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Cardiac Implantable Electric Devices: Indications and Complications

Brian Hiestand [1]

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Abstract
Purpose of Review As the evidence base for cardiac implantable electric devices (CIED), which include pacemakers, defibrillators, and devices with both functionalities, continues to expand, so does the rate of usage in patients with chronic heart failure (CHF), atrial fibrillation (AF), or both. Given that no medical intervention is ever free from complications and unintended consequences, it is expected that patients will present to the Emergency Department (ED) for unscheduled, acute care secondary to CIED complications. In this article, we will examine first the indications for CIED placement in patients with chronic cardiac disease. Then, management of various common device-related complications and pathologies will be discussed.
Recent Findings Indications for CIED continue to expand and be refined. With substantial monitoring, patients with certain CIED may undergo MRI; however, the risk-benefit ratio should be examined closely on an individual occurrence level.
Summary Understanding the indications, the complications, and how CIED affect diagnostic options is crucial to providing optimum care for these patients.

Keywords Chronic heart failure (CHF) · Atrial fibrillation (AF) · Cardiac implantable electric devices (CIED) · Emergency department (ED)

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✉ Brian Hiestand
bhiestan@wakehealth.edu

[1] Department of Emergency Medicine, Wake Forest University Health Sciences, Winston-Salem, NC, USA

Introduction

As the evidence base for cardiac implantable electric devices (CIED), which include pacemakers, defibrillators, and devices with both functionalities, continues to expand, so does the rate of usage in patients with chronic heart failure (CHF), atrial fibrillation (AF), or both. Given that no medical intervention is ever free from complications and unintended consequences, it is expected that patients will present to the Emergency Department (ED) for unscheduled, acute care secondary to CIED complications. In this article, we will examine first the indications for CIED placement in patients with chronic cardiac disease. Then, management of various common device-related complications and pathologies will be discussed.

CIED Basics

Traditional CIED consist of 1 to 3 leads, which run transvenously to implant in the myocardium, and the central canister, which contains the battery, generator, and all programming functions. The canister is generally located subcutaneously on the upper left thorax; however, right-sided or even abdominal placement may be utilized if vascular abnormalities or previous site infections render the left chest unusable. Defibrillator capability will require the presence of a shock coil, which is generally implanted in the right ventricle. Pacing leads may be implanted in the right ventricle, right atrium, or left ventricle, in the setting of bi-ventricular pacing (Fig. 1).

An alphabetic code system describes the functionality of the CIED (Table 1) [1]. The first letter delineates the cardiac chamber that is being paced. The second letter describes the chamber that is sensed by the pacemaker. The third letter describes the response of the device when a native cardiac beat is

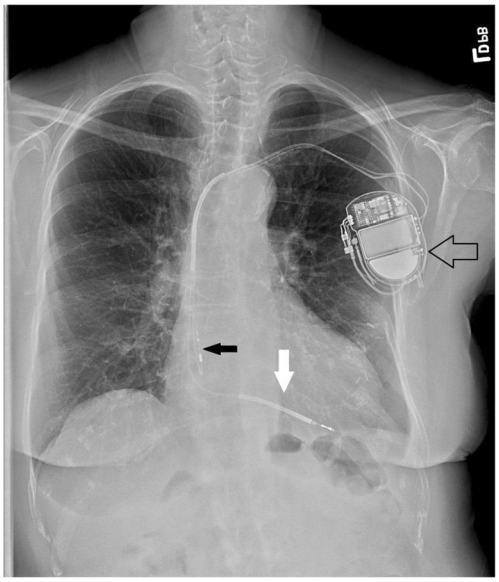

Fig. 1 Chest x-ray of patient with pacemaker with defibrillator functionality. *Open arrow*: canister. *Solid black arrow*: atrial pacing lead. *Solid white arrow*: right ventricular lead with shock coil

sensed. When a spontaneous depolarization occurs, the device may be triggered to send a pacemaker signal, may be inhibited from sending a signal, or may have a dual response (both inhibited and triggered, in the setting of dual chamber pacemakers). The fourth letter establishes whether the device can produce a variable rate based on cardiac demand ("R") or whether the output is fixed ("O"). The fifth and final position establishes whether or not the device is a multi-chamber pacemaker or not.

