Chapter 16
Pacemaker Emergencies in the ER

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16.1 The Scope of the Problem

Pacemakers have won a preponderant role in today's cardiology and nowadays are used to treat a huge variety of conditions. For instance, 425 new pacemakers are implanted per 100,000 people every year in America [1]. In addition, in 2009, 737,840 pacemakers were implanted, and 264,824 were replaced worldwide. Specifically, most of them (225,567) were implanted in the United States, whereas demographically speaking, Germany had the greatest quantity of newly implanted pacemakers per million population (927). Additionally, the most common indications for pacemaker implantation are high-degree atrioventricular block and sick sinus syndrome. The most common pacing mode is VVI/VVIR, especially in developing countries [2, 3]. Furthermore, the majority of leads are transvenous and bipolar and have an active fixation [2]. All of this obligates every emergency room (ER) physician to know how to appropriately and efficiently treat a pacemaker emergency.

16.2 Prevalence

Overall issues associated with pacemakers have a prevalence ranging from <1% to 6% [4] or 3% to 7.5% [5]. Complications can be classified according to the time elapsed after the implantation in immediate (related to the procedure), intermediate, late, and in mechanical or electrical (Table 16.1).

Moreover, rates of up to 19.5% of right ventricular (RV) pacing-induced cardiomyopathy (≥10% decrease in LVEF with LVEF <50%) were related with frequent RV pacing in patients with preserved ejection fraction. Other risk factors for pacing-induced cardiomyopathy are male sex, wide native QRS duration, and frequent RV pacing (>20%) [4, 13].

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C. Jerjes-Sánchez, *Cardiology in the ER*,
https://doi.org/10.1007/978-3-030-13679-6_16

Table 16.1 Pacemaker-associated complications [4, 6–12]

Type	Time	Complications	Frequency
Mechanical	Immediate	Pneumothorax	0.9–1.2%
		Hemothorax	<1%
		Arterial puncture (could cause unnoticed placement of the lead in the arterial system)	2.7%
		An important pocket hematoma that requires intervention	3.5%
		Cardiac perforation (pericarditis and cardiac tamponade)	<1%
	Intermediate	Twiddler's syndrome	0.07% in 10 years
		Hypertrophic scar and keloid formation	NR
		Infection	0.13–19.9%
		Venous thrombosis and stenosis	1–3%
		Right-sided lead dislodgement	1.8%
		Left ventricular lead dislodgement	5.7%
		Mechanical lead complication	<1%
		Pocket pain or arm swelling	Infrequently reported
		Tricuspid valve and subvalvular apparatus injury	NR
	Late	Lead fracture	2.6–3.6%
		Infections (pocket, lead, and valve)	0.13–19.9%
Electrical	Intermediate	Runaway pacemaker event	2–4% with 30–40% mortality
		Failure to capture	NR
		Failure to pace	NR
		Failure to sense	NR
		Pacemaker-induced tachycardia	NR
	Late	Battery depletion	NR
		Left ventricular desynchrony	NR
		Failure to pace	NR
		Failure to sense	NR
		Pacemaker-induced tachycardia	NR
Mechanical and electrical	Intermediate	Significant TV insufficiency	10–39%

NR not reported

16.3 Pacemaker Functionality Aspects

Cardiac pacing has advanced a great deal since Elmqvist's and Senning's first totally implantable pacemaker in 1958 [14]. Basically, a pacemaker consists of a pulse generator and a lead or various leads implanted in the heart's chambers.

Table 16.2 Pacemaker code reaffirmed by the HRS in 2018

First letter Chamber paced	Second letter Chamber sensed	Third letter Response to a sensed event	Fourth letter Rate Modulation	Fifth letter Multisite pacing
A (atrium)	A (atrium)	I (inhibited)	R (yes)	A (atrium)
V (ventricle)	V (ventricle)	T (triggered)	O (no)	V (ventricle)
D (dual)	D (dual)	D (dual)		D (dual)
	O (none)	O (none)		O (none)

Dual: atrium + ventricle

Nowadays pacemakers are more complex, and a five-letter code, proposed by the North American Society of Pacing and Electrophysiology and the British Pacing and Electrophysiology Group and reaffirmed by the Heart Rhythm Society in 2018, is used to describe their function (Table 16.2) [15, 16].

The first letter makes allusion to the chamber paced (V for ventricle, A for atrium, and D for dual/both), the second letter refers to the chamber sensed (V for ventricle, A for atrium, D for dual/both, and O for none), the third letter indicates how the device responds to sensed stimuli (I for inhibit, T for trigger, D for dual/both, or O for nothing), the fourth letter indicates if rate response is on (R), and the fifth letter identifies if multisite pacing is used (none O, in atrium A, in ventricle V, or in both atrium and ventricle D) [4]. The most common use of the fifth letter is for biventricular pacing used for heart failure treatment [3].

Some common pacing modes are AAI/AAIR, VVI/VVIR, VDD, DDD, DDDR, and VOO/DOO [4, 17], which are hereby presented:

- AAI/AAIR: in this mode, pacing occurs in the atrium and is inhibited by a detected P wave (atrial event). It is used when the sinus node is dysfunctional, but the AV node conduction is conserved. The main advantage of this mode (when used with a single-chamber pacemaker) is that it avoids ventricular pacing and crossing the tricuspid valve. Rate response (AAIR) is added for patients with chronotropic incompetence.
- VVI/VVIR: this mode was devised to pace the ventricle in the absence of an intrinsic ventricular event or to inhibit in the presence of one (inhibition by the QRS complex). Moreover, this mode is employed in cases of chronic atrial fibrillation, infrequent pauses, or bradycardias [4]. This is explained by the fact that VVI/VVIR is unable to sense stimuli from the atrium. Rate response (VVIR) is used in patients with chronotropic incompetence. This pacing mode can be delivered by a single-chamber pacemaker with a lead in the ventricle.
- VDD: pacing can be delivered by a single lead that senses the atrium and the ventricle but only paces the ventricle. If an atrial event is detected, after a certain time interval, the ventricle is paced. On the other hand, if the intrinsic atrial impulse travels through the AV node normally or if there is an ectopic spontaneous ventricular complex resulting in a sensed ventricular event, the pacemaker is inhibited.

- DDD/DDDR: when the sinus node is functional, but the AV conduction is abnormal, a dual-chamber pacemaker may be the option. This pacing mode is able of pacing the atrium in case the frequency drops below a set value and is also capable of pacing the ventricle if the AV conduction is dysfunctional. Additionally, by sensing the atrium, the pacemaker turns the sinus node into a biosensor for increasing the heart rate when needed [17]. Moreover, rate response (DDDR) is used as an additional indicator of physical activity for increasing the heart rate.
- VOO/DOO: although only used temporarily, this mode is of great utility in certain situations. Specifically, asynchronous stimulation is employed when there is a risk of oversensing, which means that certain electromagnetic interfering signals (MRI or electrocautery, etc.) can be taken as intrinsic cardiac events. For instance, if one of these signals is detected in the atrium, the impulse could be carried to the ventricles, which may exceed the upper limit. Also, it is possible that the interfering signal is sensed in the ventricle as a native ventricular event and hence pacing would stop, leading to bradycardia or asystole in a pacemaker-dependent patient.

16.4 Most Common Indications to Implant a Pacemaker

The most common indications to implant a pacemaker, ICD, and CRT are summarized in the following table (Table 16.3).

16.5 Main Pacemaker Malfunctions/Abnormalities

Pacemaker malfunctions/abnormalities can be divided into mechanical or electrical complications:

- Mechanical complications
  - Lead damage

Table 16.3 Common indications to implant a pacemaker

Pacemaker	Third or advanced second-degree AV block
	Sinus node dysfunction
	Chronotropic incompetence
	Carotid sinus hypersensitivity
ICD	Primary or secondary prevention of sudden death because of malignant ventricular arrhythmias
CRT	To maintain AV and interventricular synchrony by biventricular stimulation for heart failure

ICD implantable cardioverter defibrillator, *CRT* cardiac resynchronization therapy

  - Infections
  - Thrombosis
  - Lead perforation
- Electrical complications
  - Failure to capture
  - Failure to pace
  - Failure to sense
  - Pacemaker-induced tachycardia
  - Runaway pacemaker syndrome
  - Battery depletion
  - Left ventricular dyssynchrony
  - Pacemaker syndrome
- Mechanical and electrical complications
  - Tricuspid regurgitation

16.6 Mechanical Complications

16.6.1 Lead Damage

Leads may experience fracture or twisting. In a few severe cases such as Twiddler's syndrome, Reel syndrome, or Ratchet mechanism, lead dislodgement may occur due to manipulation of the generator, causing it to twist inside its pocket [8, 18]. Additionally, lead's resistance is a variable factor dependent on body position or edema (to name a few), but a resistance change of >30% might imply a lead defect/damage [4]. Moreover, it is crucial to understand that the term "impedance" (measured in ohms Ω) refers to all the forces that oppose to the current flux in an electric circuit or pacemaker [19]. The normal impedance value of a lead typically ranges from 250 to 1200 Ω, with an output of 5 V [19]. In the one hand, an impedance value lower than 250 Ω suggests that the lead's insulation may be damaged (fewer forces opposing to the current flux). On the other hand, a high impedance along with a high myocardial depolarization threshold suggests a broken lead (stronger forces opposing the current flux) [19].

16.6.2 Infections

Infections are severe complications of cardiac implantable electronic devices (CIED). For instance, device-related endocarditis has an incidence of 10–23%, while infection of a pacemaker following implantation goes from 0.13% to 19.9%. Additionally, the incidence of ICD infection ranges from 0.7% to 1.2% [9].

Cardiac device infective endocarditis has a high mortality rate of 24.5–29% (with up to a year follow-up periods) and an 80–100% explantation rate [20]. Moreover, 68–93% of infections are caused by *Staphylococci* and Gram-positive bacteria, whereas less than 18% of infections are due to Gram-negative bacteria. The fact that 15% of implantable cardiac device bacteria are culture negative must be considered [20].

