# Catheter Ablation for Premature Ventricular Contractions and Ventricular Tachycardia in Patients with Heart Failure

**Saurabh Kumar · William G. Stevenson · Roy M. John**

**Abstract** Ventricular arrhythmias (VA) are a significant contributor to morbidity and mortality in patients with heart failure (HF). Implantable cardioverter defibrillators are effective in reducing mortality, but do not prevent arrhythmia recurrence. There is increasing recognition that frequent premature ventricular contractions or repetitive ventricular tachycardia may also lead to new onset ventricular dysfunction or deterioration of ventricular function in patients with pre-existing HF. Suppression of the arrhythmia may lead to recovery of ventricular function. Catheter ablation has emerged as a safe and effective treatment option for reducing arrhythmia recurrence and for suppression of PVCs but its efficacy is governed by the nature of the arrhythmias, the underlying HF substrate and the accessibility of the arrhythmia substrates to ablation.

## Introduction

Heart failure (HF) afflicts an estimated 5.1 million Americans over the age of 20 and is a leading cause of cardiac morbidity

and mortality [1]. Ventricular arrhythmias (VAs) are a significant contributor to adverse outcomes in patients with HF. VAs and sudden death predominantly occur in the context of reduced left ventricular (LV) systolic function. They are less common in HF with preserved ejection fraction (EF) except when associated with some genetic, infiltrative or inflammatory cardiomyopathies [2••]. Rarely, frequent idiopathic premature ventricular contractions (PVCs) or repetitive non-sustained ventricular tachycardia can cause a tachycardia-mediated cardiomyopathy [3, 4]. Although, the risk of sudden death is low in such cardiomyopathies, their recognition is important as a potentially reversible cause of heart failure.

Implantable cardioverter defibrillators (ICDs) while highly effective in terminating sustained ventricular arrhythmias and reducing mortality, have no effect on the arrhythmia substrate and recurrent shocks occur in approximately 20 % of patients [5]. Shocks worsen quality of life and are associated with progression of heart failure and increased mortality [5–7]. Beta-blockers and amiodarone are moderately effective in reducing ICD therapies but drug intolerance and serious toxicities of amiodarone necessitate drug cessation in a quarter of patients [8].

Catheter ablation has emerged as an effective treatment for control of frequent ventricular tachycardia (VT) episodes and can be life saving in cases of incessant VT or VT storm [9–15]. For idiopathic VT or PVCs that worsen LV function catheter ablation may offer a more effective option than available drug therapy [16, 17].

## Types of Ventricular Arrhythmias and Related Mechanisms

The approach and outcomes of catheter ablation are dependent on the nature of the arrhythmia substrate. The presenting arrhythmia often suggests the VA mechanism and location

S. Kumar · W. G. Stevenson · R. M. John (✉)
Cardiac Arrhythmia Service, Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, 75 Francis Street, Boston, MA 02115, USA
e-mail: RJOHN2@PARTNERS.ORG

of the arrhythmia substrate. Monomorphic PVCs or VT have the same ventricular activation sequence from beat to beat. The QRS morphology is a clue to its origin. The majority of sustained monomorphic VTs (MVT) are due to scar related re-entry. The scars can remodel over time producing recurrent VT after years of stability. A single large ventricular scar can support multiple VT circuits. Once VT occurs, up to 50 % of patients will experience a recurrence within 2 years [13].

In approximately 8–10 % of patients, MVT originates from the Purkinje system due to automaticity or re-entry [18]. A specific form of VT known as bundle branch reentry is seen typically in patients with structural heart disease, and inter-ventricular conduction delay or left bundle branch block on the sinus rhythm ECG. The reentry wavefront propagates antegradely down the right bundle, then through the septum and retrogradely back up the left bundle to complete the reentrant circuit. VT has a left bundle branch block-like con-figuration, but can also occur in reverse, creating a right bundle branch block-like VT. Although catheter ablation of the right bundle is curative, most patients have other scar-related VTs as well [19].

Idiopathic VT, so termed because of the absence of a clear disease related substrate, often manifests as monomorphic PVCs or repetitive MVT and tends to have a focal origin. They are typically provoked by adrenergic stimulation and triggered automaticity is the likely mechanism. The right and left ventricular outflow tracts are the most common sites followed by regions around a valve an-nulus or within a papillary muscle [20••]. When they are present in association with LV dysfunction, it is often difficult to know if an idiopathic VA is contribut-ing to ventricular dysfunction or is not idiopathic, but a consequence of ventricular disease. A specific form of idiopathic LV VT due to re-entry within the fascicles is responsive to verapamil and amenable to ablation, but is rare in patients with heart failure [21].

Polymorphic VT is characterized by beat-to-beat variation in QRS morphology due to altering ventricular activation sequences and frequently degenerates into ventricular fibrilla-tion. These arrhythmias are usually triggered by acute myo-cardial ischemia, electrolyte disturbances, or repolariza-tion abnormalities such as $QT_c$ prolongation induced by

drugs used in HF patients (e.g., class 3 antiarrhythmic drugs). Patchy areas of myocardial fibrosis, more com-mon in the non-ischemic cardiomyopathies, can theoret-ically lead to spiral wave break up of a re-entrant VT producing a polymorphic VT.

Rarely, monomorphic PVCs of Purkinje origin can trigger ventricular fibrillation (Fig. 1) often presenting with VT storms (three or more VT or ventricular fibrillation [VF] episodes in 24 hours). Although initially described in patients without structural heart disease (idiopathic VF), this can also occur in patients with heart failure, and cardiomyopathies, including amyloidosis [22–24].

## Patient Selection for Ablation and General Considerations

Indications for catheter ablation in ventricular arrhythmias are shown in Table 1. Recent advances in imaging, ablation techniques and hemodynamic support have allowed for safe and effective ablation even in unstable patients. Hence, abla-tion should be an early consideration in the course of recurrent VT that triggers symptoms or ICD shocks. However, patient selection should consider careful balancing of risks and ben-efits. Appropriate expertise and facilities including cardiac surgical back up are needed for optimal success while mini-mizing procedural complications [25].

In patients with VAs, reversible aggravating factors such as ongoing ischemia, drug toxicity, and electrolyte disturbances must be corrected. Pre-procedural optimization of congestion and hypoperfusion is crucial [2••]. Right ventricular (RV) function is a key predictor of prognosis after VT ablation in HF patients [26]. Despite a low LV ejection fraction, patients with recurrent VT with preserved RV function without elevat-ed pulmonary pressures have a favorable prognosis after VT ablation. The presence of RV dysfunction and elevated pul-monary pressures carries a higher mortality risk [26].

