

IMPLANTABLE DEVICES

Relevant Guidelines

ACCF/AHA/HRS 2018 guideline on bradycardia and conduction delay

2018 ACC/AHA/HRS Guideline on the Evaluation and Management of Patients With Bradycardia and Cardiac Conduction Delay. *J Am Coll Cardiol*. 2019;74:932-987.

ESC 2021 guidelines on pacing and cardiac resynchronization

2021 ESC Guidelines on cardiac pacing and cardiac resynchronization therapy. *Eur Heart J*. 2021.

ESC 2022 Guidelines on ventricular arrhythmias and prevention of sudden cardiac death

2022 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. *Eur Heart J*. 2022.

ESC 2023 guidelines on infective endocarditis

2023 ESC Guidelines for the management of endocarditis. *Eur Heart J*. 2023.

ESC 2026 GL on chronic kidney disease

2026 ESC Guidelines for the management of cardiovascular disease and chronic kidney disease, in collaboration with the European Renal Association (ERA). *Eur Heart J*. 2026.

ESC 2022 Guidelines on noncardiac surgery

2022 ESC Guidelines on cardiovascular assessment and management of patients undergoing non-cardiac surgery. *Eur Heart J*. 2022

AHA/ACC 2024 Guideline on noncardiac surgery

2024 AHA/ACC/ACS/ASNC/HRS/ SCA/SCCT/SCMR/SVM Guideline for Perioperative Cardiovascular Management for Noncardiac Surgery. *JACC* 2024

ESC 2022 Guidelines on cardio-oncology

2022 ESC Guidelines on cardio-oncology developed in collaboration with the European Hematology Association (EHA), the European Society for Therapeutic Radiology and Oncology (ESTRO) and the International Cardio-Oncology Society (IC-OS). *Eur Heart J*. 2022.

ESC 2020 Guidelines on sports

Pelliccia A, Sharma S, Gati S, et al. 2020 ESC Guidelines on sports cardiology and exercise in patients with cardiovascular disease. *Eur Heart J*. 2020.

Relevant Scientific Statements and Position Papers

AHA 2016 Statement on Vascular Graft Infections, Mycotic Aneurysms, and Endovascular Infections

Vascular Graft Infections, Mycotic Aneurysms, and Endovascular Infections: A Scientific Statement From the American Heart Association *Circulation*. 2016;134:e412-e460

HRS 2017 statement on lead management and extraction.

2017 HRS expert consensus statement on cardiovascular implantable electronic device lead management and extraction. *Heart Rhythm*. 2017;14:e503-e551.

HRS/EHRA/APHRS/SOLAECE 2015 consensus statement on ICD programming and testing

2015 HRS/EHRA/APHRS/SOLAECE expert consensus statement on optimal implantable cardioverter-defibrillator programming and testing. *Europace*. 2016;18:159-83.

HRS/EHRA/APHRS/LAHRS 2019 update to 2015 statement on ICD programming and testing

HRS/EHRA/APHRS/LAHRS focused update to 2015 expert consensus statement on optimal implantable cardioverter-defibrillator programming and testing. *Europace*. 2019;21:1442-1443.

AHA/HRS 2007 statement on driving with ICDs

Public safety issues in patients with implantable defibrillators. A scientific statement from the American Heart Association and the Heart Rhythm Society. *Heart Rhythm*. 2007;4:386-93.

EHRA 2009 statement for driving by patients with ICDs.

Consensus statement of the European Heart Rhythm Association: updated recommendations for driving by patients with implantable cardioverter defibrillators. *Europace*. 2009;11:1097-107.

EHRA 2022 Statement on PPM and ICD implantation

EHRA expert consensus statement and practical guide on optimal implantation technique for conventional pacemakers and implantable cardioverter-defibrillators. *Europace*. 2022;24:523-524.

EHRA 2022 Consensus on interference due to medical procedures

EHRA consensus on prevention and management of interference due to medical procedures in patients with cardiac implantable electronic devices. *Europace*. 2022;24:1512-1537.

ESC/EHRA 2023 report on remote monitoring

2023 HRS/EHRA/APHRS/LAHRS Expert Consensus Statement on Practical Management of the Remote Device Clinic. *Europace* 2023

AHA 2024 Scientific Statement on Implantable Device Infection

Update on Cardiovascular Implantable Electronic Device Infections and Their Prevention, Diagnosis, and Management: A Scientific Statement From the American Heart Association: Endorsed by the International Society for Cardiovascular Infectious Diseases. *Circulation*. 2024;149:e201-e216.