Most of the infections related to pacemakers occur in the implantation pocket [9]. Device infection may present a few weeks later (a most common scenario) or up to 1 year after the procedure [4]. As a result of infected leads, vegetations can appear through all the lead path, which includes the tricuspid valve, the endocardium of the right atrium, and less frequently the right ventricle [9]. Echocardiography is effective in visualizing and measuring vegetations along with evaluating the hemodynamic state of the heart. Transesophageal echocardiography must be performed in pacemaker bearers with suspected infective endocarditis [21].

Clinical presentation of systemic infections and endocarditis of the leads or valves commonly are fever, chills, positive blood cultures, and intracardiac vegetation. Pocket infection signs are swelling, redness, erosion, purulent discharge, chronic pocket pain, and alterations in the scar. Pocket fluid collection (visible with ultrasonography) and soft swelling may also present [22]. In this case, recommendations are to take a blood culture, to perform sensitivity testing (if possible), and to initiate broad-spectrum antibiotics with focus on cutaneous flora (most commonly *Staphylococcus aureus* or *Staphylococcus epidermidis*) such as vancomycin [1, 22, 23]. Needle aspiration or incision of the pocket should be avoided, and the patient must be referred to a center experienced in treating infected devices to program removal and/or antibiotic therapy [4].

In case empirical treatment needs to be initiated, a list of possible antibiotics is provided according to the "Guidelines for the diagnosis, prevention, and management of implantable cardiac electronic device infection" published on behalf of the British Society for Antimicrobial Chemotherapy (BSAC) as host organization [20]: (iv, intravenous; q, every)

- Generator pocket infection without further complications
  - Vancomycin (1 g BID iv) *or*
  - Daptomycin (4 mg/kg OD iv) *or*
  - Teicoplanin (6 mg/kg to a maximum of 1 g given at 0, 12, and 24 h and then OD)
- Lead-associated infective endocarditis or lead infection or complicated generator pocket infection with pending blood cultures, like in the scenario of severe sepsis
  - Vancomycin (1 g bid iv) AND meropenem (1 g tid iv) *or*
  - Daptomycin (8–10 mg/kg od iv) AND meropenem (1 g tid iv)
- Lead-associated infective endocarditis or lead infection or complicated generator pocket infection with negative blood cultures

– Vancomycin (1 g bid iv) AND gentamicin (1 mg/kg bid iv) *or*
– Daptomycin (8–10 mg/kg od iv) AND gentamicin (1 mg/kg od iv)

It is important to consider that doses need to be adjusted to the renal state of the patient. Moreover, daptomycin may be used to replace vancomycin in glycopeptide-intolerant patients or if nephrotoxicity is an issue. When selecting gentamicin, pre-dose levels must be <1 mg/L and post-dose levels 3–5 mg/L. Additionally, gentamicin may be replaced by meropenem.

16.6.3 Thrombosis

Venous thrombosis and stenosis are severe complications of pacemakers with an incidence of 1–3% [4]. Right atrial thrombosis is an uncommon pathology that can present asymptomatically or with signs of right-sided heart failure, obstruction, or pulmonary embolism [9]. Moreover, in 2 out of 53 autopsies performed in pacemaker bearers, a large right atrial thrombus was found. Both patients were older women and presented the thrombotic event approximately 1 month after device implantation and had signs of congestive heart failure and superior vena cava syndrome [9, 24, 25].

Echocardiography is an insightful tool for determining if the thrombus is recent or longstanding. According to Almomani et al., long-standing thrombi may contain calcium and most of the times are stationary. On the other hand, recent thrombi have a lower echo density and are highly mobile [9]. General signs for thrombosis are a pain, swelling, vein distention, and shortness of breath. As for standard venous thromboembolism, anticoagulants are the core of the treatment [1]. Finally, deciding whether to remove or change a lead or not is the responsibility of the implantation team, and it is not an emergency [1].

16.6.4 Lead Perforation

Perforation by a lead of a cardiac implantable device is an uncommon complication with an incidence of less than 1%. Moreover, perforation rates for pacemakers go from 0.1% to 0.8% and for implantable cardioverter defibrillators from 0.6% to 5.2%. This type of complication can be further divided into acute perforation, commonly resulting from the procedure, and subacute or delayed perforation, which takes place past the 1 month of implantation [9]. According to Hirschl et al., atrial perforation is more common than ventricular perforation, and ventricular perforation is more frequently caused by an implantable cardioverter defibrillator than by a pacemaker [26].

Apart from cardiac perforation, pleural perforation is also an acute complication of pacemaker implantation. Figure 16.1 depicts an anteroposterior chest X-ray of a pneumothorax case with subcutaneous emphysema after pacemaker implantation.

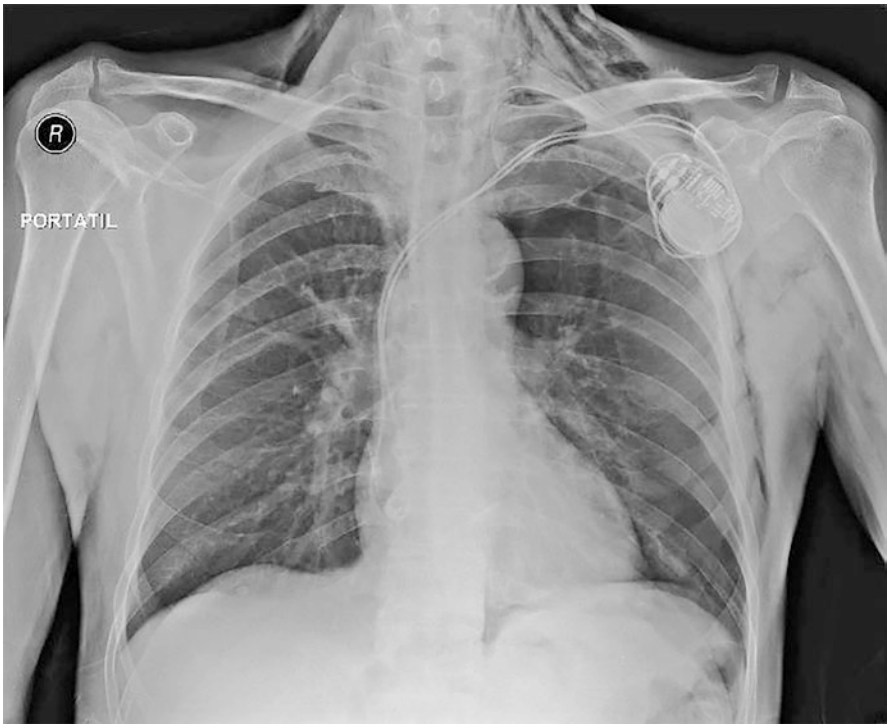

Fig. 16.1 Anteroposterior chest X-ray of a patient with pneumothorax and subcutaneous emphysema after pacemaker implantation

Almomani et al. conducted a review of 35 cases of delayed lead perforation reported in the literature in which his group concluded that the risk for cardiac tamponade and death is low [9]. Furthermore, Refaat et al. found that the symptoms accompanying a delayed perforation are variable, but some examples are syncope, chest pain, stimulation of extracardiac muscles such as the diaphragm, shortness of breath (possibly related to pneumothorax, hemothorax, hemopneumothorax, pneumomediastinum, and/or tamponade), chest discomfort (due to delayed pericarditis or mammary hematoma near the device pocket), hiccups caused by the stimulation of the phrenic nerve, swelling of the device pocket, and repetitive shocks due to a malfunctioning device. Moreover, patients may present unspecific symptoms such as dizziness or fatigue or be completely asymptomatic [27].

If the lead perforation is suspected, the following diagnostic sequence can be followed: device interrogation, chest radiography, echocardiography, and fluoroscopy [28]. Chest CT aids when other methods do not provide a clear diagnosis [9]. As such, myocardial perforation can sometimes be seen with a chest X-ray, and in much of the cases, it will show the lead's displacement to a different position from the one it was originally implanted (Fig. 16.2). Hence, when possible, it is important to compare the chest X-ray taken in the ER with a control one ideally taken within 24 h after the pacemaker implantation [29].

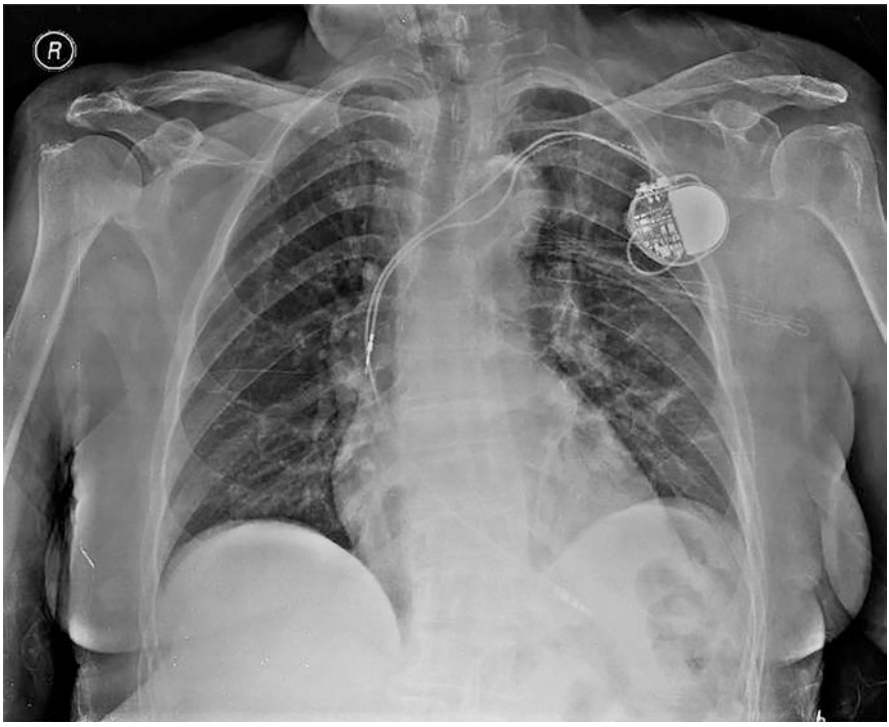

Fig. 16.2 Anteroposterior chest X-ray of a patient with dislodged and inactivated atrial lead

Two-dimensional transthoracic echocardiography is also of help to diagnose lead perforation or dislodgement, along with some accompanying pathologies such as pericardial effusion and tamponade. Since transthoracic echocardiography beam may not pass through the wire's path at first, it is important to keep in mind that multiple tomographic images should be taken to achieve a complete diagnosis [9]. Real-time 3D transthoracic echocardiography complements the 2D modality and is better and quicker to visualize the intracardiac part of the device's lead [9]. Thus, if available, real-time 3D transthoracic echocardiography should be used when lead perforation is suspected.