Prior to endocardial ablation, ventricular thrombus should be excluded by echocardiography. The presence of concomi-tant atrial fibrillation without sufficient period (>4 consecutive weeks) of therapeutic anticoagulation, must prompt consider-ation of transesophageal echocardiogram to exclude a left atrial appendage thrombus, as cardioversion of VT during



**Fig. 1** Monomorphic PVCs triggering recurrent idiopathic ventricular fibrillation (VF). Telemetric trace of a 34 year-old male with no detectable structural heart disease or ECG abnormalities, is shown. Following successful resuscitation from an out-of-hospital cardiac arrest, recurrent VF was refractory to intravenous amiodarone and lidocaine. Short-coupled monomorphic PVCs are seen prior to onset of ventricular fibril-lation (*arrows*). At electrophysiological study, earliest activation during PVCs was detected along Purkinje fibres (premature beats preceded by Purkinje potentials) in the right ventricular anterior septum. Ablation to abolish PVC prevented recurrence of VF.

**Table 1** Indications for catheter ablation for ventricular arrhythmias

In the presence of structural heart disease

  Catheter ablation is recommended for:

    1. Symptomatic sustained monomorphic VT including those treated by an implanted defibrillator that recurs despite antiarrhythmic drugs or when drugs are not tolerated

    2. Control of incessant monomorphic VT or VT storm that is not due to a reversible cause

    3. Frequent PVC or VT that is deemed to be causing ventricular dysfunction

    4. Bundle branch re-entrant VT or inter-fascicular VT

    5. Recurrent polymorphic VT of VF refractory to antiarrhythmic drugs when there is a suspected trigger that can be targeted for ablation

  Catheter ablation should be considered for:

    1. Patients with recurrent monomorphic VT and LVEF > 30–35 % even if they have not failed prior antiarrhythmic therapy or as an alternative to long-term amiodarone therapy

In the absence of structural heart disease

  Catheter ablation is recommended for:

    1. Symptomatic monomorphic VT

    2. Drug refractory monomorphic VT or when antiarrhythmic drugs are not tolerated

    3. Recurrent sustained polymorphic VT or VF that is unresponsive to antiarrhythmic medications when there is a suspected trigger that can be targeted for ablation

  Contraindications to catheter ablation for ventricular arrhythmias

    1. Presence of a mobile ventricular thrombus (epicardial ablation or alcohol ablation should be considered)

    2. For asymptomatic PVCs or non sustained VT that is not causing or contributing to ventricular dysfunction

    3. VT due to transient reversible cause or torsade de Pointes VT related to prolongation of the QT interval

*VT* ventricular tachycardia, *PVC* premature ventricular complexes, *VF* ventricular fibrillation; *LVEF* left ventricular ejection fraction

(Adapted from: Aliot EM, Stevenson WG, Almendral-Garrote JM, et al. EHRA/HRS Expert Consensus on Catheter Ablation of Ventricular Arrhythmias: developed in a partnership with the European Heart Rhythm Association (EHRA), a Registered Branch of the European Society of Cardiology (ESC), and the Heart Rhythm Society (HRS); in collaboration with the American College of Cardiology (ACC) and the American Heart Association (AHA). Europace 2009;11:771–817) [19]

ablation may revert the patient to sinus rhythm with heightened risk of thromboembolic events. General anesthesia is increasingly used in our laboratory during ablations for scar related VT. It has the advantage of keeping the patient still during long procedures, facilitates stable electro-anatomic mapping and is preferred when epicardial access is necessary [27••]. Hemodynamic support such as an intra-aortic balloon pump, or ventricular assist device may permit longer periods of mapping during hemodynamically unstable VTs by maintaining end-organ perfusion, [28] but are associated with increased risk of vascular complications (12 %) [29].

As most patients with systolic heart failure will have an ICD, the majority of VAs are documented on intracardiac electrograms (EGMs) recorded from the pacing and defibrillating electrodes. These EGMs can provide clues to the nature of the arrhythmia and are important to exclude the possibility of unnecessary ICD therapy due to atrial fibrillation or sinus tachycardia at rates above the VT detection rate. Long-short intervals from some pacing algorithms, such as in the minimal ventricular pacing mode, initiate VT in some patients and can be corrected by reprogramming [30]. The presence of a uniform PVC initiating VF can be a clue to triggers from the Purkinje fibers (Fig. 1). Intracardiac EGMs also allow documentation of the VT cycle length and the response to anti-tachycardia pacing may be helpful in assessing the nature of the arrhythmias.

Whenever possible, a 12 lead ECG recording of spontaneous VT (termed a clinical VT) should be obtained as the QRS morphology suggests the VT origin, which facilitates planning of the initial mapping approach. In lead $V_1$ a right bundle branch block-like configuration indicates likely LV origin, and a left bundle branch block-like configuration indicates a likely septal or RV origin. Superior and inferior frontal plane axes indicate exit from the inferior or anterior walls respectively. Dominant R or S waves in $V_3/V_4$ indicate a basal or apical origin respectively [20••, 27••]. These guidelines are not always reliable, particularly in the presence of extensive scars. In non-ischemic cardiomyopathy, QS complexes in lead I during VT often indicate that VT arises from a circuit in the subepicardium of the basal lateral LV. These criteria are not reliable in ischemic heart disease [27••].

## Approaches to Ablation in Structural Heart Disease

Programmed ventricular stimulation should be performed first to induce the arrhythmias to unequivocally confirm the diagnosis, assess the QRS morphology, and define an endpoint for ablation. If non–inducible or the induced arrhythmia is hemodynamically unstable, a substrate based mapping and ablation approach is generally used. If the VT is inducible and hemodynamically tolerated, the reentrant circuits can be characterized using mapping techniques and ablation applied to terminate VT, providing confirmation that the ablation target is causing the VT.

In substrate-based approaches to mapping and ablation, scar regions are defined during sinus or paced rhythm by creating voltage maps of the area of interest. There is a close correlation of endocardial and epicardial scar with low EGM voltage (bipolar EGM less than 1.5 mV on the endocardium and 1.0 mV for the epicardium) [31]. Endocardial bipolar EGMs do not reliably identify intramural or epicardial scar, but these can often be detected from analysis of unfiltered or minimally filtered unipolar EGMs, as these have a broader "field of view" [32, 33].

In addition to defining the area of low voltage, specific features of recorded EGMs allow definition of the reentry substrate. Multicomponent, fractionated EGMs, split or late potentials (occurring after the QRS complex) indicate asynchronous activation of myocyte bundles with intervening fibrosis causing slow conduction [34]. Pacing in the area of interest identifies excitable tissue in the scar, areas of slow conduction characterized by long stimulus to QRS duration and a paced QRS morphology that approximates the morphology of the VT suggesting close proximity to the VT circuit exit [35]. Ablation is performed during stable sinus or paced rhythm, targeting these abnormal areas. Our approach is to render the tissue electrically un-excitable to pacing.