"Leadless" cardiac pacemakers avoid the potential failure node of pacemaker leads via direct implantation onto the endocardial surface. The two devices under study—the Nanostim LCP (leadless cardiac pacemaker, St. Jude Medical) and the Micra TPS (transcatheter pacing system, Medtronic)—are both delivered to the right ventricle via femoral vein catheterization and affixed to the ventricular wall from inside the ventricular septum, where they then deliver single chamber ventricular pacing. Early clinical trials have demonstrated proof of functionality, long-term programming stability, and the ability to retrieve the devices when necessary [2–6]. Both devices have received the CE Mark allowing sale in the European Union. However, only the Medtronic Micra TPS has received US FDA clearance at the time of this writing.

Subcutaneous implantable cardiac defibrillation (S-ICD), while not truly leadless, avoids direct cardiac contact with the defibrillator coil, thus avoiding the risks of vascular injury and cardiac damage. Sensing leads and the coil are placed subcutaneously in the chest wall [7]. While this technology will not benefit patients requiring pacemaker functionality as well as defibrillation capability, the efficacy and safety data from both prospective non-randomized studies and registry data have been sufficient to secure FDA approval and the CE Mark for the EMBLEM S-ICD system (Boston Scientific) [8, 9]. Currently, an industry sponsored prospective randomized controlled non-inferiority study between S-ICD and conventional transvenous ICD is underway [10].

Indications for Device Therapy in Chronic Heart Failure and Atrial Fibrillation

There are two potential therapeutic modalities delivered by a CIED—defibrillation and pacing. Both functions may be potentially value added in a chronic heart failure patient. Patients with CHF are at increased risk for both ventricular and atrial dysrhythmias, including sudden cardiac death (SCD). While the annual incidence of SCD in the general population is approximately 0.2% [11], this annual rate exceeds 30% in patients with ischemic cardiomyopathy and a history of dysrhythmia [12]. SCD is the final cause of death in approximately 50% of CHF patients [13]. Other high-risk cohorts include those with a previous history of SCD or ventricular tachycardia/ventricular fibrillation (VT/VF). CIED implantation is recommended for secondary prevention of SCD in CHF patients who have survived a first episode, regardless of etiology (ischemic vs. non-ischemic) or degree of systolic function (preserved vs. reduced ejection fraction) [14]. It should be noted that this indication is only applicable for those patients in whom life-prolonging therapy is desired. Patients with purely palliative goals of care will likely not derive benefit from defibrillator placement.

Primary prevention, which describes SCD prophylaxis prior to a first event, is indicated in many patients with CHF.

Table 1 The North American Society of Pacing and Electrophysiology/British Pacing and Electrophysiology Group generic code for pacemakers

Position	I	II	III	IV	V
Category	Chamber paced	Chamber sensed	Response to sensing	Rate modulation	Multisite pacing
Nomenclature	O = None A = Atrium V = Ventricle D = Dual (A and V)	O = None A = Atrium V = Ventricle D = Dual (A and V)	O = None T = Triggered I = Inhibited D = Dual (T and I)	O = None R = Rate modulation	O = None A = Atrium V = Ventricle D = Dual (A and V)

Multiple studies have demonstrated a benefit to CIED vs. medical therapy alone in patients with heart failure with reduced ejection fraction (HFrEF). This benefit was established in landmark trials for both ischemic (MADIT, MADIT II) [15, 16] and non-ischemic (SCD-HeFT) [17] cardiomyopathies. This contention was recently challenged by the DANISH study, which failed to demonstrate an all-cause survival benefit with the addition of prophylactic CIED vs. standard medical care in patients with HFrEF (<35%) due to non-ischemic etiologies, although SCD rates were reduced [18]. However, a meta-analysis of CIED studies in patients with non-ischemic cardiomyopathy, incorporating DANISH in the data set, continued to show a reduction in all-cause mortality and SCD in the CIED population [19•].