16.7 Electrical Complications

16.7.1 Failure to Capture

In this complication, the pacing spike is delivered, but the cardiac muscle does not depolarize. On the ECG this can be identified as pacing spikes with no atrial or ventricular complexes following [1]. Figure 16.3 depicts an example of a failure to capture on the

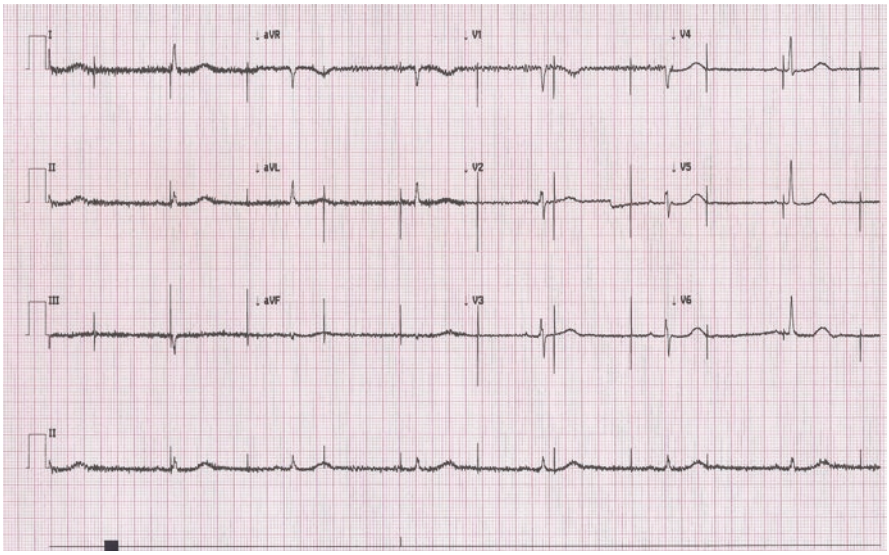

Fig. 16.3 ECG of a patient with a dislodged atrial lead (same case of Fig. 16.2) that depicts a failure to capture and to pace. On DII, pacing spikes 2, 4, 5, 7, and 9 fail to elicit a ventricular contraction. Additionally, pacing spikes 3, 6, and 8 are incorrectly delivered due to a failure to sense. ECG parameters: heart rate = 40 bpm, QRS complex = 94 ms, QT/QTc = 510/449 ms, average RR = 1485 ms, QTcB = 425 ms, QTcF = 454 ms, speed = 25 mm/s, voltage = 10 mm/mV, filter = 0.05–300 Hz W

ECG. Some common causes for this complication are lead dislodgement or malposition, inflammation of the electrode-myocardium interphase, and electrolyte imbalances. Imaging techniques, ranging from a chest X-ray or echocardiography to chest CT, are useful to determine the position of the lead. Symptoms of the disease by which a pacemaker was initially indicated can appear. Standard ACLS management is suggested, and a transcutaneous pacemaker should be considered on pacemaker-dependent patients [1].

16.7.2 Failure to Pace

Here, the pacemaker is sensing correctly but not delivering pacing spikes when needed. On the ECG, there will be no pacing spikes, and thus, the native rhythm of the patient will be observed. The most common causes are lead fracture, battery depletion, failure of the generator, and oversensing [1]. Oversensing refers to the event when the pacemaker is affected by electrical interference (muscular potentials or electrical noise) and incorrectly senses it as coming from the heart. This inhibits the delivery of stimuli.

Another important cause of oversensing is called pacemaker crosstalk. This phenomenon happens with dual-chamber devices when the lead in one chamber

delivers a pacing spike which is sensed by the lead on the second chamber as an intrinsic depolarization, therefore inhibiting the delivery of pacing spikes in the second chamber [30]. For example, the ventricular lead could sense an atrial depolarization spike as being ventricular in nature and inhibit ventricular pacing.

Causes of generator damage potentially leading to failure to pace are an internal malfunction, blunt trauma, MRI, radiation therapy, and use of electrocautery. Symptoms of pacing failure are frequently the same as those of the native pathology, such as bradycardia or high-degree atrioventricular block. Treatment consists of ACLS bradycardia management and interrogation and reprogramming of the pacemaker [1]. In the case of oversensing, switching the device into an asynchronous pacing mode (a constant frequency of 80–100 bpm) by placing a magnet over the pulse generator may help to avoid oversensing of the device (and therefore to avoid pacemaker inhibition). Extreme caution is advised in pacemaker-dependent patients [1].

16.7.3 Failure to Sense

In this malfunction, the pacemaker is not detecting the intrinsic chamber activity, and thus, regardless of the intrinsic beats, the device sends electrical impulses. Some frequent causes are lead dislodgement, lead fracture, scar tissue between the lead and myocardium interface, battery depletion, or low-amplitude cardiac signal [1]. The ECG will show inappropriately delivered pacing spikes (Fig. 16.3). Signs and symptoms of failure to sense will be those of congestive heart failure. Pacemaker under sensing must be considered when there is no obvious explanation for an exacerbation of congestive heart failure [1]. Interrogation of the device to obtain key functionality parameters is suggested alongside with pacemaker reprogramming.

16.7.4 Pacemaker-Induced Tachycardia

This complication occurs most commonly in old dual-chamber devices and is caused by atypical conduction through the heart [1]. Specifically, a retrograde P wave may initiate a reentry circuit by falling just after the preprogrammed refractory period. This will make the device deliver rapid ventricular stimuli as a result of the continuously sensed atrial impulses [1]. First-line intervention for pacemaker-induced tachycardia/runaway pacemaker syndrome is to apply a magnet since it could break the anomalous rhythm. When the above method fails in an unstable patient, possible management options are reprogramming the device or external pacing [1].

16.7.5 Runaway Pacemaker Syndrome

The present malfunction is intrinsic to the device and represents an infrequent but serious pacemaker complication with an estimated incidence of 2–4% with 30–40% mortality [7]. This malfunction also occurs with implantable defibrillators. Additionally, runaway pacemaker events have been reported to present in a wide time range, from 2 days to 9 years after implantation [7]. Nonetheless, runaway pacemaker events can occur throughout the entire lifetime of the device. They can also present intermittently and remain undetected [7].

Although some authors [1] treat runaway pacemaker syndrome and pacemaker-induced tachycardia as synonyms, they have certain specific differences and distinct treatment methods. Runaway pacemaker syndrome can present in two forms: pacemaker-induced ventricular tachycardia and extreme bradycardia as a result of ventricular capture failure (due to "rapid, low-amplitude sub-threshold pulses") [7]. In both forms mortality rates are high. Runaway pacemaker syndrome must be considered when pacing frequency exceeds the established upper limit, thus excluding pacemaker-mediated tachycardia [31].

The precise cause of runaway pacemaker is unknown, but it is associated with:

- Primary circuit failure
- Generator hermetic seal defects
- Circuit damage due to an electric scalpel or radio-frequency ablation
- Generator sterilization with heat
- Electromagnetic interference during radiotherapy [32]
- Low battery voltage [33]

It is important to mention that this syndrome is refractory to defibrillation therapy and antiarrhythmic agents since the problem is limited to the device. Poor results have been achieved by reprogramming the device or by overstimulation with a temporal electrode. Moreover, since magnet placement just disables the sensing feature of the device, this approach may be inefficient. Last treatment option is to disconnect the leads from the generator [7, 23].

16.7.6 Battery Depletion

Battery life is a variable parameter but a very important one for pacemaker-dependent patients. Hence, it is valuable to know some common clinical manifestations of a dying battery:

- Pacing mode change into an asynchronous one (VOO or AOO)
- Change on the width of the pacing spike
- Battery voltage or impedance change [19]

Two important terms to have in mind are ERI and BOL, which mean elective replacement indicator and beginning of life, respectively, and inform on the power left on the device's battery.

16.7.7 Left Ventricular Dyssynchrony

Right ventricular apical pacing is a risk factor for left ventricular dyssynchrony, which can lead to systolic and diastolic dysfunction, and ventricular remodeling. All of this is reflected clinically by worsening of heart failure. Furthermore, tissue Doppler and speckle tracking echocardiography are helpful to evaluate left ventricular dyssynchrony [9].

16.7.8 Pacemaker Syndrome

This pathology does not imply a malfunctioning pacemaker but rather a patient presenting unfavorable hemodynamics, namely, atrioventricular dissociation. This is common to see with VVI pacemakers since the synchrony between auricular and ventricular depolarization is lost. According to the Mode Selection Trial (MOST), 18.3% of the patients with sinus node dysfunction assigned to a VVIR pacing mode developed pacemaker syndrome [34]. Some of the most common symptoms presented are neurological of low cardiac output and of congestive heart failure such as general discomfort, fatigability, dyspnea, orthopnea, cough, dizziness, atypical chest discomfort, throat fullness sensation, and, less frequently, presyncope or syncope [3, 35]. Furthermore, patients may present hypotension, rales, jugular vein distention accompanied with cannon A waves, peripheral edema, and tricuspid or mitral (or both) regurgitation murmurs [3]. Lastly, when patients with a VVI pacemaker present pacemaker syndrome, a change to a dual-chamber device, such as DDD/DDDR, could be considered in some cases [3].