Mapping during an induced or spontaneous clinical VT offers the advantage of potentially identifying a critical isthmus in a re-entrant circuit where ablation can abolish VT with a limited area of ablation. During VT critical isthmuses often show EGMs that precede surface QRS (presystolic or diastolic) and pacing at a rate slightly faster than the VT rate can often entrain the tachycardia, advancing it to the pacing rate without changing its QRS morphology. Furthermore the interval from the last paced complex to the first return cycle (post pacing interval) approximates the tachycardia cycle length. Analysis of these features can be used to refine the ablation target site location. Ablation based on these criteria is likely to terminate VT although in large scars, the isthmus maybe broad and multiple ablations may be required to render VT non-inducible [36]. Multiple inducible VTs due to multiple reentry circuits are also common, requiring ablation in more than one area or over a region. Substrate guided and VT mapping approaches are often combined to address these scar related VTs.

In approximately 5 to 15 % of patients with coronary artery disease, and more than a third of patients with non-ischemic cardiomyopathy, scar-related VT circuits are located in the subepicardium and cannot be ablated from the endocardium. In the absence of pericardial adhesions from prior cardiac surgery or pericarditis the epicardium can be accessed percutaneously for mapping and ablation. Epicardial ablation is usually preceded by coronary angiography to make sure a coronary vessel does not overly the ablation target, with risk of coronary occlusion due to ablation [27••].

If clinical VT is inducible at the beginning of the procedure, non-inducibility of such VT should be the minimum procedural endpoint [27••]. Abolition of all inducible VTs has been associated with a lower risk of VT recurrence and cardiac mortality in some, but not all studies [11, 27••, 37, 38]. Location of arrhythmia substrate and the underlying disease state affects the likelihood of success. Ablation is most successful in conditions where potential re-entry channels can be defined on the endocardium or epicardium (e.g., myocardial infarction, arrhythmogenic right ventricular cardiomyopathy) but is less successful for intramural circuits or epicardial circuits in close proximity to coronary arteries [20••, 39–41]. Ablation failure is often due to inability to create transmural, durable lesions or failure to reach intramural substrate (e.g, intraventricular septum), or protection of the VT substrate by close proximity to coronary arteries or the left phrenic nerve, or by epicardial fat [20••].

Transcoronary ethanol ablation has been used in selected patients when catheter ablation fails. A coronary vessel supplying the VT substrate is identified for administration of absolute ethanol. Limitations include failure to identify a coronary target, potential for damage to large areas of myocardium and administration of intravenous contrast load in HF patients who may have pre-existing renal impairment [42]. Surgical cryoablation is also an option when catheter ablation fails and may allow separation of an overlying coronary artery from epicardial substrate and dissection through epicardial fat allowing successful ablation [43, 44].

## VT Ablation in Specific Substrates

### Ischemic Cardiomyopathy

Ventricular scar is present from prior myocardial infarction, with ongoing remodeling, creating the anatomic substrate for scar-related, often multiple, re-entrant VTs [19]. Challenges for post infarct VT ablation include: (i) inducibility of multiple VTs (on average 3/patient due to separate reentry circuits or a shared area of slow conduction with variable exits) [15, 19, 45]; (ii) likelihood of a broad reentry circuit isthmus (>2–3 cm) [46]; (iii) hemodynamically unstable VTs [19]. Hence substrate mapping is increasingly preferred to activation and entrainment mapping alone for treatment.

Multicenter studies of catheter ablation in ischemic cardiomyopathy show that at least one VT is abolished in 72–96 % of patients and all inducible VTs eliminated in 38–72 % of patients with 50–88 % of patients remaining VT free over a mean follow up of >12 months and 30–100 % continuing on previously ineffective AADs [19, 46–49]. VT episodes are markedly reduced in the majority of patients [15]. Procedural mortality is approximately 3 % with 1 year mortality of 9 to 18 % with most deaths attributed to recurrent VAs or HF [15, 19, 20••, 27••].

Early performance of catheter ablation to prevent VT after initial arrhythmia presentation in ischemic cardiomyopathy patients has been studied in two randomized trials. Catheter ablation with ICD compared to ICD alone reduced the likelihood of recurrent VT, increased time to first VT recurrence, and showed a trend to reduction in likelihood of electrical storm, but the studies were too small to assess an effect on mortality [13, 14].

## Non-ischemic Cardiomyopathy

Non-ischemic cardiomyopathy (NICM) is a heterogeneous group of diseases including unknown, genetic LV cardiomyopathies (lamin A/C, Titin mutations), arrhythmogenic RV cardiomyopathies (ARVC), inflammatory disease (sarcoid heart disease, myocarditis) and hypertrophic cardiomyopathy (HCM). Sustained monomorphic VT is due to scar mediated re-entry in over 80 % of patients with the remainder being focal in origin or due to bundle branch re-entry. Key differences compared to ischemic cardiomyopathy include: (i) generally smaller scars with multiple re-entrant VTs despite small scar; (ii) predilection for anatomical location around the valve annuli or septum; (iii) less frequent transmural scars; (iv) more frequent intramural scars; (v) extensive epicardial scarring that may occur in the presence of normal endocardium or extend beyond the region of endocardial scarring [19, 50–53]. A recent study found basal anteroseptal or inferolateral scar accounting for 89 % of arrhythmogenic substrate in patients with NICM and sustained MVT [54]. The former can be targeted endocardially whereas the latter frequently require an epicardial approach [54].

Catheter ablation for VT in the non ischemic cardiomyopathies is less well studied and VT recurrence rates are higher than in the ischemic patients, occurring in 50–60 % in short term follow up (up to 1 year), although VT burden can be significantly reduced [40, 55]. Complete non-inducibility can be achieved in 38–67 % of patients [39, 40, 55]. Persistent inducibility for VT predicts future recurrence [40, 55].

In ARVC, fibrofatty replacement progresses from the subepicardial layer to the endocardium particularly affecting the free wall of the RV along the tricuspid annulus and in the outflow tract. Epicardial scar area is often larger than endocardial scar [56]. Thus epicardial ablation is frequently required either as primary target or after failed endocardial ablation [19, 57]. Endocardial unipolar mapping with a cut off <5.5 mV identifies epicardial scar [33]. Ablation is often directed from a valve annulus to scar transecting an isthmus has been used [58, 59]. With the use of epicardial ablation freedom from VT is achieved in 77–89 % of patients, but late recurrences can occur due to the progressive nature of the disease [19, 27••, 57, 58] .