In addition to SCD prophylaxis, certain patients with CHF may benefit from direct cardiac pacing to manage the beat-to-beat conduction of the failing heart. Sluggish and asynchronous ventricular contraction can worsen cardiomyopathy, leading to unfavorable cardiac wall remodeling and worsening systolic function. The goal of cardiac resynchronization therapy (CRT) is to utilize biventricular pacing to establish synchronous depolarization and contraction of both ventricles, minimizing mechanical dyssynchrony. The use of CRT has been associated with improved ventricular remodeling, symptom improvement, and decreased days-in-hospital [20]. The patients who benefit the most from CRT have reduced ejection fraction, milder symptomatic CHF (New York Heart Association (NYHA) III vs. IV), and a wide QRS complex, indicating impaired electrical conduction [21–28]. There was controversy over whether patients with echocardiographic evidence of dyssynchrony, but a narrow QRS, would benefit from CRT. The EchoCRT study specifically addressed this concept, randomizing 809 patients with symptomatic HFrEF, QRS complex <130 msec, and echocardiographic evidence of dyssynchrony to CRT or a sham device [29]. The trial was actually stopped early due to increased mortality in the CRT arm, suggesting that in the absence of conduction dysfunction, CRT may actually be harmful. However, multiple clinical trials have established benefit in quality of life and survival for the appropriate patient.

Atrial Fibrillation

Rate control is the primary indication for CIED placement in patients with AF. This may consist of basal rate support in patients with tachy-brady syndrome or sick sinus syndrome, controlling symptoms from inappropriate bradycardia [30]. An additional indication for pacing in AF is for total rate support after AV node ablation. Ventricular pacing, independent of the atrial signal, is designed to eliminate the possibility of rapid ventricular response. This may be indicated in patients who cannot tolerate pharmacologic management and who have recurrent symptomatic episodes of rapid ventricular response. This can result in consistent improvement in symptom control and quality of life [31], but at the cost of the patient being device dependent for the remainder of their life [32]. Given that a substantial portion of these patients (~70%) cannot mount enough of a ventricular escape rhythm to sustain hemodynamically sufficient circulation, device failure would be catastrophic [33]. Patients with coexisting AF and CHF may benefit from a dual modality approach. In these patients who meet criteria for CRT, the combination of CRT and AV nodal ablation has demonstrated consistent clinical benefit [34–36].

Device Complications

Patients may experience complications related to their CIED either initially or late. Initial complications include pneumothorax, pocket hematoma, vascular injury, or wound dehiscence. Later complications include lead failure/fracture, deep venous thrombosis, and myocardial perforation. Infection can occur at any point, localizing to the site pocket or resulting in bacteremia due to infected leads.

Device Complications: Electric

CIEDs are not always successful in executing their programmed functions. “Failure to capture” refers to the inability of a discharged spike to transmit and depolarize cardiac tissue. This will be evident on an EKG when there is a pacer spike but no subsequent atrial or ventricular response. Failure to capture may be caused by battery depletion, lead damage, scar tissue or perforation at the implantation site, electrolyte abnormalities, or the effect of antiarrhythmic drugs. The term “failure to pace” refers to the failure of the device to deploy a signal at all and is generally secondary to programming, battery, or lead malfunction, as opposed to patient characteristics.

Oversensing occurs when the device misinterprets a stimulus as being an appropriate cardiac electrical conduction and can lead to the device failing to execute an appropriate response. The CIED interprets the signal as an indication that a pacemaker response is not required and therefore does not pace appropriately. In addition to lead fracture, oversensing can be triggered by lithotripsy, skeletal muscle contraction, and certain types of electromagnetic interference [37]. Undersensing refers to the converse, where the pacemaker fails to be inhibited by a native cardiac electrical pulse and a spike is delivered at an inappropriate interval. Myocardial fibrosis, electrolyte abnormalities, and lead fracture may result in undersensing.