16.8 Mechanical and Electrical Complications

16.8.1 Tricuspid Regurgitation

Severe tricuspid regurgitation due to valve interference with an intracardiac device lead is an infrequent cause of progressive right-sided cardiac insufficiency and represented 2.8% of all the tricuspid valve surgeries [9]. Higher rates of tricuspid regurgitation were reported when more than 1 RV lead is implanted and with ICD leads because of their thickness and stiffness [12]. Tricuspid regurgitation can be functional or structural. When tricuspid regurgitation is associated with a pacemaker, the most common cause is functional (87%). On the other hand, when the regurgitation is directly induced by a pacemaker, the structural causes are divided as follows: restricted leaflet mobility (41%), adherent leaflet to the leads (37%), leaflet perforation (12%), scarring of leaflets (8%), and chordal entrapment (7%). The most commonly affected leaflet was the septal one (73%) [36].

Tricuspid valve regurgitation due to a pacemaker must be suspected in every patient with progressive right-sided cardiac insufficiency with early or late onset, without an apparent cause, and in cases that are refractory to habitual diuretic treat-

ment. Echocardiography is central to the diagnosis of tricuspid regurgitation, and both 2D and 3D modalities may be used. However, 3D echocardiography has better efficacy to evaluate the route of the lead through the tricuspid valve [9].

16.9 High-Clinical Suspicion in the ER

A pacemaker emergency must be suspected when a patient arrives at the ER with low- cardiac output symptoms (hypotension, syncope, lipothymia, dyspnea, fatigability, etc.). Additionally, lead perforation should be highly suspected in thin elderly females and in patients taking steroids or anticoagulants [27]. Moreover, device infection needs to be considered in light of *Staphylococcus aureus* bacteremia, since it is the most common infectious agent related to lead endocarditis and device pocket infection [9]. Furthermore, in a patient with an embolic event (especially pulmonary embolism) and a cardiac device, a right-sided origin of the thrombus must be suspected [21]. Pacemaker undersensing (failure to sense) must be suspected when there is no obvious explanation for congestive heart failure exacerbation [1].

16.10 Risk Factors

Although establishing clear risk factors is complicated, Refaat et al. [27] reported that patients with a lower body mass and elderly female patients were specifically vulnerable to lead perforation [9, 27]. Additionally, patients with a thin myocardial wall, possibly due to dilated myocardiopathy or a previous infarction, are also vulnerable to lead perforation. However, patients with a normal myocardium or a hypertrophic one are not considered to be at lower risk [27, 29]. Twiddler's syndrome is more common in female, elder, obese, and psychiatric patients [8]. Risk factors for pacemaker infection (pocket, endovascular leads, and valves) are diabetes, heart failure, renal failure, corticosteroid use, postoperative hematoma, lack of antibiotic prophylaxis, oral anticoagulation, previous cardiac device infection, generator change, and use of temporary pacemaker [4]. Finally, passive fixation leads and coronary sinus pacing leads (LV) have a higher risk of dislodgement [4].

16.11 Clinical Presentation

A typical patient with a malfunctioning pacemaker presents with bradycardia and/or hemodynamic instability due to abnormal stimulation. Additionally, the baseline rhythm of the patient (his indication for pacing) may manifest due to the malfunctioning device. Patients may also present tachycardia due to oversensing

(pacemaker-mediated tachycardia). In either case, low cardiac output symptoms are common. On the other hand, patients with pocket infections more commonly present local signs of erythema or edema. Finally, hemodynamic instability could also be due to severe cases of lead infection or thrombosis.

16.12 Main Clinical Characteristics

- Low cardiac output symptoms
  - Hypotension, dizziness, syncope, dyspnea, lipothymia, and fatigability
- Return to baseline rhythm before pacemaker implantation (bradycardia or advanced degree AV block)
- High pacing frequencies
- Shock or hemodynamic instability
- Suggestive signs of pocket infection such as erythema, edema, or tenderness to palpation

16.12.1 Physical Examination

Physical examination and device interrogation are the cornerstone to identify a pacemaker complication. When myocardial perforation is suspected, mammary hematoma, pericardial/pleural effusion, and chest wall bruising are key signs that may support the diagnosis [27]. Moreover, setting the device to a maximal stimulation output and hence the stimulation of the right or left hemidiaphragm or the chest wall indicate most of the times that a lead has perforated the atrial or ventricular wall. Additionally, interrogation of the device may show change in impedance, change in pacing parameters, loss of capture, elevated capture threshold, undersensing, and a noisy electrogram [27]. Nevertheless, normal parameters do not exclude lead perforation, and in case some of the above signs are found, image confirmation must be undertaken.

16.12.2 Clinical Stability

Some patients with a pacemaker complication may be asymptomatic, as in the case of lead perforations or right atrial thrombus discovered incidentally by chest CT or echocardiography, respectively [9, 26].

16.12.3 Clinical Instability

Since pacemakers are essentially antibradycardia devices, bradycardia or asystole in pacemaker-dependent patients may occur. Although some of the patients with right atrial thrombosis are asymptomatic, they can also present with symptoms of right-sided heart failure, obstruction, or embolization of the pulmonary artery [9]. Patients may present with septic shock in less than 10% of the cardiac device infection cases [20].

16.12.4 Chest X-ray

Chest X-ray is helpful in identifying twisted, fractured, or dislodged pacemaker cables (Fig. 16.3). It is also valuable to diagnose myocardial perforation by a pacemaker lead, since the migrated lead may be appreciated outside the heart. Furthermore, lead perforation must be suspected when the separation between the electrode tip and the epicardial fat is less than 3 mm [9]. In addition to posteroanterior chest radiography, a lateral projection is also of help to assess for the correct position of the device's leads [29].

16.12.5 Electrocardiogram

The electrocardiogram is an important part of the clinical assessment of a pacemaker. A functional pacemaker produces a spike or artifact on the surface ECG. Commonly, these spikes will anticipate atrial or ventricular depolarization [1]. These spikes are often difficult to appreciate, but setting the ECG filter to 150 or 300 Hz should make them more visible.

Most of the atrial leads are placed in the right atrial appendage, and thus P waves are normally positive on the inferior wall, DI, and AVL. An apical pacing lead will be seen as a left bundle block (QS or rS morphology in V1–V2 and wide QRS) since the depolarization stimulus travels from the RV to the LV. Moreover, the QRS complex will be discordant from the T wave [1]. On the other hand, a right bundle branch block suggests that the lead is in the left ventricle, which can result in thromboembolism or in ventricular arrhythmias. If this is discovered during the implantation procedure, leads must be repositioned. If this is detected after the implant, anticoagulation must be initiated, and a repositioning procedure must be planned [4].

Monophasic pacemakers (older devices) produce a clearly noticeable artifact on the ECG, while biphasic pacemakers (modern devices) produce a mostly indiscernible spike [1]. In the case of biphasic pacemakers, sometimes it is useful to increase the amplitude of the ECG to make the pacing spike noticeable [1]. Biphasic pacemakers (which can also act as monophasic) reduce the risk of over detecting muscular potentials, far-field detection, and stimulating the skeletal muscle [19]. In case

of lead perforation suspicion, right bundle branch morphology might be seen in V1, while the right ventricle is paced [27].

As it was previously stated, pacing leads are normally placed on the apex of the RV. Other implantation sites higher up in the septum are also possible, but the left bundle branch block morphology will persist. However, inferior ECG leads can have a variable axis [37]. The following table summarizes the most common electrocardiographic features found according to the lead implantation site as reported in [38] (Table 16.4).

Finally, a recently published algorithm called TBC helps to quickly assess for complications in the electrocardiograms of patients with pacemakers [39]. This method is easy to remember since each of its letters represents a sign of alarm:

- Tachycardia with spikes (T): spikes (pacing artifacts) stimulating at a frequency of 120 bpm or more (2.5 big squares [500 ms] or less after the previous QRS complex)
- Bradycardia without spikes (B): no QRS complex during a 1500 ms time period (7.5 big squares) after the previous QRS, which translates in a frequency of 40 bpm
- Chaos (C): spikes with no relation to the QRS complex (pacing artifacts within the QRS-T complex or not followed by a QRS and at different distances from the following QRS complex)

If the T criterion is found (most commonly produced by pacemaker-mediated tachycardia), elective referral to a specialist is recommended. On the other hand,

Table 16.4 Identification of lead position according to the electrocardiogram pattern

Lead position	Electrocardiographic features
RV apex (E.g. Fig. 16.4)	The impulse travels from right to left and from the apex to the base Left bundle branch block morphology VI: predominantly negative QRS of more than 120 ms Inferior leads (DII, DIII, aVF): negative QRS
A higher portion of the septum (E.g. Fig. 16.5)	The impulse travels from the right ventricle outflow tract to the inferior wall Inferior leads (DII, DIII, aVF): positive QRS Narrower QRS than with apical stimulation (leads on the higher portion of the septum are closer to the cardiac conduction system)
The lower portion of the septum (E.g. Fig. 16.6)	The impulse has two components: one travels from the inferior part of the septum to the right ventricle outflow tract and the other travels to the apex Inferior leads (DII, DIII, aVF): rS morphology R wave is proportional to the height at which the lead is implanted on the septum
Unnoticed placement of the lead in the LV	Right bundle branch block morphology
Biventricular stimulation (E.g. Fig. 16.7)	QRS has a combined morphology of the depolarization stimuli of both ventricles