SMVT is rare in patients with HCM, and usually due to scar-mediated re-entry [60]. Combined epicardial and endocardial mapping and ablation are feasible and can reduce spontaneous VTs but data on outcome is limited [60–62].

### Ablation for VT Storm

Electrical or VT storm is associated with a high mortality. A meta-analysis of all VT storm studies found a mortality of 17 % a little over 1 year follow up with heart failure accounting for ~ two-thirds of all deaths post ablation [10]. Catheter ablation controls VT in >90 % of patients with 74–92 % remaining free of incessant VT or recurrent VT storm [10, 12, 15]. Single episodes of VT recurrences occur in a third of patients [19]. Sympathetic denervation or renal artery denervation are emerging potential therapies [63]. In rare patients, recurrent VT is provoked by unifocal PVCs from the Purkinje fibers, outflow tracts, or papillary muscles that can be targeted for ablation (see above).

### Cardiomyopathy Related to Idiopathic PVCs or VT

Very frequent PVCs or non-sustained VT can induce a cardiomyopathy that can be reversed with either pharmacological suppression or catheter ablation [3, 4, 64••]. Such arrhythmias may also exacerbate pre-existing left ventricular function and is a cause of loss of effective biventricular pacing in patients dependent on cardiac re-synchronization therapy [65]. The diagnosis of PVC cardiomyopathy is one of exclusion and often retrospective based on recovery of LV function after control of the arrhythmia.

The exact mechanism of PVC-induced cardiomyopathy is unclear. Ventricular dyssynchrony, alterations in intracellular calcium handling, changes in heart rate dynamics, hemodynamic parameters as well as changes in myocardial and peripheral autonomic function have been postulated [64••]. The frequency of PVCs correlates with the severity of left ventricular dysfunction at the time of initial presentation [66–69]. However, in some patients, a high PVC burden does not impair LV function whereas in others a lower PVC burden may do so. The lowest PVC burden resulting in a reversible cardiomyopathy was noted to be 10 % in one study [69]. This relationship is likely to be complex however, not appreciated by a single "cut off" value. PVC duration (≥140 ms [70] to 150 ms [71]), an epicardial site of origin, [71] PVC coupling intervals≤600 ms, [72] PVC interpolation, [73] long history of palpitations (>60 months), and the complete absence of symptoms [74] have all been associated with higher likelihood of LV dysfunction.

Most idiopathic PVCs originate from the ventricular outflow tracts and tend to be monomorphic [70]. Myocardial extension for a variable distance above the aortic and pulmonic valves is thought to form the necessary electrophysiological substrate for idiopathic PVCs. An autopsy study showed that extensions are most common in the right coronary cusp (55 %) than the left coronary cusp (24 %) and non-coronary cusp (<1 %). In contrast, myocardial extensions above the pulmonic valves are evenly distributed (45 to 60 %) [75]. Other sites for idiopathic PVCs include the papillary muscles, areas close to the AV valves and from the fascicles of the conduction system. An epicardial peri-venous focus has been identified in some patients and may need mapping in the great cardiac vein or the anterior inter-ventricular branch [76].

Beta blockers and calcium channel blockers have a modest effect in suppressing PVCs. Antiarrhythmic drugs such as flecainide, mexilitine [77, 78] and amiodarone are more effective but long term use is limited by side effects and flecainide is avoided in the presence of heart disease or depressed ventricular function. Hence, catheter ablation is an attractive option. A recent randomized study of patients with frequent PVCs from the RV outflow tract found a greater decrease in burden of PVCs compared to drug therapy although LV function improved in both groups [16]. Whether PVC ablation is superior to drugs in reversing LV dysfunction is unknown.

The ECG can help localize site of origin, with most having a left bundle branch morphology with precordial transition at $V_3$ and inferiorly directed axis, consistent with a RV outflow tract origin. A prominent R wave in $V_1$ or early transition before $V_3$ suggests a left ventricular outflow tract origin. However, the complex anatomy of the outflow tract and variable distance of myocardial extensions above the valves precludes precise localization from ECG criteria [75]. Systemic mapping of the RV outflow tract pulmonary artery, great cardiac/anterior interventricular vein via the coronary sinus followed by the aortic root and cusps, and LV outflow tract is often required [20••]. Papillary muscle VTs tend to have a broader QRS (typically greater than 150 milliseconds), present a monophasic R or qR in $V_1$, and Q waves tend to be absent. Axis is superiorly directed for those originating from the posterior LV papillary muscle and inferiorly directed when the origin is the anterior papillary muscle [79, 80].

Catheter ablation is successful in achieving greater than 80 % reduction in ectopic activity in 70–90 % of patients, with highest success rates for arrhythmias arising from the RV outflow tract [4, 64••, 70]. The efficacy of ablation for the less common foci such as the LV outflow tract, are not well characterized. A common reason for failure is the inability to induce the arrhythmia for adequate mapping.

In our lab, we place ECG leads on arrival to the lab in an effort to document the 12-lead morphology and avoid excessive administration of lidocaine for local anesthesia to prevent systemic absorption and suppression of PVCs. In the absence of spontaneous arrhythmia, aggressive provocation with isoproterenol, epinephrine, or atropine with or without burst pacing is used. Documentation of the PVC morphology aids approximate localization with pace-mapping although activation mapping is more accurate especially in the aortic root where preferential conduction can yield misleading information with pace maps [81]. Combined bipolar and unipolar recordings are utilized for mapping earliest activation sites. At the site of origin, bipolar recording typically precede QRS onset by 30 milliseconds or more. The corresponding unipolar signal displays the earliest rapid negative deflection. The use of a three dimensional mapping system and intra-cardiac echocardiography is particularly valuable in defining anatomy for idiopathic VA originating in the LV outflow region, coronary cusps, and papillary muscles. Coronary angiography may be necessary to confirm a safe distance (>5 mm) when the ablation target is in the aortic root or great cardiac vein.

There is limited data on the time course of recovery of LV function after ablation of PVCs. One study showed that 68 % of patients recover within 4 months with 32 % requiring a mean of 12+/−9 months (range 5−45 months) for recovery [82]. Early improvement (within 1 week) predicted near complete reversibility [83].

In patients with heart disease PVCs may be idiopathic or intimately related to an abnormal substrate, such as within an infarct scar or at its border, and the site of origin may correspond to the exit site of an inducible reentrant VT [84, 85].

