

# Acute Heart Failure and Implantable Cardiac Devices in the Acute Care Setting

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**Abstract** The use of implantable cardiac devices continues to increase in heart failure patients. The potential roles for these devices can include defibrillation, pacing, resynchronization, and physiologic monitoring. While referral for implantable devices generally occurs in the outpatient setting, an acute care encounter may provide an opportunity for recognition of a patient with an appropriate indication for a device. Therefore, we briefly discuss the indications for an implantable cardiac device in heart failure patients, so as to facilitate recognition and potential referral. We will also discuss the data elements that can potentially be obtained from cardiac devices, as well as how this data can potentially aid in the diagnosis and treatment of acute heart failure.

**Keywords** Acutely decompensated heart failure · Emergency department · Defibrillator · Pacemaker · Sudden cardiac death · Atrial fibrillation

## Introduction

Heart failure hospitalizations continue to increase, with the majority of these encounters beginning in the emergency department (ED) [1, 2]. A rapid, accurate diagnosis and early initiation of appropriate therapy is required for optimal outcomes [3]. Unfortunately, the typical presenting

complaint for acute heart failure, dyspnea, is common to many disease states. It is frequently a challenge for the physician caring for the patient in the acute setting to determine the etiology of the presenting symptoms. Lab, radiology, and clinical findings are frequently insufficiently specific to definitively establish the diagnosis.

An overlooked potential source of additional information in heart failure patients is the implantable cardiac device. In addition to their therapeutic indications, these devices record data that may assist in diagnostic and therapeutic decision making. There are several potential indications for cardiac devices in patients with heart failure; therefore, these devices are frequently encountered in the acute care setting. Other patients with heart failure may have an indication for an implantable cardiac device but have not been recognized or referred for consideration of implantation. In this paper, we will provide an overview of the common indications for implantable cardiac devices in patients with heart failure. The acute care physician may be in a unique position to recognize and refer appropriate patients for potential device therapy. In the second part, we will discuss the data that can be available in implantable devices and describe how these elements can aid in the diagnosis and treatment of acute heart failure. This article does not contain any studies with human or animal subjects performed by any of the authors.

## Indications for Device Therapy

The therapeutic functions of implantable devices fall into two general categories—primary pacing and arrhythmia termination. Both overdrive pacing and defibrillation may be used to terminate malignant ventricular tachyarrhythmias, depending on the capabilities of the implanted

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device. Both atrial and ventricular tachydysrhythmias can affect patients with heart failure. In the general population in the United States, the estimated annual incidence of sudden cardiac death (SCD) is approximately 2 per 1000 people [4]. In patients with ischemic cardiomyopathy with inducible tachydysrhythmias (the highest risk subgroup), that incidence increases markedly to 30 %. Other heart failure patients at high risk are survivors of prior cardiac arrest and those with a left ventricular ejection fraction (LVEF) less than 35 % [5]. In patients with severely impaired LVEF, SCD is responsible for about 50 % of all deaths [6].

Heart failure patients with a previous history of SCD, ventricular tachycardia (VT) or ventricular fibrillation (VF) are at high risk for a repeat event. This is regardless of ventricular function or etiology. Therefore, in these patients, implantable cardioverter-defibrillator (ICD) placement is recommended for secondary prevention—in other words, prevention of a recurrent event [7]. ICD placement is not indicated in patients with end-stage disease in which life-prolonging therapies are not under consideration.

As opposed to secondary prevention, which is defined by an a priori VT/VF/SCD event, primary prevention is the use of an ICD in a patient who is at high risk for a potentially fatal dysrhythmia but has not yet experienced such an occurrence. Multiple trials have shown that device-based therapy confers a survival benefit over medical therapy alone for the primary prevention of SCD in heart failure patients with reduced ejection fraction. This benefit in patients with an LVEF < 35 % has been demonstrated in both ischemic cardiomyopathy (MADIT, MADIT II) [8, 9] and non-ischemic cardiomyopathy (SCD-HeFT) [10] and symptomatic heart failure (New York Heart Association (NYHA) class II–III). Therefore, patients with reduced ejection fraction and symptomatic heart failure should be considered for referral, after stabilization and treatment, for consideration of primary ICD placement.