The incidence of pacemaker-induced tachycardia has been limited due to algorithms present in the programming of newer devices [37]. In a dual chamber pacemaker, a premature atrial beat could trigger a re-entry tachycardia, with the pacemaker circuit itself acting as the accessory pathway to

facilitate retrograde conduction. AV nodal blocking medications such as adenosine are effective, as is the use of a magnet to switch device programming to simple asynchronous pacing. If a magnet is used to terminate the arrhythmia, interrogation and reprogramming of the device will be required.

Patients may occasionally present with multiple serial electrical shocks from their defibrillator. This may be inappropriate, caused by misinterpretation of the underlying rhythm by the device, or appropriate, in the setting of recurrent VT/VF. Less likely, this may occur with battery failure, as impotent but palpable shocks are delivered but fail to terminate the arrhythmia. While placing the magnet over the CIED canister will halt the defibrillation activity, the clinician must be prepared to externally defibrillate/cardiovert should the CIED have been delivering appropriate therapy for a lethal arrhythmia all along. Once the defibrillator function has been deactivated by the magnet, further resuscitation protocols do not differ simply due to the presence of the CIED. The only consideration is that, when possible, the external pads should not be placed over the CIED itself [38].

When CIED programming malfunction is suspected in the acute care setting, care should focus on identifying and correcting underlying metabolic abnormalities while providing supportive care. Device interrogation should occur expeditiously. While ED personnel can be trained to acquire CIED data [39, 40] and more device manufacturers are establishing a remote interrogation/central interpretation product for acute care settings [41, 42], these patients will frequently require programming management. This is especially true if the use of a magnet is required.

Device Complications: Mechanical

Lead failure is an infrequent event. In a pooled meta-analysis of observational studies, lead failure was documented in 1265 cases of 136,509 observed lead years, resulting in an approximate rate of 0.93 failures per 100 lead years [43]. Leads may fail due to component fracture or dislocation and will frequently present with failure to sense or failure to pace. Lead dislodgement typically occurs early after implantation, but late inflammation can develop in the myocardium resulting in the lead coming loose. Although direct trauma has been reported to cause lead or canister damage [44–49], in general, the patient is at far more risk of severe injury from the trauma than their CIED is [50]. The presentation of lead failure may be dramatic or subtle and may only be detected on device interrogation.

Lead migration may cause phrenic nerve stimulation, resulting in intractable hiccoughs [37]. Myocardial perforation may also occur, with severity ranging from asymptomatic pericardial effusion all the way to pericardial tamponade. Although the majority of perforations occur during implantation, a non-insignificant number will present in delayed fashion [51]. The therapeutic action taken will depend on the hemodynamic status of the patient—watchful waiting vs. simple lead removal and replacement may suffice in the stable patient [51, 52]. However, pericardiocentesis may be required in the setting of tamponade and shock, either as a temporizing measure before open repair or as a definitive therapy in the case of self-limited microperforation.

Infection

Infection can occur at any time after implantation, from immediate post-implantation pocket infection to secondary infection occurring years after placement. Both intracardiac and extracardiac components of the CIED may be affected. Epidemiologic data is difficult to firmly establish due to heterogeneity of included cohorts, definitions, and variance in length of follow-up; however, it is clear that device-associated bacteremia and endocarditis carry a higher mortality rate (approximately 30%) than local pocket infection does (~5–6%) [53••]. Fortunately, endocarditis represents less than 10% of device-related infections. The majority of infections are caused by Gram positive bacteria, predominantly staphylococcal species and skin flora [53, 54]. Retrospective registry work suggests that staphylococcal bacteremia from another infectious source will result in device infection approximately one-third of the time [55]. While overt pocket infection is easy to recognize in a delayed presentation (especially if the device has eroded through the skin and is exposed), it may be difficult in the immediate post-operative period to distinguish between early cellulitis and the inflammatory changes of routine wound healing. Malaise, night sweats, recurrent low-grade fevers, and distal septic emboli may be the only signs of lead infection or device-associated endocarditis. Fortunately, overt sepsis is infrequent. After initial stabilization and evaluation, including blood cultures prior to antibiotic treatment, admission to a center with experience in dealing with CIED is warranted for echocardiography and further evaluation of potential device infection in these patients [53].