## Ventricular Arrhythmias in Patients with Ventricular Assist Devices

Implantable ventricular assist devices (VADs) are increasingly used in patients with advanced HF as destination therapy, bridge to recovery, bridge to cardiac transplant [86] or in patients with refractory VT [87]. Ventricular arrhythmias occur in 22–52 % of patients during left VAD (LVAD) support [88••]. The presence of VAs prior to LVAD placement is a predictor of post-LVAD arrhythmias [89]. Intravascular volume depletion or high LVAD flow rates (creating suction events) can trigger excess mechanical stimulation and nonsustained VT that may precipitate sustained VT [90]. VAs can cause a drop in VAD output, with concern for increased risk of VAD thrombosis, reduced likelihood of surviving a subsequent cardiac transplant, and precipitation of right ventricular failure (if incessant) or death [89, 91]. Catheter ablation in VAD patients poses unique challenges. In patients with pre-existing ventricular arrhythmias, pre-operative mapping of the arrhythmia origin with a view to intra-operative cryoablation is a reasonable consideration. In one study, such an approach with lesions created to connect scar to fixed anatomic borders in the region of the clinical ventricular arrhythmia reduced the risk of VAs with reduction in re-intubation rates and hospitalization times [92]. Percutaneous catheter ablation in LVAD patients may be complicated: (i) vascular access may be difficult due to loss of systemic pulsatile flow in continuous flow VADs; (ii) retrograde aortic access may be limited due to restricted aortic valve opening with potential of valve calcification; (iii) catheter manipulation may be difficult in the collapsed LV; (iv) there may be a tendency of the catheter to be drawn into the inflow cannula from suction effects with risk of entrapment or damage to the cannula (v) progressive RV failure due to long activation mapping during VT. Despite aforementioned risks, catheter ablation can be safe and effective when performed in experienced hands [93]. Single center experience report success rates of 85 % albeit, with a

recurrence rate of 33 % within 6 months, often requiring repeat procedures [94].

## Conclusion

Patients with HF represent a heterogeneous population with various structural and electrical substrates for PVCs and VT. Ventricular arrhythmias are an important cause of morbidity and mortality in the HF population. AADs are limited in their efficacy and catheter ablation has emerged as a useful therapy. HF remains the major cause of mortality after VT ablation, consistent with the concern that VT is an indication of disease progression, warranting attention to heart failure therapies. The efficacy of catheter ablation for SMVT varies with underlying disease etiology, the pattern and distribution of scar, proximity of scar to critical epicardial structures and accessibility of scar areas for catheter ablation. Novel technologies are hoped to improve future outcomes. PVC-induced cardiomyopathy is important to recognize as a reversible condition. Further work is needed to elucidate the mechanism of PVC-induced cardiomyopathy and the relationship between PVC burden and development of cardiomyopathy.

### Compliance with Ethics Guidelines

**Conflict of Interest**    Saurabh Kumar declares that he has no conflict of interest.

William G. Stevenson has a patent for needle ablation.

Roy M. John reports personal fees from St. Jude Medical and Medtronic.

**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.

## References

Papers of particular interest, published recently, have been highlighted as:
•• Of major importance

1. Go AS, Mozaffarian D, Roger VL, et al. Heart disease and stroke statistics–2014 update: a report from the American Heart Association. Circulation. 2014;129:e28–e292.
2.•• John RM, Stevenson LW. Ventricular arrhythmias in heart failure. In: Zipes DP, Jalife J, editors. Cardiac electrophysiology: from cell to bedside. 6th ed. Philadelphia: WB Saunders; 2013. *This book chapter highlights the important points in treating patients with heart failure who develop ventricular tachycardia.*
3. Duffee DF, Shen WK, Smith HC. Suppression of frequent premature ventricular contractions and improvement of left ventricular function in patients with presumed idiopathic dilated cardiomyopathy. Mayo Clin Proc. 1998;73:430–3.
4. Sarrazin JF, Labounty T, Kuhne M, et al. Impact of radiofrequency ablation of frequent post-infarction premature ventricular complexes on left ventricular ejection fraction. Heart Rhythm. 2009;6: 1543–9.
5. Poole JE, Johnson GW, Hellkamp AS, et al. Prognostic importance of defibrillator shocks in patients with heart failure. N Engl J Med. 2008;359:1009–17.
6. Powell BD, Saxon LA, Boehmer JP, et al. Survival after shock therapy in implantable cardioverter-defibrillator and cardiac resynchronization therapy-defibrillator recipients according to rhythm shocked. The ALTITUDE survival by rhythm study. J Am Coll Cardiol. 2013;62:1674–9.
7. Kamphuis HC, de Leeuw JR, Derksen R, Hauer RN, Winnubst JA. Implantable cardioverter defibrillator recipients: quality of life in recipients with and without ICD shock delivery: a prospective study. Europace. 2003;5:381–9.
8. Connolly SJ, Dorian P, Roberts RS, et al. Comparison of beta-blockers, amiodarone plus beta-blockers, or sotalol for prevention of shocks from implantable cardioverter defibrillators: the OPTIC Study: a randomized trial. JAMA. 2006;295:165–71.
9. Mallidi J, Nadkarni GN, Berger RD, Calkins H, Nazarian S. Meta-analysis of catheter ablation as an adjunct to medical therapy for treatment of ventricular tachycardia in patients with structural heart disease. Heart Rhythm. 2011;8:503–10.
10. Nayyar S, Ganesan AN, Brooks AG, Sullivan T, Roberts-Thomson KC, Sanders P. Venturing into ventricular arrhythmia storm: a systematic review and meta-analysis. Eur Heart J. 2013;34: 560–71.
11. Calkins H, Epstein A, Packer D, et al. Catheter ablation of ventricular tachycardia in patients with structural heart disease using cooled radiofrequency energy: results of a prospective multicenter study. Cooled RF Multi Center Investigators Group. J Am Coll Cardiol. 2000;35:1905–14.
12. Carbucicchio C, Santamaria M, Trevisi N, et al. Catheter ablation for the treatment of electrical storm in patients with implantable cardioverter-defibrillators: short- and long-term outcomes in a prospective single-center study. Circulation. 2008;117:462–9.
13. Kuck KH, Schaumann A, Eckardt L, et al. Catheter ablation of stable ventricular tachycardia before defibrillator implantation in patients with coronary heart disease (VTACH): a multicentre randomised controlled trial. Lancet. 2010;375:31–40.
14. Reddy VY, Reynolds MR, Neuzil P, et al. Prophylactic catheter ablation for the prevention of defibrillator therapy. N Engl J Med. 2007;357:2657–65.
15. Stevenson WG, Wilber DJ, Natale A, et al. Irrigated radiofrequency catheter ablation guided by electroanatomic mapping for recurrent ventricular tachycardia after myocardial infarction: the multicenter thermocool ventricular tachycardia ablation trial. Circulation. 2008;118:2773–82.
16. Ling Z, Liu Z, Su L, et al. Radiofrequency ablation vs. antiarrhythmic medication for treatment of ventricular premature beats from the right ventricular outflow tract: a prospective randomized study. Circ Arrhythm Electrophysiol. 2014. doi:10.1161/CIRCEP.113. 000805.
17. Zhong L, Lee YH, Huang XM, et al. Relative efficacy of catheter ablation vs antiarrhythmic drugs in treating premature ventricular contractions: a single-center retrospective study. Heart Rhythm. 2014;11:187–93.
18. Lopera G, Stevenson WG, Soejima K, et al. Identification and ablation of three types of ventricular tachycardia involving the his-purkinje system in patients with heart disease. J Cardiovasc Electrophysiol. 2004;15:52–8.
19. Aliot EM, Stevenson WG, Almendral-Garrote JM, et al. EHRA/HRS Expert Consensus on Catheter Ablation of Ventricular Arrhythmias: developed in a partnership with the European Heart Rhythm Association (EHRA), a Registered