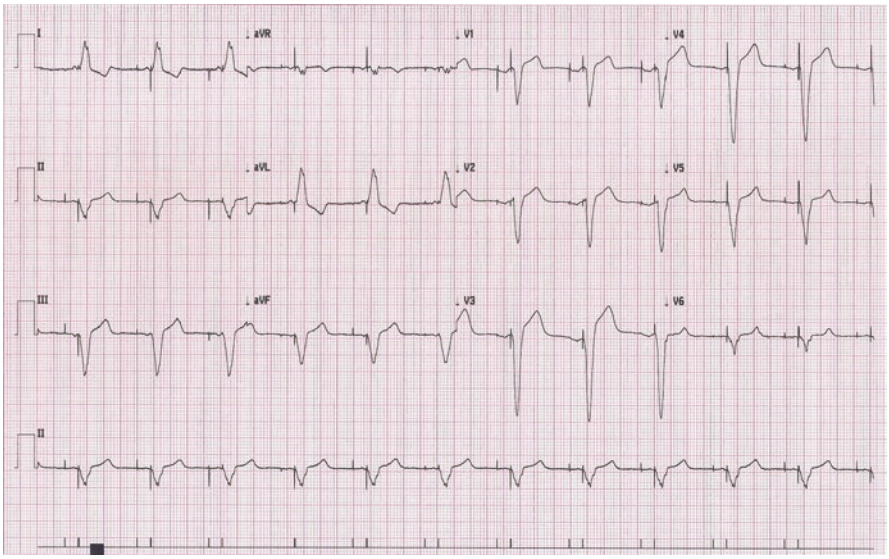

Fig. 16.4 RV apex

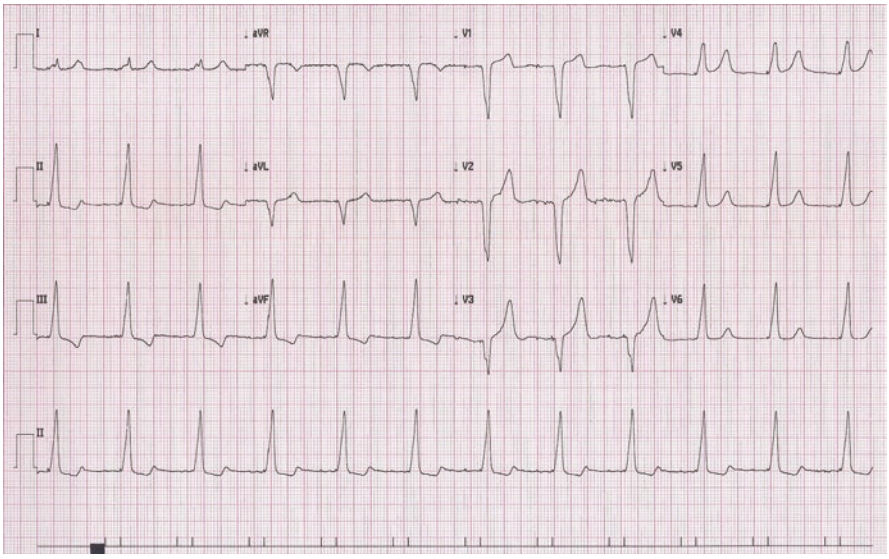

Fig. 16.5 Higher portion of the septum

both B and C require urgent pacemaker evaluation by a specialist and are indicative of severe malfunctions.

The sensitivity and specificity of this quick test are high, with 86.3% and 94.2%, respectively. Moreover, it has a positive predictive value of 88% and a negative predictive value of 93.3%, which means that if none of the above criteria are meet,

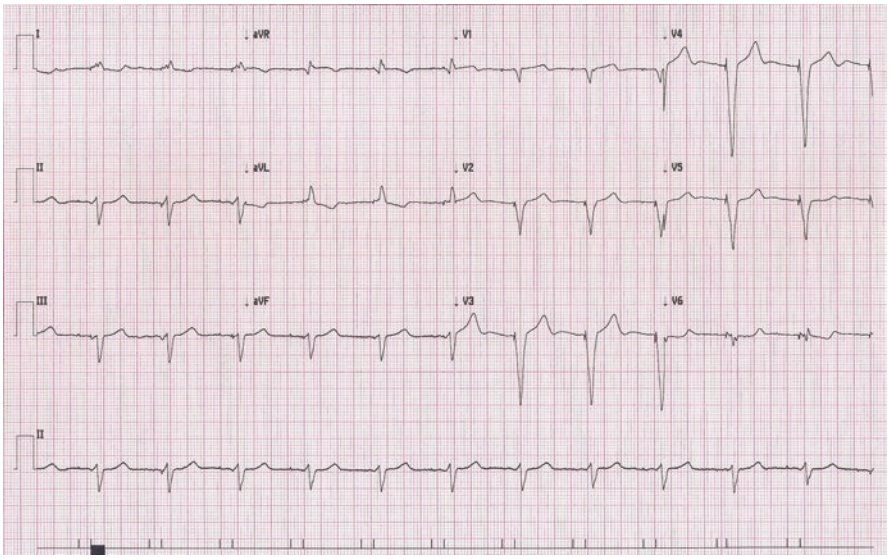

Fig. 16.6 Lower portion of the septum

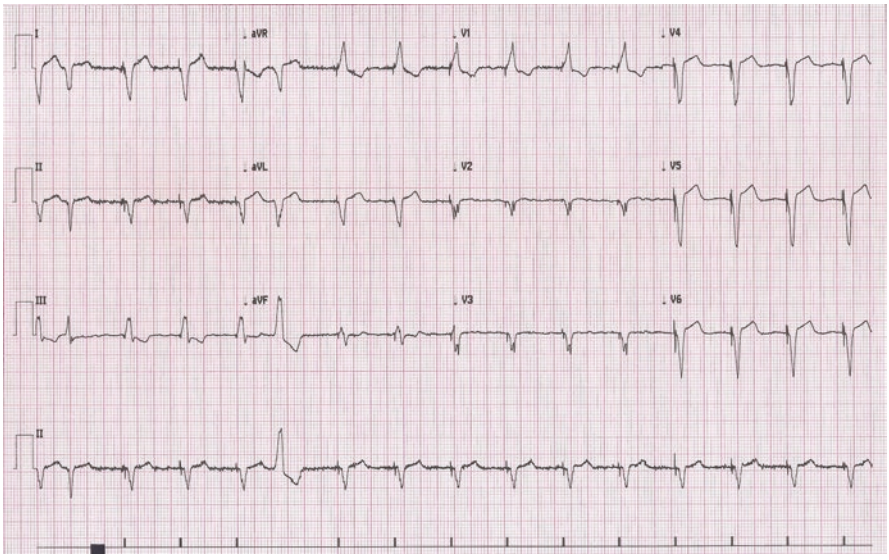

Fig. 16.7 Biventricular stimulation

the chances of finding an abnormal device are very low. Additionally, the algorithm improved the diagnostic and referral ability of non-cardiologist (including ER physicians) when dealing with patients with pacemakers. Unfortunately, atrial lead dysfunction, VOO programming, and advanced pacemaker functions are part of the limitations of this method [39].

16.12.6 *Transthoracic, Transesophageal, and 3D Echocardiography*

Echocardiography is a convenient diagnostic tool for detecting and, thus, properly treating pacemaker-related complications. Transthoracic echocardiography is useful to locate the path of pacemaker leads within the heart cavities (Figs. 16.8 and 16.9) and identify lead dislodgement, cavity perforation by lead, hemopericardium, or images suggesting a thrombus, but the diagnosis must be confirmed by other means such as transesophageal echocardiography, which is more sensible. Moreover, transesophageal echocardiography can be used to inspect for vegetations or masses with a sensibility of 92–96%, compared to a 22–30% of the transthoracic echocardiography [9]. Specifically, transesophageal echocardiography may be used when a thrombus on a pacemaker lead is suspected [40]. Real-time three-dimensional echocardiography, along with 2-dimensional echocardiography, is also helpful in the diagnosis of pacemaker complications, especially lead issues [9]. It is important to keep in mind that due to right ventricular pacing, patients may normally present paradoxical septal motion as a cause of the anticipated electrical activation of the right ventricle [9].

Transthoracic echocardiography may be limited as a result of a poor acoustic window and because of the presence of lead reverberation artifacts. Additionally,

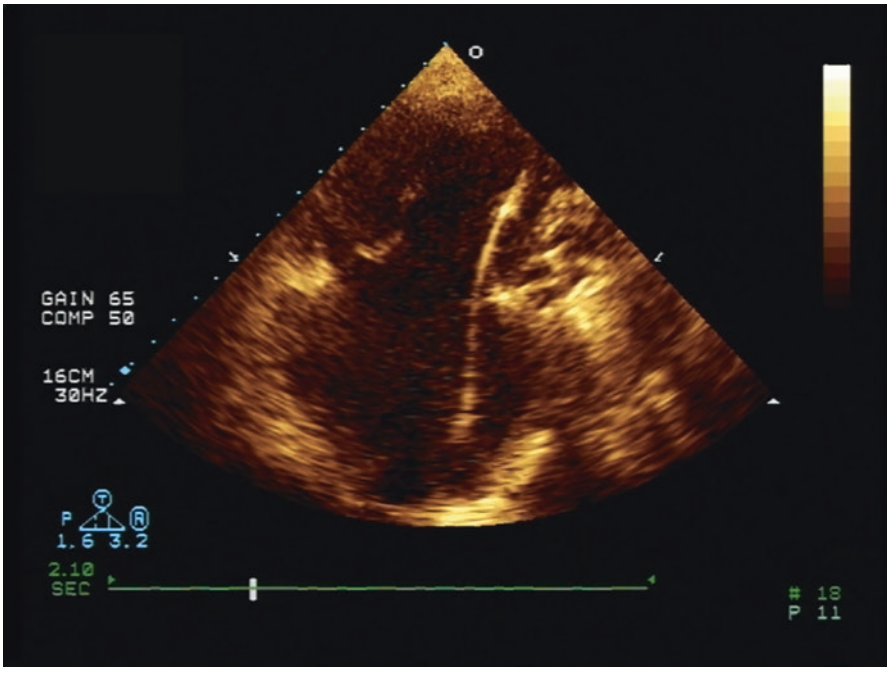

Fig. 16.8 Transthoracic echocardiography depicting a modified projection for RV which shows the complete lead path within right cavities in a patient with inactive rheumatic cardiopathy, mitral prosthetic mechanical valve, and total hip replacement