AHA 2024 Scientific Statement on Periprocedural Management of Patients With Cardiac Implantable Electronic Devices

Periprocedural Management and Multidisciplinary Care Pathways for Patients With Cardiac Implantable Electronic Devices: A Scientific Statement From the American Heart Association. *Circulation*. 2024;150:e183-e196.

TECHNICAL ISSUES

The number of pacemakers (PMs), implantable cardioverter-defibrillators (ICDs), and implantable loop recorders (ILRs), collectively called cardiac rhythm for management devices (CRMD), implanted each year has increased exponentially. Automated CaRDIA-X is a comprehensive algorithm for CRMD identification using X-rays.¹

Permanent Pacemakers

Pacemaker Modes

A 5-letter code is used to describe the pacemaker mode. The first letter refers to the chamber that is paced (atrium, ventricle, dual), the second letter refers to the chamber sensed (atrium, ventricle, dual), the third letter refers to the response to sensing (inhibit, trigger, dual), the fourth letter indicates the presence or absence of rate modulation, and the fifth letter indicates multisite pacing (Table 1).²⁻³ Thus, a VVI pacemaker is a ventricular only pacemaker, a DDDR a dual chamber pacemaker with rate response ability, a DDI pacemaker is a non-tracking dual chamber pacemaker that paces both the atrium and the ventricle but does not respond to intrinsic sinus or atrial rate changes (no atrial synchronous pacing). DDI can be useful to avoid tracking atrial tachyarrhythmias. Single-chamber atrial or ventricular pacemakers sense myocardial signals emanating from the corresponding cardiac chamber and deliver a pacing stimulus if no signal is sensed at the programmed lower rate. Dual-chamber pacemakers sense and pace both the atrium and the ventricle. Depending on the particular clinical situation and programming, sensed events trigger or inhibit pacing. For example, in the DDD pacing mode and during atrioventricular sequential pacing, atrial pacing takes place at the lower rate limit. Atrial pacing triggers ventricular pacing once the programmed AV delay has timed out. If the atrial rate is faster than the programmed lower rate, atrial pacing is inhibited. After the sensed atrial signal, ventricular pacing would occur only if no ventricular event is sensed by the end of the programmed sensed AV delay.⁴

Rate response

In the presence of chronotropic incompetence, rate-adaptive pacemakers are useful.⁵ These devices have special sensor(s) that, when triggered during exercise, increase the pacing rate. Most commonly used sensors monitor body movement by detecting vibration (activity sensor or accelerometer) (Table 2). More physiologic sensors detect changes in minute ventilation by measuring changes in thoracic impedance with ventilation or changes in QT interval that reflect sympathetic drive. Some pacemakers incorporate more than 1 sensor to limit disadvantages of individual sensors, and enhance specificity without compromising

sensitivity. A commonly used combination is an activity sensor combined with a QT interval sensor or a minute ventilation sensor. Careful programming by considering a short walk or exercise is often required to achieve optimal clinical results.⁵

Choice of Pacemaker Mode

Recommendations by HRS/ACC and ESC are presented in the Chapters on Bradycardia.

Pacemaker syndrome

Ventricular pacing without proper atrial synchronization (as happens with VVI pacing in the absence of AF) results in loss of the atrial contribution to ventricular filling, and atrial contraction may occur against closed AV valves. Thus, pacemaker syndrome develops in 5-25% of patients with VVI pacing, and usually manifests itself by prominent V waves in the JVP. Pacemaker syndrome can lead to symptoms such as dizziness, weakness, heart failure, and presyncope or syncope, and predisposes the patient to the development of atrial fibrillation and increased incidence of stroke.