Branch of the European Society of Cardiology (ESC), and the Heart Rhythm Society (HRS); in collaboration with the American College of Cardiology (ACC) and the American Heart Association (AHA). Europace. 2009;11:771–817.

20.•• Stevenson WG. Current treatment of ventricular arrhythmias: state of the art. Heart Rhythm. 2013;10:1919–26. *A concise review of the state of the art in the management of ventricular arrhythmias.*

21. Nogami A, Naito S, Tada H, et al. Verapamil-sensitive left anterior fascicular ventricular tachycardia: results of radiofrequency ablation in six patients. J Cardiovasc Electrophysiol. 1998;9:1269–78.

22. Marrouche NF, Verma A, Wazni O, et al. Mode of initiation and ablation of ventricular fibrillation storms in patients with ischemic cardiomyopathy. J Am Coll Cardiol. 2004;43:1715–20.

23. Haissaguerre M, Shah DC, Jais P, et al. Role of Purkinje conducting system in triggering of idiopathic ventricular fibrillation. Lancet. 2002;359:677–8.

24. Haissaguerre M, Shoda M, Jais P, et al. Mapping and ablation of idiopathic ventricular fibrillation. Circulation. 2002;106:962–7.

25. Tokuda M, Kojodjojo P, Epstein LM, et al. Outcomes of cardiac perforation complicating catheter ablation of ventricular arrhythmias. Circ Arrhythm Electrophysiol. 2011;4:660–6.

26. Ujeyl A, Inada K, Hillmann K, et al. Right heart function prediction of outcome in heart failure patients after catheter ablation for recurrent ventricular tachycardia. JACC Heart Fail. 2013;1:281–9.

27.•• Wissner E, Stevenson WG, Kuck KH. Catheter ablation of ventricular tachycardia in ischaemic and non-ischaemic cardiomyopathy: where are we today? A clinical review. Eur Heart J. 2012;33:1440–50. *A comprehensive review of the current management of ventricular arrhythmias.*

28. Miller MA, Dukkipati SR, Chinitz JS, et al. Percutaneous hemodynamic support with Impella 2.5 during scar-related ventricular tachycardia ablation (PERMIT 1). Circ Arrhythm Electrophysiol. 2013;6:151–9.

29. Reddy YM, Chinitz L, Mansour M, et al. Percutaneous left ventricular assist devices in ventricular tachycardia ablation: a multicenter experience. Circ Arrhythm Electrophysiol. 2014. doi:10.1161/CIRCEP.113.000548.

30. Sweeney MO, Ruetz LL, Belk P, Mullen TJ, Johnson JW, Sheldon T. Bradycardia pacing-induced short-long-short sequences at the onset of ventricular tachyarrhythmias: a possible mechanism of proarrhythmia? J Am Coll Cardiol. 2007;50:614–22.

31. Marchlinski FE, Callans DJ, Gottlieb CD, Zado E. Linear ablation lesions for control of unmappable ventricular tachycardia in patients with ischemic and nonischemic cardiomyopathy. Circulation. 2000;101:1288–96.

32. Hutchinson MD, Gerstenfeld EP, Desjardins B, et al. Endocardial unipolar voltage mapping to detect epicardial ventricular tachycardia substrate in patients with nonischemic left ventricular cardiomyopathy. Circ Arrhythm Electrophysiol. 2011;4:49–55.

33. Polin GM, Haqqani H, Tzou W, et al. Endocardial unipolar voltage mapping to identify epicardial substrate in arrhythmogenic right ventricular cardiomyopathy/dysplasia. Heart Rhythm. 2011;8:76–83.

34. de Bakker JM, Wittkampf FH. The pathophysiologic basis of fractionated and complex electrograms and the impact of recording techniques on their detection and interpretation. Circ Arrhythm Electrophysiol. 2010;3:204–13.

35. Nayyar S, Wilson L, Ganesan AN, et al. High-density mapping of ventricular scar: a comparison of ventricular tachycardia (VT) supporting channels with channels that do not support VT. Circ Arrhythm Electrophysiol. 2014;7:90–8.

36. Stevenson WG, Khan H, Sager P, et al. Identification of reentry circuit sites during catheter mapping and radiofrequency ablation of ventricular tachycardia late after myocardial infarction. Circulation. 1993;88:1647–70.

37. Della Bella P, Riva S, Fassini G, et al. Incidence and significance of pleomorphism in patients with postmyocardial infarction

ventricular tachycardia. Acute and long-term outcome of radiofrequency catheter ablation. Eur Heart J. 2004;25:1127–38.

38. Della Bella P, Baratto F, Tsiachris D, et al. Management of ventricular tachycardia in the setting of a dedicated unit for the treatment of complex ventricular arrhythmias: long-term outcome after ablation. Circulation. 2013;127:1359–68.

39. Tokuda M, Tedrow UB, Kojodjojo P, et al. Catheter ablation of ventricular tachycardia in nonischemic heart disease. Circ Arrhythm Electrophysiol. 2012;5:992–1000.

40. Piers SR, Leong DP, van Huls van Taxis CF, et al. Outcome of ventricular tachycardia ablation in patients with nonischemic cardiomyopathy: the impact of noninducibility. Circ Arrhythm Electrophysiol. 2013;6:513–21.

41. Desjardins B, Yokokawa M, Good E, et al. Characteristics of intramural scar in patients with nonischemic cardiomyopathy and relation to intramural ventricular arrhythmias. Circ Arrhythm Electrophysiol. 2013;6:891–7.