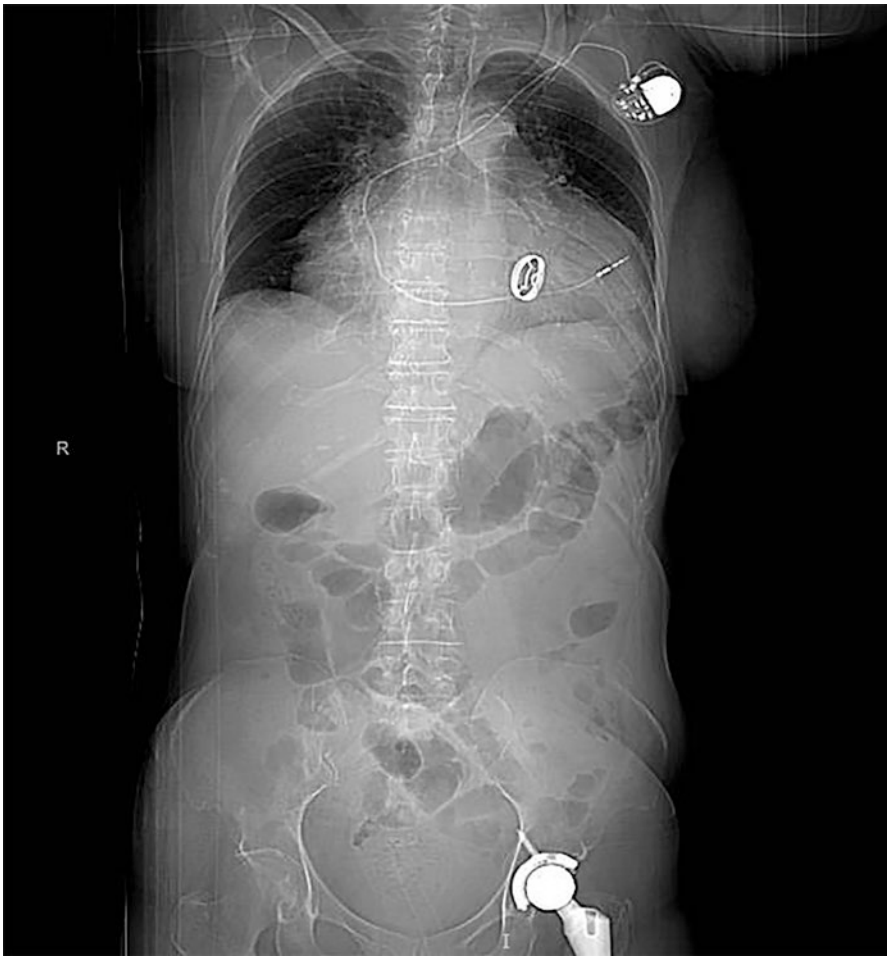

Fig. 16.9 CT scan of the patient described in Fig. 16.8 mitral prosthetic mechanical valve, total hip replacement, and a VVI pacemaker can be appreciated

sometimes it is difficult to distinguish between the lead tip, abnormal masses, or the tricuspid valve with a transthoracic echocardiogram due to poor echogenicity, limited window, or artifacts. On the contrary, transesophageal echocardiography is better to view the entire lead passage through the heart cavities. Furthermore, real-time transthoracic 3D echocardiography offers multiple views from a single acquisition and is helpful in the assessment of masses adhered to the leads [9].

Echocardiography is the preferred imaging technique to inspect masses on cardiac device leads since MRI is contraindicated in some types of pacemakers and CT is generally affected by metal artifacts. Vegetation usually looks as an oscillating intracardiac mass located on the pacemaker leads, valve leaflets, or endocardium [9]. Nevertheless, distinguishing between thrombus or vegetation as the origin of

the mass is complicated. Hence, echocardiography must always be complemented with clinical and laboratory evidence [9]. Finally, echocardiography is an operator-dependent study, and thus, having an echocardiography expert perform the studies in pacemaker patients could be an important factor to achieve a correct diagnosis.

16.12.7 Chest Cardiac Tomography (CT)

Chest CT is an important diagnostic tool for pacemaker complications. It is of special utility when lead perforation is suspected, and other diagnostic modalities were inconclusive. For instance, 15 of 100 completely asymptomatic patients with a cardiac device were incidentally diagnosed with subacute lead perforation when they underwent a CT whose primary clinical indication was other than lead perforation [26]. Leads create a star artifact when imaged with a CT, a common artifact caused by metal implants (Fig. 16.10). Commonly, the lead tip may be defined as the center of the star artifact [26].

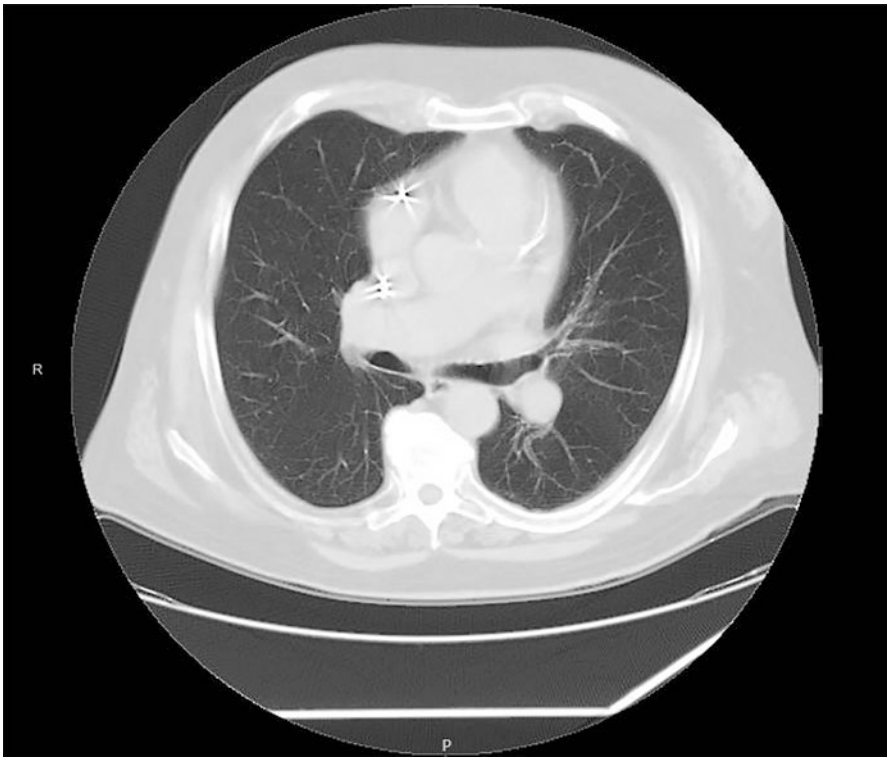

Fig. 16.10 CT scan is showing the star artifact caused by pacemaker leads

ECG synchronized chest CT can be used in the diastole phase to assess for myocardial lead perforation [29]. CT 3D reconstruction could also be performed and offers good visualization of the lead. Finally, chest CT is safe to use with cardiac device bearers with no serious or permanent complications reported [28].

16.13 Laboratory Evaluation

Laboratory test is of special utility since a failure to capture or undersensing may be due to electrolyte imbalances [30]. Moreover, blood and lead tip cultures may help to identify a pacemaker infection. Especially, *Staphylococcus aureus* bacteremia could be related to lead endocarditis or device pocket infection [9].

16.14 Multimodal Diagnosis Approach

Some pacemaker complications may be asymptomatic, but others generally present as palpitations, anxiety, lightheadedness, or as full cardiac arrest (Fig. 16.11). If a pacemaker abnormality is suspected, the patient must be connected to a cardiac monitor. Next, a 12-lead ECG (to evaluate cardiac rhythm and to look for electrical malfunctions) and a chest X-ray should be taken (to assess for mechanical problems such as a lead fracture or dislodgement). General laboratory tests are also suggested since the myocardial depolarization threshold could increase (leading to failure to capture) with electrolyte imbalances or ischemia [1].

16.15 Differential Diagnosis

Pacemaker complications are subject to be confused with a wide range of pathologies. For instance, paradoxical septal motion, which is a normal echocardiographic finding in some patients with right ventricular pacing, can also be observed in patients with RV volume/pressure overload or that have undergone cardiac surgery [9]. Additionally, observing noninfected strands adhered to the cardiac device leads is frequent. Those strands typically measure between 1 and 2 mm in width and 3 and 5 mm in length and are commonly localized in the right atrium [9]. Nevertheless, 6% of patients with an infection presented abnormal long filaments of more than 3 mm in width which were infected [9, 41]. Hence, clinical correlation is central to adequate differentiation between fibrin deposits, vegetations, or thrombi. Thus, it is vital to apply a multimodal diagnosis approach in which data from the image studies, the electrocardiogram, and the echocardiogram are fully integrated with the clinical history.