In addition to arrhythmia termination, cardiac devices may be indicated to manage the beat-to-beat conduction of the failing heart. Slowed ventricular contraction can worsen pre-existing cardiac dysfunction, resulting in deteriorating cardiac function as well as dysfunctional cardiac remodeling. Cardiac resynchronization therapy (CRT) utilizes biventricular leads to establish synchronous depolarization of both ventricles. This modality has been proven to enhance quality of life, decrease heart failure symptoms, and reverse remodeling [11].

The COMPANION trial evaluated CRT in 1520 patients with highly symptomatic (NYHA III or IV) heart failure, reduced LVEF ( $\leq 35\%$ ), and impaired electrical conduction, demonstrated by a QRS  $\geq 120$  ms and PR interval  $\geq 150$  ms [12]. Patients were randomized to CRT, CRT with defibrillator (CRT-D), or optimal medical

therapy. Both CRT arms were associated with a decrease in the combined primary endpoint of death or rehospitalization, and the combination of CRT and defibrillation was associated with a decreased incidence of death when compared to the medical therapy group.

The MIRACLE trial enrolled 453 patients with reduced ejection fraction, prolonged QRS, and symptomatic heart failure [13]. All patients received an implantable cardiac device capable of CRT; however, patients were randomized to 6 months of CRT or no pacing (device implantation without programming turned on). Improvements were seen in the CRT group in NYHA class, quality of life scores, and 6-min walk tests as a measurement of exercise capacity. In addition, the CRT group had fewer days in-hospital during the study period (83 hospital days vs. 363 hospital days), although mortality was similar between the two groups.

CRT benefits patients with milder heart failure (NYHA III) more so than those with severe symptoms [14], and to date has not shown a benefit in patients in the absence of a widened QRS complex. This has been confirmed by the EchoCRT study, which randomized 809 patients with reduced ejection fraction, NYHA III or IV symptoms, QRS  $\leq 130$  ms, and echocardiographic evidence of mechanical dyssynchrony to CRT therapy or device implantation without programming turned on [15]. The trial was stopped early by the data and safety monitoring board due to an increased rate of death in the CRT group, suggesting that CRT is not helpful and may be harmful in patients with a narrow QRS complex. However, in the proper setting, multiple studies have established improvement in quality of life metrics and survival with the use of CRT [12, 13, 16–20].

It is not our intent to define a new standard of care that recognition of implantable device indications and specialist referral should routinely occur in the setting of acute heart failure. However, the utilization of these devices, even in patient populations with a strong evidence base suggesting benefit, is only about 40–50 % [21]. The medical safety net provided by the ED/acute care setting may represent the only opportunity for recognition and referral, especially in medically underserved populations. Even in academic centers, standard practices result in missed chances to provide device-based therapies to at-risk patients [22]. Providers managing patients with acute heart failure should be aware, however, of opportunities to refer patients for therapies that may decrease hospital readmissions and improve the quality of life for our patients.

## Cardiac Device Data

So that implantable devices can perform their therapeutic functions, they must record and process the patient's intrinsic cardiac rhythms. Different devices record modestly

different data elements, although there are some consistent parameters monitored between devices and manufacturers. In addition to cardiac rate, rhythm, and device response data, there are an increasing number of devices that collect advanced data, such as patient activity level, heart rate variability, intracardiac pressure, and intrathoracic impedance. Both conventional and advanced monitoring parameters may assist with the diagnosis and management of the patient with suspected acute heart failure.

### Cardiac Rhythm

Atrial fibrillation frequently coexists with chronic heart failure; even patients presumed to be rhythm controlled can experience clinically silent paroxysms of atrial fibrillation [23]. New onset atrial fibrillation portends worsening long-term survival, and many heart failure patients experience worsening cardiac symptoms with atrial fibrillation [24]. In addition, data suggests that chronic volume overload can produce atrial dysrhythmias, possibly due to electrical irritability resulting from atrial distension [25]. Identification of atrial fibrillation as an index event for decompensated heart failure could emphasize different therapeutic strategies that may not have otherwise been selected, such as initiating rhythm or rate control medications, anticoagulation for stroke prophylaxis, or altering pacemaker programming.

Ventricular tachycardia may also occur without obviously attributable symptoms in the setting of chronic heart failure. Device-based monitoring has demonstrated episodes of heart failure decompensation associated with both sustained and non-sustained ventricular tachyarrhythmias, similar to atrial dysrhythmias [26–28]. The discovery of a high rate of ventricular dysrhythmias in the setting of acute heart failure should prompt a search for electrolyte abnormalities and cardiac ischemia as potential causes of decompensation. Also, should the patient have a device that is not programmed for, or not capable of, defibrillation, the presence of a substantial burden of ventricular arrhythmias should lead to prompt consultation with the patient's electrophysiologist to consider defibrillator therapy.