Pacemaker-mediated arrhythmias

Endless loop tachycardia (ELT) and **repetitive non-re-entrant ventriculoatrial synchrony (RNRVAS)** are pacemaker-mediated arrhythmias that may occur in the presence of ventriculoatrial conduction.⁶ Endless loop tachycardia or repetitive reentrant VA synchrony occurs when the pacemaker senses retrograde P waves and triggers pacing in the ventricle. Ventricular pacing causes retrograde conduction to the atria, and the ELT circuit is established. AV desynchronization arrhythmia or RNRVAS occurs when the pacemaker does not sense retrograde P waves. In this form of VA synchrony, the atrial stimulus is ineffectual because it falls in the atrial myocardial refractory period generated by the preceding unsensed retrograde P wave. A long atrioventricular interval and a relatively fast lower rate (or sensor-driven rate with DDDR pacing) favor the development of AV desynchronization arrhythmia. Most pacemakers have algorithms to recognize and terminate these arrhythmias. Alternatively, adjustment of AV delays or post-ventricular pacing atrial refractory periods (PVARP) are necessary. A novel pacemaker-mediated arrhythmia that closely mimics repetitive non-re-entrant ventriculoatrial synchrony but in the absence of ventriculoatrial conduction has also been described (so called **pseudo-RNRVAS**) and can induce atrial fibrillation.⁷ **Atrial fibrillation** may also be induced by competitive atrial pacing as a consequence of the attempts to maintain the algorithm's prescribed rate, or to compensate for premature ventricular contractions.⁸ Pacemaker component failures are responsible for other unusual but dangerous causes of high pacing rate, such as **runaway pacemaker** and **sensor-driven tachycardia**.⁴ Figure 1 presents an algorithm for evaluation of suspected pacemaker malfunction.

Upper rate behaviour

In dual-chamber pacing mode, the upper rate is determined by the total atrial refractory period (TARP), that is a combination of AV delay and the PVARP. If properly programmed, the device tracks in a 1:1 fashion until reaching the programmed upper rate. As the atrial rate increases, ventricular pacing cannot violate the upper rate limit, resulting in progressively longer AV intervals. This is referred to as pacemaker Wenckebach. However, if the TARP is excessively long, abrupt 2:1 block may develop with a sudden slowing of the ventricular rate, which may cause symptoms. Effectively, the ventricular pacing rate is one-half of the maximum tracking rate. Undesirable upper rate behavior is avoided by meticulous attention to intervals, by programming to allow a Wenckebach interval between the maximum tracking rate and the 2:1 block rate, and by allowing rate-adaptive shortening of the AV and PVARP intervals.³

Leadless pacing

Leadless pacing by means of implantation of a self-contained, very small and potentially retrievable (<8 months post-implantation) VVIR unit within the right ventricle is available (Table 3).^{9,10} VVIR pacing is provided by the Micra and AVEIR systems. The Micra AV offers AV synchronous pacing, but it is limited by the lack of atrial pacing and restricted in providing AV synchrony pacing in higher heart rates. The helix-fixation device (AVEIR VR LP) is designed to be retrievable with a reported success rate of up to 86%.¹¹ An AVEIR atrial module has also been introduced to offer both atrial pacing and can be combined with a ventricular unit for dual-chamber leadless pacing.¹⁰ Overall complications such as infections, haematoma, pneumothorax, and lead fracture or dislocation and reintervention rates appear to be lower with leadless than with conventional VVI pacemakers. There is, however, a higher risk of pericardial effusion and/or perforation.^{12,13 10}

Novel Therapeutic Approaches

The revised WiSE-CRT system provides **wireless pacing** by transmitting acoustic (ultrasonic) energy from a pulse generator transmitter, implanted subcutaneously over the ribcage, to a receiver electrode implanted in the LV.¹⁴ Dual-chamber leadless pacing consists of atrial and ventricular leadless pacing units that communicate bidirectionally and on a beat-to-beat basis to continually deliver AV synchronous pacing. Initial clinical results are satisfactory.^{15,16}

Genetic therapies and **biological pacemakers** are also exciting therapeutic developments.^{17,18}

Biventricular Pacing

Cardiac resynchronization therapy (CRT) is an integral component of modern heart failure therapy for patients with severe symptoms, LVEF $\leq 35\%$, and a wide QRS complex (> 150

ms) in the context of LBBB (see Chapter 33). Biventricular pacing by atrial-synchronized pacing of the RV and the LV via the coronary sinus to the basal or midventricular but not apical LV region,¹⁹ accomplishes reverse remodeling of the LV. The use of the posterior or lateral LV regions is also associated with decreased risk of arrhythmic events in comparison with anterior lead location and ICD-only patients.²⁰ A nonapical positioning of the RV lead has not been considered important to influence outcome,²¹ and might even be proarrhythmic.²² However, in a recent meta-analysis, both that a non-apical and an apical RV lead position were equally effective in CRT.²³ Simultaneous His-bundle and LV pacing have also been found beneficial thus supporting the hypothesis of the crucial role of intrinsic right ventricular conduction in optimal cardiac resynchronization therapy.²⁴