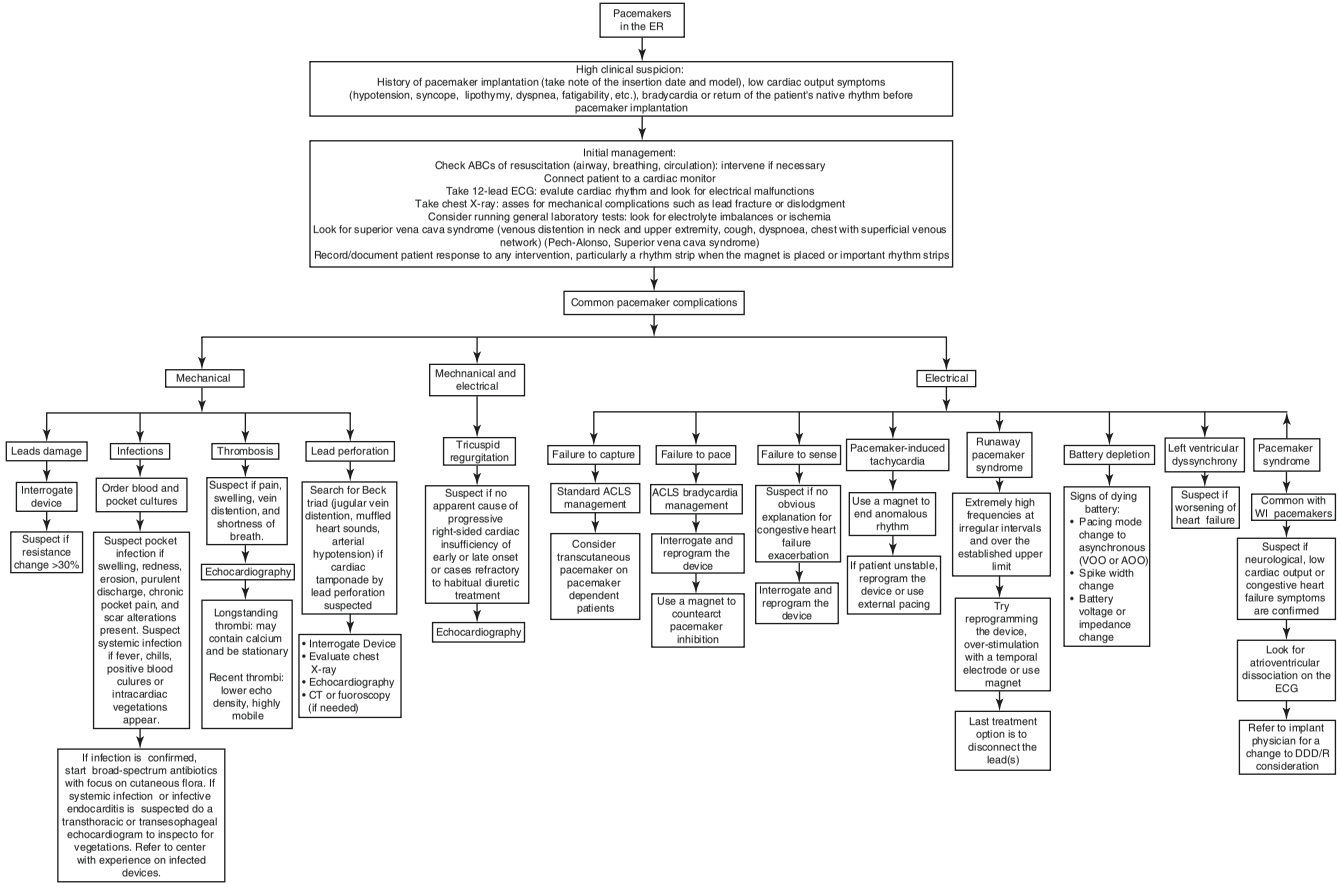

Fig. 16.11 Algorithm for pacemakers in the ER

16.16 Treatment

In case of a failure to capture, ACLS management is suggested in addition to a transcutaneous pacemaker, or a temporal venous pacemaker if available, in case of a pacemaker-dependent patient [1]. Moreover, in case of a failure to pace, ACLS bradycardia management is recommended. Then, the device must be interrogated and reprogrammed [1].

Transesophageal echocardiography is of great help when establishing treatment for device-related infections. Indeed, if the patient presents myocardial abscess or lead vegetation bigger than 5 cm, surgery may be preferred over percutaneous extraction [9]. Apart from device removal, antibiotic therapy must be started [9].

When lead perforation is confirmed, there is a vast set of possible treatments according to the characteristics of the perforation and the device. If the electrode tip is inside the mediastinum and no bleeding events are registered, then a second cable may be implanted without the retraction of the perforating lead [27]. Nevertheless, maintaining an inoperative lead must be weighed against the risk for further migration of the perforating lead. In the presence of a cardiac tamponade possibly caused by lead perforation, drainage of the pericardial effusion and conservative management are recommended [27]. Extraction must be performed in a patient with uncontrolled bleeding or evolving hematoma and lead migration outside of the pericardium with the risk of vascular, pulmonary, or adjacent structures injury [27]. When micro-perforation is suspected, indications for repositioning a lead are refractory pericarditis pain, persistent effusion, or pacemaker malfunction (pacing or sensing abnormalities) [4].

The method of choice to extract a perforating lead depends on the fixation system. If the lead has an active fixation system, transvenous extraction can be performed with a low complication risk according to some electrophysiologists [27]. The above procedure must be executed under TEE vigilance, general anesthesia, and if possible with excimer laser sheath [27]. Moreover, the procedure can be done both in the electrophysiology laboratory or in the operating room, but the cardiac surgery service must be present in case of an emergency [27]. On the other hand, if the electrode has a passive fixation system, two-stage cardiac surgery is preferred since this type of electrodes is thicker and has a higher chance of injuring tissue if retracted. Finally, the risk of bleeding or injuring nearby tissues during extraction is diminished by cutting the lead tip first [27].

16.17 Response to Magnet

The following table applies to most of the devices. For a specific list, please consult the references listed (Table 16.5).

In most of the devices, when the magnet is removed, the device will return to normal programmed function. However, reprogramming might be needed by some ICD models after being exposed to a magnet [1]. It is important to remember, that

Table 16.5 Expected CIED response to magnet application according to manufacturer [42–46]

Manufacturer	Device type	Response to magnet
Boston Scientific	Pacemaker and CRT-P	Asynchronous pacing at 100, 90, or 85 bpm (depending on the model)
		Asynchronous pacing at 85 bpm means that the device is near to the replacement date, contact the patient's device following physician
	ICD and CRT-D	Tachy therapy inhibited during magnet application
		Beeping tones produced one per second or R wave synchronous (depending on the model)
		No change to pacing therapy
	S-ICD	Tachy therapy inhibited during magnet application
		Beeping tone when the magnet is detected, then R wave synchronous beeping for 60 seconds, then beeping stops
Medtronic	IPG and CRT-P	Asynchronous pacing (DOO, VOO, or AOO) induced at 85 or 65 bpm (pacing rate may vary for some models or older devices)
		If device conditions are normal, the pacing rate will be 85 bpm If a recommended replacement time (RRT) or an electrical reset has occurred, the pacing rate will be 65 bpm
	ICD and CRT-D	Magnet application will not induce asynchronous pacing
		Magnet application can be used to check device status alerts
	ICD	If a programmed device condition (low battery voltage, lead impedance out of range, etc.) has occurred since the last time the device was interrogated, a tone will be emitted
		If the magnet is placed over the ICD for another time, the tone will be repeated
		Tachyarrhythmia detection and therapy operations are suspended while the magnet is placed
		Bradycardia pacing operations are not affected by the magnet
St. Jude Medical	Pacemaker	The device will pace asynchronously for the duration of the magnet placement (Magnet Mode parameter must be enabled)
		Devices at BOL pace at 100–98.6 bpm and at ERI at 85–86.3 (depending on the model)
		Dual-chamber mode devices (DDD, DDDR, DDI, DDIR) pace with an AV delay of 120 ms
		The device will go to a high output mode for the duration of the magnet placement if AutoCapture is enabled
		When the magnet is removed, AutoCapture will initiate a threshold search
	ICD	Tachyarrhythmia detection disabled during magnet placement
		Bradycardia pacing function is not affected

Table 16.5 (continued)

Manufacturer	Device type	Response to magnet
Biotronik	Pacemaker	Biotronik pacemakers have three different pacing modes induced by a magnet: asynchronous, synchronous, and auto (depends on manufacturer programming)
		Asynchronous mode at BOL paces at 90 bpm
		Asynchronous mode at ERI/EOL paces at 80 bpm
	ICD	Detection suspended
		No effect of a magnet on pacing
Sorin (ELA Medical)	Pacemaker	BOL asynchronous pacing at 96 bpm
		ERI asynchronous pacing at 80 bpm
	ICD	Detection and therapy suspended
		Magnet effect on pacing: pacing at 96 (BOL) or 80 (ERI) bpm

CIED cardiac implantable electronic device, *S-ICD* subcutaneous implantable cardioverter defibrillator, *ICD* implantable cardioverter defibrillator, *CRT-D* cardiac resynchronization therapy-defibrillators, *CRT-P* cardiac resynchronization therapy-pacemakers, *IPG* implantable pulse generator, *BOL* beginning of life, *ERI* elective replacement interval, *EOL* end of life

no matter the manufacturer, pacing behavior at or below EOL is unpredictable. Finally, physicians will be in warning when applying a magnet, and to ponder its usage against reprogramming the device instead, since asystole complication have been reported with it use. Consider the limited availability of technicians with the skills to reprogram the device and the time this process could take reprogramming the device (time is taken to reprogram and availability of technicians with such skills), since asystole cases have been reported [47, 48].

16.18 Electrosurgery

Electrosurgery alludes to the usage of electric scalpels during a surgical procedure. Its main risk with pacemakers is the production of electromagnetic interference potentially leading to pacing inhibition, rapid delivery of stimuli, tissue damage, or sudden change in pacing parameter (power-on reset). There are two modalities of electrosurgery monopolar and bipolar. In monopolar electrosurgery, the active electrode is included in the cautery pen, but a dispersive electrode needs to be placed on the patient. In the case of bipolar electrosurgery, both electrodes are built into the cautery pen, making the electric current to be localized. For this reason, in patients with pacemakers, bipolar electrosurgery should be used when possible. If the monopolar modality is selected, the current pathway between the active and return electrodes should avoid the generator (at least 6 inches away from the device) [49]. Additionally, cautery burst duration should be limited to 5 seconds with 5 seconds or more gap between bursts [4].

When the surgical site is below the umbilicus, and the dispersive path is placed on the lower limbs, there is no need to reprogram the device, except when several inhibition events are observed [4, 49]. In case the operation site is over the umbilicus, and

especially for pacemaker-dependent patients, the device must be interrogated before the procedure; pacing mode should be changed to asynchronous (DOO, AOO, VOO), either by reprogramming or by using a magnet; and at the end of the procedure, the device must be reprogrammed to its original parameters [4]. Finally, always have magnet ready to use during the procedure, especially if no device reprogramming is decided [49].

16.19 Magnetic Resonance Imaging (MRI): Yes or No?

MRI is a powerful diagnostic tool in clinical practice. It is estimated that half of the patients with a cardiac implantable electronic device will need an MRI scan once in their life [50]. MRI conditional systems include both a generator and leads that were specifically tested in combination. Thus, an MRI conditional generator with non-MRI conditional leads is not considered to be an MRI conditional system. Abandoned or fractured leads, epicardial leads, or components from multiple vendors make an MRI nonconditional system [50]. Especially, patients with epicardial leads should not be scanned with MRI.