### Patient Activity

Many devices report a measurement of hours per day that a patient is non-sedentary, using accelerometers within the device itself. Unfortunately, these metrics do not capture the actual degree of exertion. Exercise tolerance deteriorates and physical activity decreases with worsening heart failure [29]. Conversely, improvements in NYHA class are associated with increased daily activity levels and exercise tolerance [30]. Decreased physical activity levels

have been predictive of impending (within 30 days) decompensated heart failure, when monitored in concert with other cardiac device parameters [31].

### Heart Rate Variability

There is a natural variability in the heart rate of healthy individuals due to both responses to physiologic demand as well as diurnal patterns. As the cardiac system is stressed, however, this variance diminishes due to an increase in the sympathetic drive and concomitant decrease in the parasympathetic nervous system output [32]. When the intrinsic sinoatrial rate is sensed by the implanted device, heart rate variability can easily be monitored. The association between heart rate variability and decompensated heart failure was established in a secondary analysis of the MIRACLE study [13]. Those patients that received CRT therapy experienced improved cardiac function that was associated with a substantial increase in heart rate variability [33].

In addition to serving as a marker of response to therapy, heart rate variability can serve as a predictor of adverse outcomes. In a prospective study of 288 patients receiving CRT for symptomatic heart failure with reduced ejection fraction, heart rate variability was significantly lower in patients experiencing death or repeated hospitalization during the study period [29]. The decline in heart rate variability was detectable at a median of 16 days prior to hospitalization for acute heart failure. However, other illnesses that can manifest with an increase in sympathetic tone can also decrease heart rate variability, such as exacerbation of chronic obstructive pulmonary disease [34] or systemic infection [35]. Ongoing, remote monitoring of heart rate variability has been associated with false positive alerts, at approximately 2.4 per patient-year [29].

### Intrathoracic Impedance

Intrathoracic impedance monitors the electrical conductivity between a pulse generator (pacemaker lead) and a sensor (generally the device canister itself). As the amount of tissue fluid increases, electrical resistance, also known as impedance, decreases. Therefore, low intrathoracic impedance is a marker of pulmonary fluid congestion. Intrathoracic impedance correlates with wedge pressures and fluid loss during hospitalization, and begins to drop several days prior to the need for hospitalization [36]. Intrathoracic impedance has been examined as a predictor of impending decompensation in several studies [28, 31, 37–39]. For example, in the FAST study [37], intrathoracic impedance monitoring was substantially more sensitive for

heart failure decompensation than daily weight monitoring (76 vs. 23 %) and had fewer false positives (1.9 vs. 4.3 events per patient-year). Unfortunately to date, no prospective studies have been able to successfully use impedance monitoring in the outpatient setting to avoid hospitalizations for acute heart failure.

However, of potential interest in the acute setting, Small et al. established, in a secondary analysis of a CRT-based impedance monitoring registry, a low likelihood of heart failure hospitalization in patients whose intrathoracic impedance did not drop below a programmed threshold, compared to those patients with multiple threshold events (0.14 hospitalizations per patient-year vs. 0.76 hospitalizations per patient-year) [40]. This suggests that, if prospectively confirmed, in the absence of an impedance drop, a dyspneic patient being evaluated in the acute setting may have a disease process other than acute heart failure that is responsible for their symptoms.

### Hemodynamic Monitoring

At the time of this writing, implantable cardiac devices that directly monitor hemodynamic status are undergoing investigation. The CardioMEMS Heart Failure Sensor (CardioMEMS, Atlanta, Georgia) utilizes a pressure transducer implanted in the pulmonary artery to transmit data wirelessly to a handheld recorder [41]. In the CHAMPION study, a 550-subject prospective randomized trial of protocol-driven modulation of therapy based on daily pulmonary artery pressure readings, heart failure hospitalizations were reduced by 37 % compared to the standard care control group. This improvement in rehospitalization was more marked in the subgroup of patients with preserved ejection fraction (rate ratio 0.50, 95 % CI 0.35–0.70) [42••].