42. Tokuda M, Sobieszczyk P, Eisenhauer AC, et al. Transcoronary ethanol ablation for recurrent ventricular tachycardia after failed catheter ablation: an update. Circ Arrhythm Electrophysiol. 2011;4:889–96.

43. Sarkozy A, Tokuda M, Tedrow UB, et al. Epicardial ablation of ventricular tachycardia in ischemic heart disease. Circ Arrhythm Electrophysiol. 2013;6:1115–22.

44. Anter E, Hutchinson MD, Deo R, et al. Surgical ablation of refractory ventricular tachycardia in patients with nonischemic cardiomyopathy. Circ Arrhythm Electrophysiol. 2011;4:494–500.

45. Bogun F, Li YG, Groenefeld G, et al. Prevalence of a shared isthmus in postinfarction patients with pleiomorphic, hemodynamically tolerated ventricular tachycardias. J Cardiovasc Electrophysiol. 2002;13:237–41.

46. de Chillou C, Lacroix D, Klug D, et al. Isthmus characteristics of reentrant ventricular tachycardia after myocardial infarction. Circulation. 2002;105:726–31.

47. Soejima K, Stevenson WG, Maisel WH, Sapp JL, Epstein LM. Electrically unexcitable scar mapping based on pacing threshold for identification of the reentry circuit isthmus: feasibility for guiding ventricular tachycardia ablation. Circulation. 2002;106:1678–83.

48. Soejima K, Suzuki M, Maisel WH, et al. Catheter ablation in patients with multiple and unstable ventricular tachycardias after myocardial infarction: short ablation lines guided by reentry circuit isthmuses and sinus rhythm mapping. Circulation. 2001;104:664–9.

49. Arenal A, Glez-Torrecilla E, Ortiz M, et al. Ablation of electrograms with an isolated, delayed component as treatment of unmappable monomorphic ventricular tachycardias in patients with structural heart disease. J Am Coll Cardiol. 2003;41:81–92.

50. Nazarian S, Bluemke DA, Lardo AC, et al. Magnetic resonance assessment of the substrate for inducible ventricular tachycardia in nonischemic cardiomyopathy. Circulation. 2005;112:2821–5.

51. Soejima K, Stevenson WG, Sapp JL, Selwyn AP, Couper G, Epstein LM. Endocardial and epicardial radiofrequency ablation of ventricular tachycardia associated with dilated cardiomyopathy: the importance of low-voltage scars. J Am Coll Cardiol. 2004;43:1834–42.

52. Hsia HH, Callans DJ, Marchlinski FE. Characterization of endocardial electrophysiological substrate in patients with nonischemic cardiomyopathy and monomorphic ventricular tachycardia. Circulation. 2003;108:704–10.

53. Nakahara S, Tung R, Ramirez RJ, et al. Characterization of the arrhythmogenic substrate in ischemic and nonischemic cardiomyopathy implications for catheter ablation of hemodynamically unstable ventricular tachycardia. J Am Coll Cardiol. 2010;55:2355–65.

54. Piers SR, Tao Q, van Huls van Taxis CF, Schalij MJ, van der Geest RJ, Zeppenfeld K. Contrast-enhanced MRI-derived scar patterns

and associated ventricular tachycardias in nonischemic cardiomyopathy: implications for the ablation strategy. Circ Arrhythm Electrophysiol. 2013;6:875–83.

55. Dinov B, Fiedler L, Schonbauer R, et al. Outcomes in catheter ablation of ventricular tachycardia in dilated nonischemic cardiomyopathy compared with ischemic cardiomyopathy: results from the Prospective Heart Centre of Leipzig VT (HELP-VT) Study. Circulation. 2014;129:728–36.

56. Haqqani HM, Tschabrunn CM, Betensky BP, et al. Layered activation of epicardial scar in arrhythmogenic right ventricular dysplasia: possible substrate for confined epicardial circuits. Circ Arrhythm Electrophysiol. 2012;5:796–803.

57. Garcia FC, Bazan V, Zado ES, Ren JF, Marchlinski FE. Epicardial substrate and outcome with epicardial ablation of ventricular tachycardia in arrhythmogenic right ventricular cardiomyopathy/ dysplasia. Circulation. 2009;120:366–75.

58. Marchlinski FE, Zado E, Dixit S, et al. Electroanatomic substrate and outcome of catheter ablative therapy for ventricular tachycardia in setting of right ventricular cardiomyopathy. Circulation. 2004;110:2293–8.

59. Verma A, Kilicaslan F, Schweikert RA, et al. Short- and long-term success of substrate-based mapping and ablation of ventricular tachycardia in arrhythmogenic right ventricular dysplasia. Circulation. 2005;111:3209–16.

60. Ueda A, Fukamizu S, Soejima K, et al. Clinical and electrophysiological characteristics in patients with sustained monomorphic reentrant ventricular tachycardia associated with dilated-phase hypertrophic cardiomyopathy. Europace. 2012;14:734–40.

61. Santangeli P, Di Biase L, Lakkireddy D, et al. Radiofrequency catheter ablation of ventricular arrhythmias in patients with hypertrophic cardiomyopathy: safety and feasibility. Heart Rhythm. 2010;7:1036–42.

62. Inada K, Seiler J, Roberts-Thomson KC, et al. Substrate characterization and catheter ablation for monomorphic ventricular tachycardia in patients with apical hypertrophic cardiomyopathy. J Cardiovasc Electrophysiol. 2011;22:41–8.

63. Vaseghi M, Gima J, Kanaan C, et al. Cardiac sympathetic denervation in patients with refractory ventricular arrhythmias or electrical storm: Intermediate and long-term follow-up. Heart Rhythm. 2014;11:360–6.

64.•• Lee GK, Klarich KW, Grogan M, Cha YM. Premature ventricular contraction-induced cardiomyopathy: a treatable condition. Circ Arrhythm Electrophysiol. 2012;5:229–36. *This article provides an excellent review on the current state of knowledge on the treatment of premature ventricular contractions.*

65. Lakkireddy D, Di Biase L, Ryschon K, et al. Radiofrequency ablation of premature ventricular ectopy improves the efficacy of cardiac resynchronization therapy in nonresponders. J Am Coll Cardiol. 2012;60:1531–9.

66. Niwano S, Wakisaka Y, Niwano H, et al. Prognostic significance of frequent premature ventricular contractions originating from the ventricular outflow tract in patients with normal left ventricular function. Heart. 2009;95:1230–7.

