequate) shock energy
- Ineffective polarity
- Sub-optimal “tilt”
- Ineffective pacing sequence
- Pacemaker polarity switch
- Atrial arrhythmias
- Sinus tachycardia
- “VT Storm”

Changes to the myocardial substrate following successful ICD implantation may result in delayed or unsuccessful antiarrhythmic therapy. Acute myocardial infarction, severe electrolyte or metabolic imbalance or initiation of antiarrhythmic drug therapy may increase the defibrillation threshold. Amiodarone is frequently utilized in patients presenting with life-threatening arrhythmias and may increase the defibrillation threshold. Some patients will require defibrillation threshold testing after amiodarone initiation to verify successful conversion with ICD shock delivery. Other drugs may act proarrhythmic to the effect that the arrhythmia fails to convert or resumes immediately after conversion. Lead fracture or insulation failure will reduce the actual amount of energy delivered to the heart and may impact the delivery of an effective ICD shock. Lead movement may alter the shock vector resulting in suboptimal current flow between anode and cathode.

Programming the shock energy below maximum output will conserve battery life, allow quicker shock delivery, and cause less pain to the patient. However, an insufficient safety margin between defibrillation threshold and applied energy reduces the probability of successful conversion. The shock duration (pulse width) is programmable on some devices and set automatically on others. If set too short or overly long, defibrillation will be unsuccessful. The optimal shock duration varies based on the resistance. The positive and negative phases of the shock wave may be programmable in duration and can significantly affect efficiency of therapy. Furthermore, anti-tachycardia pacing or low-energy shock delivery may accelerate ventricular tachycardia or cause degeneration into ventricular fibrillation.

2.2.2.1. Corrective action

Immediately correct reversible metabolic, drug or electrolyte abnormalities. Lead or device problems will often require surgical revision. Reprogram ICD to a different rate detection zone and/or reassess additional criteria applied for tachycardia recognition. Atrial arrhythmias may require drug therapy, catheter ablation to definitive treatment of the clinical arrhythmia or ablation of the AV node. Appropriate pacemaker selection and programming are mandatory if separate devices are used in the same patient. Strongly consider replacement for a single device.

2.2.3. Inappropriate ICD therapy

Inappropriate ICD shocks are far more common than failure to convert or failure to deliver therapy. Patients may think an ICD shock was delivered inappropriately, while thorough evaluation of telemetry data and stored electrograms confirms proper device therapy. If the ICD shock was determined inappropriate, the triggering event needs to be elucidated and corrected quickly. Repeat ICD shocks are poorly tolerated by the conscious patient because of pain encountered and fear of future episodes. The patient may voice anger and frustration or demand device removal. Although inappropriate shocks are less likely to result in patient death, immediate diagnosis and correction of the underlying cause are warranted. Causes for inappropriate ICD therapy are as follows [16]:

1. Oversensing

- a. Electromagnetic interference
- b. Interaction with another implanted device
- c. Lead fracture
- d. Lead insulation failure
- e. Loose connections
- f. Myopotentials
- g. T-Wave oversensing
- h. Pacing impulse from permanent pacemaker
- i. "Y" adapted biventricular adapters and connectors

2. Detection rate set too low

3. Supraventricular arrhythmias

- a. Paroxysmal supraventricular tachycardia
- b. Atrial fibrillation
- c. Atrial flutter
- d. Sinus tachycardia

Inappropriate shocks are most commonly encountered in the presence of atrial fibrillation. Many patients who undergo ICD implantation demonstrate enlarged hearts predisposing them to atrial tachyarrhythmias. Patients with a history of slow ventricular tachycardia may experience overlap with sinus tachycardia at the lower rate limit of the detection zone. This may occur during exercise, sexual intercourse or emotional stress and result in ICD shock.

Oversensing may lead to inappropriate detection as detailed above. Interactions may result from separate pacemaker and ICD implantation in the same patient. In the presence of a unipolar and some bipolar pacemakers, the ICD may sense the ventricular and/or atrial pacing spike resulting in double-counting of the ventricular rate during VVI pacing or triple-counting of the ventricular rate during DDD pacing. Double-sensing may also be seen with some biventricular devices if the right and left ventricles are wired into the same sensing circuit, for example, when using a “Y” adapter on the pacing lead to connect to a single ventricular connector on the device. It may also be the result of an older ICD design where, despite separate connectors available for the RV and left ventricle (LV) lead, the leads are interconnected within the device and run through a single pace/sense circuit. The net result of both of these configurations is the same, with the RV and LV lead being sensed on the same channel. Double-counting may occur due to the long conduction delay between RV and LV if the patient has a heart rate in excess of the URL, or one of the leads fails to capture.

2.2.3.1. Corrective action

The ICD detection rate should be increased if the sinus rate overlaps with the lower rate limit of the detection zone. Beta-blocker therapy should be initiated or uptitrated to reduce the sinus rate. Furthermore, additional discrimination criteria such as sudden onset, rate stability, and QRS morphology should be activated. Catheter ablation to treat the clinical atrial arrhythmia or ablation of the AV node may be an option in select patients. Interaction between pacemaker and ICD will require reprogramming to a lower output and pulse width, using bipolar polarity or upgrading to an integrated pacemaker and ICD system. The latter is often necessary if double-sensing occurs while using retained older leads or ICD connector designs. In some situations, the pacing lead may require repositioning. Lead failure and connection problems will often require urgent surgical correction. If EMI is detected, the patient should be advised to avoid the source of interference. For some patients, this may involve reassignment of duties at work or even a change in employment. Most ICD malfunctions and pseudo-malfunctions are readily diagnosed after obtaining a careful patient history, use of fluoroscopy, and device interrogation. Unnecessary replacement of the ICD will be avoided and patient safety and comfort assured if competent personnel addresses the device problem in a consistent manner.

3. Conclusion

In order to troubleshoot implantable cardiac devices, the clinician should have a thorough understanding of the underlying physics and signal processing techniques. Device implantation and follow-up requires knowledge of the most common causes for device malfunction. While device reprogramming may offer a permanent solution for some pacemaker or ICD malfunctions, others will require surgical correction as appropriate first-line therapy.

Author details

Sorin Lazar*, Henry Huang and Erik Wissner

*Address all correspondence to: slazar1@uic.edu

Division of Cardiology, Section of Cardiac Electrophysiology, University of Illinois at Chicago, Chicago, IL, USA

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