Thrombosis

Upper extremity vascular stenosis and thrombosis may occur secondary to direct endothelial activation from mechanical stimulation. The incidence of reported lead-associated stenosis and thrombosis varies markedly in different cohorts (from 14 to 64%) and also varies by diagnostic modality involved [56–58]. Frank obstruction resulting in superior vena cava syndrome is much less frequent, occurring in less than 1% of patients [59, 60]. Usually, stenosis, lead thrombi, and even small pulmonary emboli are sub-clinical [61]. The rate of overt, clinically significant pulmonary emboli in a retrospective cohort of CIED patients was no different than age- and sex-matched historical controls [62]. Conservative

management is likely appropriate in asymptomatic cases, while patients with vascular complications may need anticoagulation, device extraction, or even mechanical repair of the vessel [59].

Additional Considerations

Conducted Electrical Weapons Discharge

Conducted electrical weapons (CEW) are those which utilize pulses of conducted electricity to induce painful muscle spasms [63]; the TASER (TASER International, Scottsdale, AZ) is typical of the class of weapons. Although in-custody cardiac deaths after CEW deployment have occurred, the role of the CEW has not been definitively established. A review of 178 law enforcement records of CEW deployment into the chest recorded no immediate cardiac events [64]. Concern for cardiac events after CEW discharge persists to the point that a CEW probe capable of cardiac telemetry post-deployment is in development [65]. In addition, there is a concern for CIED presence as a conduit for external electrical stimulation. Cao et al. presented a case of a male with a CRT device receiving CEW discharge to the thorax [66]. Subsequent interrogation of the device noted that the device recorded the electrical impulse of the CEW as well as evidence of transient electrical capture by the CEW. Likewise, there has been a case report of a defibrillator recording and interpreting a CEW discharge as ventricular fibrillation; as the episode was brief and not repeated, no shock was delivered [67]. Animal models suggest that there is unlikely to be direct impact on device programming by CEW discharge [68]. However, until there is a larger body of literature of patients with CIED receiving CEW, it would seem prudent to perform device interrogation on those patients presenting for care after such an event.

MRI and CIED

For years, dogma has been that the presence of a CIED precludes MRI. The potential complications induced in early generation CIED components, when exposed to the intense magnetic field of the MRI, are legion. Lead and canister physical dislodgement, heat-induced tissue damage, or device reprogramming are all possible sequelae of MRI exposure of cardiac devices [69]. In order to meet clinical demand, manufacturers have developed *MRI-conditional* devices that are more robust to the rigors of the magnetic fields. These devices are specifically engineered to minimize risks of complications when paired with specific MRI protocols. It must be emphasized that at this time, there is no device that is unconditionally *MRI safe*. Cohort studies examining site-specific imaging and patient-management protocols have suggested that, with diligence, MRI can be performed without overt long-term issues [70, 71]. The most recent cohort analysis of patients receiving non-thoracic MRI found a low, but non-zero, rate of complications [72•]. In 1500 scans, there were 6 partial programming resets. ICD devices were more subject to complications—one required immediate generator change and 16% experienced resistance change at the defibrillation coil immediately after the scan. Ten percent of all defibrillator scans resulted in detectable differences in lead performance at 6 months. It should be noted that these protocols are resource intense, requiring device interrogation and programming changes both before and after the scan and cardiac monitoring during the imaging itself [73]. When presented with a patient with an indication for MRI and a CIED, it is recommended that the clinician consider carefully the risk-benefit ratio and assess how likely it is that the specific patient before them will derive benefit from the MRI, as opposed to the approach of, "We always do MRI for this condition."

Conclusion

The evidence base for CIED in chronic cardiac diseases continues to increase, and we can expect to encounter more and more patients with devices in the acute care setting. Understanding the indications, the complications, and how CIEDs affect diagnostic options is crucial to providing optimum care for these patients.

Compliance with Ethical Standards

Conflict of Interest Dr. Hiestand declares no conflicts of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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- • Of importance
- •• Of major importance

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