The most frequent effect of MRI on pacemakers is an increase in the pacing capture threshold. Battery level could also result affected, and power-on reset events may occur [50, 51]. Apart from MRI, radiotherapy can also interfere with pacemakers, while high-dose radiation may create electrical currents in the semiconductor circuit of the device (Table 16.6) [28].

Table 16.6 Possible detrimental effects of MRI on cardiac implantable electronic devices according to HRS guidelines [50]

Effect	Comment
Force and torque induced by the magnetic field	Extremely unlikely since the generator is in a subcutaneous position and because leads contain a not significant amount of ferromagnetic material
Electrical current induced by gradient magnetic field	Might cause unintended myocardial capture or arrhythmias (atrial or ventricular)
Heating and tissue damage by radio-frequency fields	MRI nonconditional devices might heat and damage the adjunct tissue. Sensing or capture thresholds might change
Effects on reed switch activity	Reed switch activity on nonconditional devices might be affected with subsequent asynchronous pacing or inhibition of tachycardia therapies
Electrical reset	Electromagnetic interference cold cause power-on reset (backup mode) leading to inhibition of pacing, activation of tachyarrhythmia therapy, change to unipolar pacing, pacing below the threshold, battery status changes, and unreliable function
Inappropriate function and therapies	Could cause oversensing with the following consequences: asystole in pacemaker-dependent patients, inappropriate shocks in implantable cardioverter defibrillators, or programming changes

Note: Reed switch makes possible to program a device with the help of a magnet

It is important to know that MRI conditional generators have an MRI programming pathway that must be turned on before the scan and off after the scan. Scanning should be performed with the prerequisites specified for the device (**I A** HRS recommendation).

MRI conditional devices have an exempt period in which the conditionality does not apply (commonly 3 months after implantation). Despite the later, it is reasonable to perform an MRI scan during this period with a profound risk-benefit analysis (**IIa C-EO** HRS recommendation).

In the case of MRI nonconditional devices, risk-benefit must be thoroughly pondered. MRI scans are reasonable for patients with cardiac implantable electronic devices if the following criteria are meet, no fractured, epicardial, or abandoned leads, and MRI is superior to other testing modalities (**IIa B-NR** HRS recommendation). In such cases, pacemakers should be programmed to an asynchronous pacing mode, and tachyarrhythmia detection should be disabled on implantable cardioverter defibrillators (**I B-NR** HRS recommendation).

16.20 A Brief Comment on the Physical Bases of Pacemakers and MRI Compatibility

MRI has its theoretical basis on nuclear magnetic resonance (NMR) spectroscopy. This essentially consists of analyzing the radio-frequency energy absorbed and emitted by certain atomic nuclei placed in an artificial magnetic field. Hydrogen is the most commonly used atom for clinical purposes. Moreover, MRI is especially useful when imaging regions with a high quantity of water and fat since hydrogen atoms are densely present in those tissue components [50].

It is important to have in mind that MRI scan procedures require the use of the following fields: static magnetic, gradient magnetic, and radio frequency. All these fields might interfere negatively with susceptible electronic devices, including cardiac electronic implantable devices. For instance, the static magnetic field strength used by MRI scanners ranges from 0.2 to 9 Tesla, which could lead to mechanical injuries by moving objects if the appropriate security standards are not followed [50].

Apart from Tesla, gauss is an alternative unit for measuring the strength of magnetic fields [52]. To convert these units, the following formula is used:

$$1\,\text{Tesla} = 10{,}000\ \text{gauss} \tag{16.1}$$

The clinical importance of this formula resides in the fact that the "safe" magnetic field strength area is 5 gauss [50].

A final comment is to be made on the meaning of SAR, a concept commonly used when talking about the energy absorbed by a tissue due to exposure to

a radio-frequency field on MRI. Specifically, SAR is used to limit the energy delivered to a tissue to avoid thermic damage. The following formula is used to calculate SAR:

$$\mathrm{SAR} = \frac{\sigma |E|^2}{2\rho} \tag{16.2}$$

where E represents the peak electric field strength, σ the local tissue conductivity, and ρ the local tissue mass density [53]. Thus, the clinical significance of the formula is that SAR depends on both scanner parameters (electric field) and tissue factors (conductivity and mass density). As such, the effect of MRI scanning on patients with pacemakers is determined by the device, patient's tissue condition, and the pulse sequence used for the study.

16.21 Guideline Recommendations

A selection of guideline recommendations in relation to pacemaker emergencies is given in Table 16.7.

Table 16.7 Current international guideline recommendations

HRS CIED lead management and extraction [54]	COR	LOE
Drawing at least two sets of blood cultures before starting antibiotic therapy is recommended for all patients with suspected CIED infection to improve the precision and minimize the duration of antibiotic therapy	I	C-LD
Evaluation by physicians with specific expertise in CIED infection and lead extraction is recommended for patients with documented CIED infection	I	C-EO
TEE can be useful for patients with CIED pocket infection with and without positive blood cultures to evaluate the absence or size, character, and potential embolic risk of identified vegetations	IIa	B-NR
HRS MRI and radiation exposure in patients with CIEDs [50]	COR	LOE
MR conditional devices should be considered MR conditional only when the product labeling is adhered to, which includes programming the appropriate "MR mode" and scanning with the prerequisites specified for the device	I	A
It is reasonable for patients with an MR nonconditional CIED system to undergo MR imaging if there are no fractured, epicardial, or abandoned leads; the MRI is the best test for the condition, and there are an institutional protocol and a designated responsible MR physician and CIED physician	IIa	B-NR
It is recommended that for the patient with an MR nonconditional CIED who is pacing-dependent to program their device to an asynchronous pacing mode with deactivation of advanced or adaptive features during the MRI examination, and the pacing rate should be selected to avoid competitive pacing	I	B-NR

Table 16.7 (continued)

HRS CIED lead management and extraction [54]	COR	LOE
All tachyarrhythmia detections for patients with an ICD should be disabled prior to MRI	I	B-NR
For a patient with an MR nonconditional CIED who is not pacing-dependent, it is reasonable to program their device to either a nonspacing mode (OVO/ODO) or to an inhibited mode (DDI/VVI), with deactivation of advanced or adaptive features during the MRI examination	IIa	B-NR
It is reasonable to program patients with an MR nonconditional CRT device who are not pacing-dependent to an asynchronous pacing mode (VOO/DOO) with deactivation of advanced or adaptive features during the MRI examination and with a pacing rate that avoids competitive pacing	IIa	C-EO
It is recommended that patients with a CIED undergo clinical diagnostic CT without any additional device interrogation, programming, or monitoring	I	B-NR
ESC Guidelines on cardiac pacing and cardiac resynchronization therapy: magnetic resonance in patients with implanted cardiac devices [55]	COR	LOE
Conventional cardiac devices: in patients with conventional cardiac devices, MR at 1.5 T can be performed with a low risk of complications if appropriate precautions are taken	IIb	B
MR-conditional PM systems: in patients with MR-conditional PM systems, MR at 1.5 T can be done safely following manufacturer instructions	IIa	B
BSAC implantable cardiac electronic device infection [20]	COR	LOE
A chest X-ray should be carried out in all patients with suspected ICED infection	–	C
CT scanning or CT pulmonary angiography should be considered when ICED infection is suspected, and echocardiography is non-diagnostic	–	C
Echocardiography should be carried out as soon as possible (within 24 h) after a diagnosis of ICED infection is considered	–	C
Blood cultures should be taken prior to starting antimicrobial therapy	–	B
Apply meticulous aseptic technique when taking blood cultures to reduce the risk of contamination with skin commensals	–	B
Antimicrobial treatment strategies should be discussed by the multidisciplinary team and should be determined by plans to remove or attempt to salvage an infected ICED, the presence of ICED-IE, and any extracardiac foci of infection	–	C
When there is clinical evidence of generator pocket infection, empirical antimicrobial therapy should be commenced	–	C
Local antimicrobial instillation into an infected generator pocket is not recommended	–	C
The need for empirical antimicrobial treatment for ICED-LI or ICED-IE (prior to the availability of microbiological data) is a clinical decision based on the severity of the infection	–	C

COR class of recommendation, *LOE* level of evidence, *HRS* Heart Rhythm Society, *CIED* cardiac implantable electronic device, *TEE* transesophageal echocardiography, *MRI* magnetic resonance imaging, *MR* magnetic resonance, *ICD* implantable cardioverter defibrillator, *CRT* cardiac resynchronization therapy, *CT* computed tomography, *ESC* European Society of Cardiology, *T* Tesla, *PM* pacemaker, *BSAC* British Society for Antimicrobial Chemotherapy, *ICED* implantable cardiac electronic device, *ICED-IE* ICED lead-associated infective endocarditis, *ICED-LI* ICED lead infection

16.22 Additional Clinical Practice Takeaways

- It is important to remember that pacemakers are essentially antibradycardia devices. Hence, patients with a malfunctioning pacemaker may present to the ER with bradycardia or low cardiac output symptoms.
- Not all pacemaker complications imply an abnormally functioning device. For instance, pacemaker syndrome is caused by the adverse hemodynamics created by atrioventricular dissociation.
- The decision to remove or to implant a new lead without removing the previous one must be accompanied by the clinical data, a multimodality image approach (chest X-ray, echocardiography, fluoroscopy, and tomography), and device interrogation.
- If the decision has been taken to extract an electrode in the case of a subacute (late) lead perforation, the cardiac surgery service must be called even if the lead is going to be transvenous extracted or repositioned.
- In case electrosurgery is needed, try to direct the electrical current pathway at least 6 inches away from the device, and always have a pacemaker magnet ready to use during the procedure.

Acknowledgments Images are courtesy of Ana Lilia Rayas Gómez, MD, Hospital San Jose, Queretaro, Mexico.

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