Specific problems of biventricular pacing are implant failure due to inability to obtain LV pacing (7-10%), lead dislodgement (5-10%), and coronary sinus dissection (<1%).^{25,26} Adverse events, such as failure to implant, pneumothorax, pocket hematoma, and infection, are significantly higher among patients subjected to CRT than ICD alone.^{25,26} Proarrhythmia in the form of VT storm may also rarely be seen, especially in patients with nonischemic cardiomyopathy.²⁷ There are no significant differences in clinical outcomes or complication rates between upgrades of existing devices and de novo CRT procedures.²⁸ Recommendations on indications as well as on lead placement and follow-up of devices have been recently published.^{29,30}

Patients with CRT-D who achieve LVEF normalization (>50%) have very low risk of ventricular arrhythmias the risk of inappropriate ICD therapy is still present. Thus, these patients could be considered for downgrade from CRT-D to CRT-P at time of battery-depletion if no ventricular arrhythmias have occurred.³¹

Specific **His** and, especially, **LBBB pacing** are pacing modes clearly preferable to apical RV pacing, and seem to offer similar or better results than biventricular pacing.

Implantable Cardioverter Defibrillators

Secondary and primary prevention randomized trials to assess the impact of ICD are presented in Tables 4 and 5.

ICD are the only means for preventing sudden cardiac death in certain clinical settings, and the development of leadless (subcutaneous) devices for pacing and defibrillation is a novel possibility.³²⁻³⁴ It avoids intracardiac leads but at the expense of lack of bradycardia support or antitachycardia pacing. Several important points should be noted:

1. In patients with documented sustained ventricular arrhythmias and/or cardiac arrest, implantable cardioverter-defibrillators (ICD) confer a survival benefit (secondary prevention of sudden cardiac death). In several clinical settings this might be lost when modern medical

therapy including beta blockers is implemented.³⁵ Repeat shocks may lead to worsening of heart failure and a decline in survival.^{36,37}

2. In a pooled analysis 5 landmark ICD trials (MADIT-II, MADIT-RISK, MADIT-CRT, MADIT-RIT, RAID, the underlying arrhythmic substrate rather than the ICD therapy is the more important determinant of mortality in ICD recipients. The risk of death following an appropriate ICD shock was 38% over a 3-year period, whereas the group that did not have an ICD shock had a 3-year mortality approximating 10%.³⁸

3. In patients without sustained ventricular arrhythmias or cardiac arrest, ICD confer a survival benefit (primary prevention of sudden cardiac death) only in high-risk patients with ischemic cardiomyopathy and LVEF $\leq$ 35% due to a remote MI at least >40 days and, especially, >18 months.^{35,39} The 40 days period is controversial (DAPA trial).⁴⁰ Patients with a recent myocardial infarction and LVEF $<25\%$ and/or wide QRS do not benefit from ICD.⁴¹ The value of ICD in nonischemic heart failure, and in patients >60 years old or diabetics, is controversial, but most probably ICD is beneficial,⁴²⁻⁴⁶ and recommended by current guidelines in patients with LVEF $\leq 35\%$. In a long-term follow-up (13 years) of the DANISH RCT, although ICD therapy continued to reduce sudden cardiac death by 46%, this benefit was attenuated with advancing age, and no benefit was observed in patients >70 years of age.⁴⁷ The optimal timing of implantable cardioverter-defibrillator placement for primary prevention following an index hospitalization for heart failure is not established. These patients are at increased of implantation-related complications.⁴⁸

4. Concerns have been raised about an excessive and inappropriate number of ICD implants in genetic channelopathies.⁴⁹

5. The strongest predictor of SCD remains reduced LVEF, with a cutoff of $\leq 35\%$, as determined in randomized, controlled studies. Sudden death characterizes the mode of death in 30–50% of patients with chronic heart failure and a reduced ejection fraction. This may be due to a first-time ventricular tachyarrhythmia that is responsive to the shock delivered by an ICD, or it may present as an acute mechanical failure, which manifest as asystole, or electromechanical dissociation, or as incessant ventricular fibrillation that persists despite repetitive ICD discharges; in both instances, the sudden deaths cannot be prevented by an ICD. Thus, 30–70% of the sudden deaths in clinical trials of HFrEF are not prevented by an ICD.⁵⁰ Of note, more individuals with LVEF $>35\%$ die suddenly than those with lower LVEF.⁵¹