67. Takemoto M, Yoshimura H, Ohba Y, et al. Radiofrequency catheter ablation of premature ventricular complexes from right ventricular outflow tract improves left ventricular dilation and clinical status in patients without structural heart disease. J Am Coll Cardiol. 2005;45:1259–65.

68. Bogun F, Crawford T, Reich S, et al. Radiofrequency ablation of frequent, idiopathic premature ventricular complexes: comparison with a control group without intervention. Heart Rhythm. 2007;4:863–7.

69. Baman TS, Lange DC, Ilg KJ, et al. Relationship between burden of premature ventricular complexes and left ventricular function. Heart Rhythm. 2010;7:865–9.

70. Del Carpio MF, Syed FF, Noheria A, et al. Characteristics of premature ventricular complexes as correlates of reduced left ventricular systolic function: study of the burden, duration, coupling interval, morphology and site of origin of PVCs. J Cardiovasc Electrophysiol. 2011;22:791–8.

71. Yokokawa M, Kim HM, Good E, et al. Impact of QRS duration of frequent premature ventricular complexes on the development of cardiomyopathy. Heart Rhythm. 2012;9:1460–4.

72. Sun Y, Blom NA, Yu Y, et al. The influence of premature ventricular contractions on left ventricular function in asymptomatic children without structural heart disease: an echocardiographic evaluation. Int J Cardiovasc Imaging. 2003;19:295–9.

73. Olgun H, Yokokawa M, Baman T, et al. The role of interpolation in PVC-induced cardiomyopathy. Heart Rhythm. 2011;8:1046–9.

74. Yokokawa M, Kim HM, Good E, et al. Relation of symptoms and symptom duration to premature ventricular complex-induced cardiomyopathy. Heart Rhythm. 2012;9:92–5.

75. Gami AS, Noheria A, Lachman N, et al. Anatomical correlates relevant to ablation above the semilunar valves for the cardiac electrophysiologist: a study of 603 hearts. J Interv Card Electrophysiol. 2011;30:5–15.

76. Daniels DV, Lu YY, Morton JB, et al. Idiopathic epicardial left ventricular tachycardia originating remote from the sinus of Valsalva: electrophysiological characteristics, catheter ablation, and identification from the 12-lead electrocardiogram. Circulation. 2006;113:1659–66.

77. Capucci A, Di Pasquale G, Boriani G, et al. A double-blind crossover comparison of flecainide and slow-release mexiletine in the treatment of stable premature ventricular complexes. Int J Clin Pharmacol Res. 1991;11:23–33.

78. Singh SN, Fletcher RD, Fisher SG, et al. Amiodarone in patients with congestive heart failure and asymptomatic ventricular arrhythmia. Survival Trial of Antiarrhythmic Therapy in Congestive Heart Failure. N Engl J Med. 1995;333:77–82.

79. Yamada T, Doppalapudi H, McElderry HT, et al. Idiopathic ventricular arrhythmias originating from the papillary muscles in the left ventricle: prevalence, electrocardiographic and electrophysiological characteristics, and results of the radiofrequency catheter ablation. J Cardiovasc Electrophysiol. 2010;21:62–9.

80. Yamada T, Doppalapudi H, McElderry HT, et al. Electrocardiographic and electrophysiological characteristics in idiopathic ventricular arrhythmias originating from the papillary muscles in the left ventricle: relevance for catheter ablation. Circ Arrhythm Electrophysiol. 2010;3:324–31.

81. Yamada T, Murakami Y, Yoshida N, et al. Preferential conduction across the ventricular outflow septum in ventricular arrhythmias originating from the aortic sinus cusp. J Am Coll Cardiol. 2007;50: 884–91.

82. Yokokawa M, Good E, Crawford T, et al. Recovery from left ventricular dysfunction after ablation of frequent premature ventricular complexes. Heart Rhythm. 2013;10:172–5.

83. Hasdemir C, Kartal Y, Simsek E, Yavuzgil O, Aydin M, Can LH. Time course of recovery of left ventricular systolic dysfunction in patients with premature ventricular contraction-induced cardiomyopathy. Pacing Clin Electrophysiol. 2013;36:612–7.

84. Bogun F, Crawford T, Chalfoun N, et al. Relationship of frequent postinfarction premature ventricular complexes to the reentry circuit of scar-related ventricular tachycardia. Heart Rhythm. 2008;5: 367–74.

85. Sarrazin JF, Good E, Kuhne M, et al. Mapping and ablation of frequent post-infarction premature ventricular complexes. J Cardiovasc Electrophysiol. 2010;21:1002–8.

86. Kirklin JK, Naftel DC, Kormos RL, et al. Second INTERMACS annual report: more than 1,000 primary left ventricular assist device implants. J Heart Lung Transplant. 2010;29:1–10.

87. Wadia Y, Delgado 3rd RM, Odegaard P, Frazier OH. Jarvik 2000 FlowMaker axial-flow left ventricular assist device support for management of refractory ventricular arrhythmias. Congest Heart Fail. 2004;10:195–6.

88.•• Cesario DA, Saxon LA, Cao MK, Bowdish M, Cunningham M. Ventricular tachycardia in the era of ventricular assist devices. J Cardiovasc Electrophysiol. 2011;22:359–63. *This paper provides a concise review on the management of ventricular arrhythmias in patients with ventricular assist devices.*

89. Brenyo A, Rao M, Koneru S, et al. Risk of mortality for ventricular arrhythmia in ambulatory LVAD patients. J Cardiovasc Electrophysiol. 2012;23:515–20.

90. Ziv O, Dizon J, Thosani A, Naka Y, Magnano AR, Garan H. Effects of left ventricular assist device therapy on ventricular arrhythmias. J Am Coll Cardiol. 2005;45:1428–34.

91. Shirazi JT, Lopshire JC, Gradus-Pizlo I, Hadi MA, Wozniak TC, Malik AS. Ventricular arrhythmias in patients with implanted ventricular assist devices: a contemporary review. Europace. 2013;15: 11–7.

92. Mulloy DP, Bhamidipati CM, Stone ML, et al. Cryoablation during left ventricular assist device implantation reduces postoperative ventricular tachyarrhythmias. J Thorac Cardiovasc Surg. 2013;145:1207–13.

93. Dandamudi G, Ghumman WS, Das MK, Miller JM. Endocardial catheter ablation of ventricular tachycardia in patients with ventricular assist devices. Heart Rhythm. 2007;4:1165–9.

94. Cantillon DJ, Bianco C, Wazni OM, et al. Electrophysiologic characteristics and catheter ablation of ventricular tachyarrhythmias among patients with heart failure on ventricular assist device support. Heart Rhythm. 2012;9:859–64.