The HeartPOD system (St. Jude Medical, Minneapolis, MN) monitors left atrial pressure via a sensor implanted into the atrial septum [43]. Early observational data indicated that pressure-guided titration of therapy was able to decrease left atrial pressure, ejection fraction, and NYHA class [44]. This device is now being evaluated further in a prospective randomized trial, the LAPTOP-HF study (ClinicalTrials.gov Identifier NCT01121107), with an anticipated completion date in 2016.

The RemonCHF device (Boston Scientific, Natick, MA) measures pulmonary artery pressures by way of a pressure transducer located in the pulmonary artery. A hand-held unit that can be operated by the patient provides on-demand interrogation. To date, this device has undergone observational studies demonstrating agreement with invasive measurement of pulmonary artery pressure [45], but has not been studied as a basis for treatment modification to date.

### Device Data in the Acute Care Setting

Clinical trials of cardiac device data have been directed at outpatient management modulation to prevent patients from decompensating and requiring ED or hospital-based care in the first place. As a result, there is very little data examining the use of device data in the diagnosis and management of suspected acute heart failure in the ED and early hospital stay. Once the patient with an implantable cardiac device presents with symptoms such as dyspnea that may be due to acute heart failure, several challenges exist for treating physician. First, the doctor must determine if the patient's symptoms are truly due to decompensated heart failure. Given that the patient has severe enough heart failure to warrant placement of an implantable device, one might consider the a priori probability of decompensation to be relatively high. However, the use of implantable device data may either serve as valuable confirmation of the presence of acute heart failure or suggest another pathologic process is the etiology of the patient's symptoms. We have previously established in a prospective convenience sample that ED personnel can safely interrogate implantable cardiac devices, and that such data can frequently confirm or rule out suspected diagnoses in the ED [46••]. However, at this time no studies have evaluated the diagnostic performance of implantable cardiac device data in differentiating acute heart failure from other disease entities that may present in similar fashion.

Once the physician has determined that acute heart failure is present, the next step must be to determine how best to treat the patient. The therapies required (diuresis, afterload reduction, and inotropic support) will depend greatly on the clinical severity of symptoms, perfusion status, and the volume status. Although respiratory compromise and systemic perfusion will be fairly obvious with routine exam, volume status may at times be difficult to discern—especially in the obese. Devices that measure volumetric data, such as intrathoracic impedance or direct hemodynamic monitors, may provide insight into the degree of volume overload that is present. This may allow the physician to adequately remove volume while avoiding the complications of overdiuresis and subsequent renal stress.

Finally, in the patient with acute heart failure, it becomes critical to understand the root causes of decompensation in the first place. Examination of the historic data contained within the implantable device may provide illumination of the underlying mechanisms that brought the patient to this state. Rhythm data may indicate paroxysms of atrial fibrillation, which could require pacemaker reprogramming, pharmacologic management, or even A-V nodal ablation to improve hemodynamic function. Given

that abnormalities in heart rate variability, patient activity levels, and fluid accumulation precede clinical decompensation by several days [27, 29, 36, 39], going over temporal data with the patient to evaluate medication, diet, and other lifestyle events may establish a causative link to behaviors that led to the acute decompensation.

Unfortunately, these possibilities, although conceptually sound, have yet to be validated beyond anecdote. As stated previously, the research effort to date has been directed at keeping the patient from requiring acute care in the first place. While this is definitely a worthy goal and will benefit the patient, the truth of the matter remains that over one million hospitalizations for heart failure will occur annually [47•]. There remains a need for research establishing the additive value of basic and advanced implantable device data for the evaluation and management of the patient with suspected acute heart failure. Until such research is established, however, it is certainly reasonable for those of us caring for patients who have this data readily available to evaluate and consider the recorded information in the context of the patient's presentation.

## Conclusion

Chronic heart failure prevalence in the population continues to increase, and it is reasonable to assume that patients with acute heart failure will continue to present to the ED in substantial numbers. Many of these patients will have implantable cardiac devices, which contain untapped information that could potentially assist with the diagnosis and stabilization of the patient with potential acute heart failure. Further research is needed to establish optimum diagnostic thresholds and treatment strategies based on device data in the acute setting.

### Compliance with Ethics Guidelines

**Conflict of Interest** Dr. Brian Hiestand declares that he has no conflicts of interest to declare.

**Human and Animal Rights and Informed Consent** This article contains no studies with human or animal subjects performed by the author.

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