6. The therapeutic effect of prophylactic ICD implantation on mortality can be predicted by measuring periodic repolarisation dynamics, a novel electrophysiological marker that quantifies sympathetic-activity-related low-frequency oscillations of cardiac repolarisation instability.⁵² The MADIT-ICD benefit score (<https://is.gd/madit>) is also useful for estimation of the expected benefit of the ICD implanted for primary prevention (Figure 2).⁵³

7. The benefits of ICD in the elderly, especially those with nonischemic cardiomyopathy are not established.^{9,54-56} Rates of appropriate shocks are similar across age groups,⁵⁷ but data from Medicare and Medicaid indicate that one-third of 66,974 ICD recipients die within 3 years, reflecting a population with more advanced age and progressed heart failure than seen in trial populations for primary prevention ICD.⁵⁸ While high-risk patients may show the greatest short-term gain, it is the lower risk patients, e.g. primary prevention ICD implantation, who gain the most life-years over their lifetime.⁵⁹ The ESC 20022 GL on VA and SCD recommend that in elderly patients in whom a benefit from the defibrillator is not expected due to the patient's age and comorbidities, omission of ICD implantation for primary prevention may be considered (IIb B).⁶⁰
8. Prophylactic implantable cardioverter-defibrillator therapy in patients undergoing long-term dialysis with left ventricular ejection fraction $\geq 35\%$ does not reduce the rate of sudden cardiac death or of all-cause mortality.⁶¹
9. With current prices, ICD are definitely cost-effective only when used in high-risk patients without associated comorbidities that limit the life expectancy to <10 years.³⁵
10. Implant failure rates are 0.1%
11. Recently, concerns have been raised about the possibility of ICD inactivation by mobile phones with powerful magnets such as the iPhone 12.⁶²
12. The efficacy and safety of ICD and CRT devices have not been established for patients with advanced chronic kidney disease, and available data suggest that they may be less favorable than in patients without advanced chronic kidney disease.⁶³

The **subcutaneous ICD** is an approach useful for primary prevention in young patients without need of pacing or in cases of difficult venous access.^{64,65} Subcutaneous ICD devices reduce lead-related complications compared with transvenous ICDs, but concerns have been raised about delivery of inappropriate shocks mainly due to T wave oversensing.⁶⁶ However, in the PRAETORIAN RCT, although patients in the S-ICD group were more likely to receive an ICD shock, the total number of appropriate shocks was not different between the two groups,⁶⁷ and complication rates were significantly lower with the subcutaneous ICD.⁶⁸ In the UNTOUCHED trial, the inappropriate shock rate was low (3.1% at 1 year) indicating high efficacy and safety with contemporary S-ICD devices and programming.⁶⁹ In the PRETORIAN-XL RCT, patients with a transvenous ICD had an increased risk of lead-related and major complications. There was a higher generator change rate in the subcutaneous ICD group.⁷⁰

The **extravascular ICD** has a single lead implanted substernally to enable pause-prevention pacing, antitachycardia pacing, and defibrillation energy similar to that of transvenous ICDs. Initial clinical experience is promising.⁷¹

Practical points regarding the choice and programming of ICD devices:

1. Single-coil leads are recommended for primary prevention ICDs.⁷²⁻⁷⁴ Single-coil leads have a marginally higher, but probably clinically insignificant, defibrillator thresholds (DFT).⁷⁵ They are preferred to dual-coil leads because they are easier to be extracted.
2. Single-coil leads can either be true (dedicated) bipolar ie between the tip electrode and a small ring electrode or integrated bipolar ie between the tip electrode and right ventricular coil. The larger integrated bipolar antenna is more susceptible to external electromagnetic interference, R-wave double counting, and diaphragmatic myopotentials. True bipolar leads are more susceptible to T-wave oversensing, probably because of greater variations in R-wave amplitude, and this could be of importance in patients with hypertrophic cardiomyopathy, dilated cardiomyopathy, cardiac sarcoidosis, long- and short-QT syndrome, and Brugada syndrome, in whom T-wave oversensing has been reported to be more frequent.⁷⁶
3. Dual-chamber ICDs are associated with a higher risk of complications than single-chamber ICDs,⁷⁷ and discrimination of ventricular arrhythmias alone is not a valid reason to implant a dual-chamber device. Atrial or ventricular pacing as well as rate responsive pacing should be avoided, if possible, to increase generator longevity and avoid disruption of normal ventricular conduction.
4. Defibrillation testing is not necessary with current technologies.^{78 79} It might be considered in secondary prevention and conditions associated with higher defibrillation thresholds (DFT), such as right-sided implants and amiodarone therapy. It is also not necessary with subcutaneous ICD when the PRAETORIAN is <90. The PRAETORIAN score stratifies the risk of conversion failure into three categories: low risk (<90 points), intermediate risk (90–<150 points), and high risk (≥ 150 points). After confirming that the chest X-rays meet the required criteria to calculate the PRAETORIAN score, the score is derived through a four-step process. Steps 1 and 2 are based on the lateral chest X-ray: step 1 quantifies the amount of adipose tissue between the coil and the sternum, while step 2 evaluates the position of the generator relative to the midline of the left thoracic wall. Step 3 is determined from the posteroanterior chest X-ray and measures the amount of fat tissue between the closest point of the generator and the thoracic wall. In step 4, 40 points are deducted in patients with a body mass index ≤ 25 kg/m² when the total score is ≥ 90 points.⁸⁰
5. Inappropriate ICD shocks may cause myocardial damage and have been associated with increased mortality.³⁷ Strategies to avoid inappropriate shocks should be sought.⁸¹ Carvedilol is probably superior to metoprolol in preventing inappropriate shocks.⁸² Extension of detection duration to prevent treatment of self-terminating tachycardia (30-40 intervals) is the most useful strategy to avoid unnecessary shocks. Higher cutoff rates for arrhythmia therapy should be used, ie >170 bpm for VT monitoring, antitachycardia pacing for VT with CL<300

ms (>200 bpm) unless if a clinical slower VT had been recorded, and shocks for VF with CL<200 ms (>300 bpm) or CL<300 ms (>200 bpm) after failed antitachycardia pacing and a 2.5 s monitoring delay. The application of SVT-VT discrimination (onset, stability, and morphology algorithms) measures is also useful. These measures have been found useful both for primary⁸³⁻⁸⁶ and secondary prevention.⁸⁷ Anti-tachycardia pacing, however, may be considered for slower VT episodes,⁸⁸ especially for monomorphic VT that has been shown to respond to pacing at EP study. In the PRAETORIAN trial, efficacy of the first antitachycardia pacing attempt was 46% in monomorphic VT.⁶⁷ Burst appears to be more efficacious than ramp in this respect.⁸⁹ Treatment-induced increase in plasma potassium levels (potassium chloride tablets, 750 mg -approximately 10 mmol, a mineralocorticoid receptor antagonist, or both) lead to a significantly lower risk of appropriate ICD therapy, unplanned hospitalization for arrhythmia or heart failure, or death from any cause than standard care.⁹⁰

6. A railroad track pattern refers to the alternation of ventricular intervals that produces the appearance of a railroad tracks, due to various types of oversensing, usually T wave oversensing. It may also caused by R wave double counting, triggered repetitive programming changes, such as ventricular safety pacing, or cardiac resynchronization therapy response to premature ventricular complexes.⁹¹

7. The use of discrimination algorithms active from 200 to 250 bpm, should be adopted routinely in patients with subcutaneous ICD to avoid unnecessary shocks.⁶⁹

8. ICD patients often die in the hospital, and one third have ventricular tachyarrhythmia events with shocks close to death. Deactivation of the ICD should be considered when a DoNotResuscitate order is decided to avoid unnecessary painful shocks.⁹²

9. Approximately 25% of patients who receive primary prevention ICDs may no longer meet guideline indications for ICD use at time of generator replacement, and these patients receive subsequent ICD therapies at a significantly lower, but not negligible, rate per year (up to 5%).^{93,94}

10. The HRS now recommend remote monitoring as the new standard of care for patients with cardiac devices, with alert-driven in-person evaluation replacing most routine office interrogations.⁹⁵

11. The ESC recommendations for appropriate ICD programming are presented in Table 6. The HRS/EHRA/APHRS/SOLAECE have also published recommendations on optimal ICD programming and testing.⁹⁶ A 2019 update also provided manufacturer-specific recommendations.⁸⁸

Remote Monitoring

Remote monitoring is beneficial for the management of patients with cardiovascular implantable electronic devices by impacting morbidity and mortality. Incorporation of remote monitoring into follow up practice, and integrating this technology with a modified frequency of conventional in-person evaluation ensures greater patient retention, and improves adherence to scheduled evaluations. Thus, remote monitoring represents the new standard of care for patients with cardiac devices, with alert-driven in-person evaluation replacing most routine office interrogations. ESC/EHRA have published legal requirements and ethical principles of remote monitoring emphasizing the need for cybersecurity precautions.⁹⁷

Detailed recommendations have also been published in the 2023 HRS/EHRA/APHRS/LAHRs Expert Consensus Statement.⁹⁸

Antibiotics for Implantation

Pre-procedural cefazolin 1 to 2 g IV 60 min before skin incision or vancomycin 1 to 1.5 g IV administered within 120 min before skin incision in penicillin-allergic patients, is the standard prophylaxis for device implantation. However, cefazolin does not protect against methicillin-resistant gram-positive organisms, such as *Staphylococcus aureus* and *S. epidermidis*, which are responsible for 20% of device infections. A regime of incremental antibiotics, with pre-procedural cefazolin plus vancomycin, intra-procedural bacitracin pocket wash, and 2-day post-procedural oral cephalexin or clindamycin resulted in lower but not statistically significant infection rate compared to standard prophylaxis (PADIT trial).⁹⁹ An antibiotic-eluting PPM envelope has also reduced pocket infections and may be considered, especially in immunocompromised patients.¹⁰⁰ The routine use of postoperative antibiotics is not supported by published data.¹⁰¹

Device implantation in the anticoagulated patient

In patients on chronic anticoagulation, bridging is not required, especially in patients at low risk of thrombosis (Table 7).¹⁰²⁻¹⁰⁸ In the continued-warfarin group, the INR in the day of surgery should be ≤ 3 , except for patients with one or more mechanical valves, for whom an INR ≤ 3.5 or less is permitted.¹⁰² Continued or interrupted DOAC at the time of device implantation/revision are both acceptable because both are associated with a low risk of hematoma formation.¹⁰¹ It has been postulated that during minor surgery, hemostasis is primarily dependent on capillary and platelet function as opposed to the coagulation cascade itself, and antithrombotics that interfere with the former (e.g., heparin and antiplatelet drugs) are less well tolerated than those that do not. Another explanation is the concept of an “anticoagulant stress test.” That is, if patients undergo surgery while receiving full-dose

anticoagulation therapy, any excessive bleeding will be detectable and appropriately managed while the wound is still open.¹⁰⁵ For the new, direct, oral anticoagulants (DOAC), a 24 h discontinuation with reinitiation 48 h after implantation (24 h in patients with a CHADS₂DS₂VASC score >4) is recommended.^{105,109} In other patients, DOACs may briefly interrupted or not interrupted at all.¹⁰⁸

Perioperative Management of Patients with Devices

Recommendations are provided in Figure 3 and Table 8. The AHA has also published a statement on the issue.¹¹⁰ Recommendation sfor patients with chronic kidney disease are provided in Table 9.

Magnetic Resonance Imaging

Up to 75% of patients with implanted conventional devices (not MRI-compatible) may need an MRI in the future, and this can now be performed safely even in older, non-MRI compatible devices, as long as a 1.5 T magnet is used, and a number of precautions are taken (Table 10 and Figure 4).¹¹¹⁻¹¹³

Currently, however, most manufacturers propose devices that are MRI conditional. It is the entire CIED system (i.e. combination of generator and leads,) that determines MRI conditionality, and not the individual elements. MRI scans may be limited to 1.5 T and a whole-body specific absorption rate (SAR) <2 W/kg (head SAR <3.2 W/kg), but some models allow 3 T and up to 4W/kg whole-body SAR.¹¹¹ Patients with abandoned CIED leads may be safely subjected to MRI, including examination of the thorax.¹¹⁴

Electromagnetic Interference

The main cause of electromagnetic interference is cautery during surgical operations.¹¹³ Daily life electromagnetic interference of low frequency do not disturb sensing capabilities of ICDs, especially in patients with true bipolar lead.¹¹⁵ Electric automobiles are also safe.¹¹⁶ Strong interference ($\geq 50\text{Hz}$), present in certain occupational environments, such as high voltage power lines and installations, may cause inappropriate sensing potentially leading to false detection of atrial/ventricular arrhythmic events.¹¹⁷ Current-generation electric cars should not have any significant impact on device function.¹¹⁸ However, the impact of “super-charged technology” on device function is unknown.¹¹⁹ For protection from static magnetic fields, the International Organization for Standardization (ISO) 14117:2019 standard specifies a limit in magnetic flux density (B field) of 1 mT, up to which magnet-sensitive switches of CIEDs should remain unaffected. Devices with a powerful magnet array such as the iPhone 12 and

MagSafe accessories should not be put directly on the skin, and ideally should be kept at least 15 cm away from the devices to avoid potential interaction.¹²⁰

Radiotherapy

Radiotherapy can cause malfunction of cardiac implantable electronic devices (CIEDs). The risk generally increases with the radiation dose, although the strongest predictor of malfunction is the magnitude of exposure to neutron emission from high-energy photon RT, conventionally defined as a beam energy >10 megavolts (MV) (Figure 5).^{113,121} Non-neutron-producing treatment is therefore preferable (up to 5.4 Gy). In a recent study comparing neutron producing radiotherapy with 15- or 18-MV photons and non-neutron producing with electrons, GammaKnife, or 6-MV photons, all cases of single-event upset malfunction occurred in the setting of notable neutron production.¹²²

With photon radiation applying energy >6-10 MV, the risk of malfunctions (usually soft errors) is due to secondary neutron production, is not associated with the target zone, and cannot be shielded. With a cumulative dose reaching the device >2 Gy (moderate risk) or >10 Gy (high-risk): the dose reaching the pacemaker can be estimated before and measured during treatment, is correlated with the target zone, and can be shielded.¹¹¹

If the CIED is > 10 cm from the edge of the radiation treatment area, estimation of the cumulative dose (which will be <2 Gy) with *in vivo* dosimetry is not necessary. If the CIED is between 3 and 10 cm from the edge of the radiation treatment area, an *in vivo* dosimeter should be placed over the device to measure the dose from the first treatment fraction. If the nearest edge of the CIED generator is <3 cm from the edge of the radiation area, the maximum dose to the CIED can be estimated using the treatment planning system.¹²³

Experience with **proton radiation therapy** in CIED patients is limited. However, compared with photon irradiation, this radiation modality produces more secondary neutrons, which may affect the risk of device errors or failure.¹¹¹ With newer CIEDs implementing the complementary metal-oxide semiconductor (CMOS) technology which allows the miniaturization of CIED, it is also more susceptible to RT.¹²³

At the completion of RT, the device should be evaluated to determine whether it may need to be reprogrammed or replaced. The patient should continue to be monitored following RT at 1 and 6 months after the last RT fractions to assess possible late radiation-induced effects, such as device malfunction, functional changes, and increased sensitivity to electromagnetic interference.¹²³

The ESC recommendations are presented in Figures 5-8, and Table 11.

Noncardiac Surgery

Recommendations for patients with transvenous or nontransvenous devices are provided in Table 12 and Figures 9 and 10.

Follow-Up

Recommendations are provided in Table 13.

Driving, Sports, and Sexual Activity

Driving is allowed 4 weeks after permanent pacemaker implantation.¹²⁴ Following ICD implantation professional driving is not allowed.^{124,125} Private driving is allowed as indicated in Table 14. Restriction of 2 to 4 months following an appropriate shock was proposed by a European study,¹²⁶ whereas in a US analysis the incidence of a second shock after an initial ICD discharge at 6 months was approximately 28.7%, which is lower than previously reported and depended on several programmed ICD variables.¹²⁷ In a nationwide Danish survey, many ICD patients were unaware of driving restrictions, and more than one third resumed driving during a driving restriction period. However, the rate of reported ICD shocks while driving was very low, with an estimated risk of harm was 0.0002% per person-year.¹²⁸ The results of the DREAM-ICD-II study suggest that a 3-month driving restriction for private driving after the index cardiac event may be safe and adequate in recipients of a secondary prevention implantable cardioverter defibrillator.¹²⁹

Current recommendations do not allow intensive or competitive sports in patients with ICDs. However, a more liberal policy may be considered in some competitive athletes and in those who want to perform mild-to-moderate recreational activities.¹³⁰ The ESC recommendations are presented in Table 15. Sexual activity is reasonable for patients with an ICD used for secondary prevention in whom moderate physical activity (>3–5 METS) does not precipitate ventricular tachycardia or fibrillation (AHA/ESC 2013 consensus on sexual counselling, IIa-C).¹³¹

Pregnancy

The management of patients with ICD and/or pacemakers during pregnancy is described in Table 16 and Figures 11 and 12.¹³²

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