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Ventricular Tachycardia and Heart Failure

Hakan Altay

Abstract

Ventricular tachycardia (VT) is a common arrhythmia seen in patients with heart failure (HF) and is now seen more frequently as these patients survive longer with modern therapies. In patients with HF, half of the deaths are sudden due to life-threatening ventricular arrhythmias, including VT. Although disease modifying drugs, such as beta blockers, mineralocorticoid drugs, and angiotensin receptor neprilysin inhibitors, prevent the occurrence of VT to some extent, the mainstay of therapy is the antiarrhythmic drug therapy, implantable cardioverter-defibrillator (ICD) implantation, and traditional radiofrequency catheter ablation. Autonomic nerve system modulation and stereotactic body radiation therapy have emerged as novel techniques for the management of refractory VT cases. Patients with refractory VT and repetitive ICD shocks should be further evaluated regarding the candidacy for left ventricular assist device and transplantation.

Keywords: ventricular tachycardia, heart failure, antiarrhythmic therapy, implantable-cardioverter defibrillator, ablation

1. Introduction

Ventricular tachycardia (VT) is common in patients with heart failure (HF). The presence and severity of VT increase as the severity of HF increases. Larger infarcts with greater left ventricle (LV) systolic dysfunction are more likely to be associated with VT. VT forms one of the most common electrical mechanisms responsible for sudden cardiac death (SCD) in HF. Patients with LV systolic dysfunction who develop VT are at increased risk of SCD from subsequent VT or ventricular fibrillation [1].

Patients with VT and HF may present either with cardiac arrest to the emergency department or with palpitations, syncope, chest pain, or ICD shocks to cardiology outpatient clinics, varying according to the hemodynamic stability of VT. Both non-sustained VT (VT duration < 30 sec) and sustained VT (VT duration > 30 sec) in patients with HF are associated with significant morbidity and mortality. VT storm (three or more separate episodes of sustained VT requiring intervention (such as ICD shock or ATP) within 24 hours) is the most troublesome condition related with VT and HF.

Although half of the patients with HF have preserved ejection fraction and SCD is also a common issue in these patients, there is no proved treatment either by ICD or drugs [2]. Because of this, VT and HF will be discussed in the context of HF with reduced ejection fraction (HFREF).

2. Epidemiology

Ventricular tachycardia is common in patients with HF, with up to 20% of patients developing VT in 5 years after an ICD was placed [3]. In patients with HF, SCD occurs 6–9 times more often than the general population [4]. The most studied and proven predictor of ventricular tachyarrhythmia and SCD is left ventricle ejection fraction (LVEF) [5]. It has been shown that once the LVEF recovered, the incidence of ventricular tachyarrhythmia decreases [6].

The threshold of LVEF <35% represents an accepted threshold at which SCD risk is increased and primary prevention is indicated. Several other risk predictors of VT, such as non-sustained VT, programmed ventricular stimulation on electrophysiological study (EPS), microvolt T-wave alternans, late potentials on signal-averaged electrocardiogram, absence of heart rate variability, QT wave dispersion, baroreflex sensitivity, and heart rate turbulence have been proposed for patients with HF. However, none of these predictors has influenced the clinical practice.

3. Pathophysiology

There are multiple mechanisms that play a role in the occurrence of VT in patients with HF (**Table 1**). Adverse remodeling and progressive fibrosis occur in the ventricle following myocardial infarction (MI) or in association with non-ischemic cardiomyopathy. These structural alterations as well as the ion channel changes form the essential substrate for the induction of VT [7].

The most common mechanism for VT is electrical reentry within and around patches of heterogenous myocardial fibrosis, most commonly occurring in areas of scar post-myocardial infarction or non-ischemic cardiomyopathy [8]. The scar-related VT is typically monomorphic with single QRS morphology. Induction of monomorphic VT during EPS predicts patients who have the risk of spontaneous VT. Polymorphic VT is defined as a continually changing QRS morphology, often associated with acute ischemia, drugs which lead to QT prolongation or electrolyte imbalance.

Increased sympathetic nervous system (SNS) activation is another trigger for induction of VT. SNS activation, via beta-adrenoreceptors activates ryanodine receptor on the sarcoplasmic reticulum inside the cardiomyocytes leading to efflux of calcium and increase of intracellular concentration which is a trigger for VT. This is the rationale under the effect of beta blockers in suppressing VT, as well as SCD in HF patients.

Mechanisms
Positive remodeling and fibrosis
Myocardial scar
Electrolyte abnormalities
Increased sympathetic tone
Ischemia
Abnormal calcium handling
Delayed after depolarization
Drugs

Table 1.
Mechanisms of VT occurrence in patients with heart failure.

VT occurring within 24–48 hours of acute MI is called primary VT, and acute ischemia is considered to be the transient or correctable cause of VT in this case. Revascularization is the primary management form of primary VT. VT occurring after 48 hours of acute MI is called secondary VT, which is associated with worse clinical outcomes.

Increased diastolic calcium levels, early and delayed after depolarizations, and some of the drugs also cause VT. Antiarrhythmic drugs are the foremost drugs causing VT. Digoxin that is commonly used in the management of HF is an arrhythmogenic drug. Dobutamine treatment for acute decompensated HF has also been associated with VT [9]. Because of this, patients should be continuously monitorized during treatment with dobutamine. VT can also present as a complication of left ventricular assist device in an advanced HF patient. Most of these types of VT occur perioperatively [10]. It is important to find out the definite mechanism of VT in order to implement the best effective treatment. In patients with sarcoidosis, for example, VT can occur as a result of inflammation, scar or both. If VT is thought to be due to inflammation, best treatment is antiarrhythmic drug and immunosuppressive, whereas if VT is of scar related, best treatment is antiarrhythmic drug and catheter ablation [11].

4. Management

Management of VT in heart failure poses a great challenge to cardiologists since antiarrhythmic drugs are limited by incomplete efficacy and unfavorable adverse effect profile, ICD is complex and expensive and may affect the quality of life adversely because of inappropriate shocks, and invasive catheter ablation owns the risk of complication and recurrence. Therefore, multidisciplinary team approach including electrophysiologists, heart failure specialists, general cardiologists, intensivists, and cardiovascular surgeon should be used to tackle such a difficult disease.

VT is a life-threatening condition and needs urgent management. Acute management of VT in HF patients depends on the hemodynamic stability of the patient. In hemodynamically unstable VT, the priority is electrical direct current cardioversion [12]. If the patient is hemodynamically stable, a trial of antiarrhythmic treatment should be applied. Intravenous amiodarone is the most effective and safe antiarrhythmic treatment in this case [12].

Slow VT (<150 beats/minute) may be tolerated in the short term (**Figure 1**). However, slow VT in the presence of poor ventricular function may cause hemodynamic compromise in the long term. It is important to closely monitor the patient while administering antiarrhythmic therapy. If the antiarrhythmic therapy does not cardiovert the patient, shock should be applied as early as possible since sustained VT can compromise hemodynamic status of the patient with left ventricular dysfunction in due course. The initial approach to the management of VT should include evaluation for correctable causes of VT (e.g., electrolyte abnormalities and ischemia). Electrolyte abnormalities, particularly hypokalemia and hypomagnesemia which are known to facilitate VT in HF patients should be corrected. Potassium and magnesium levels should be kept >4 meq/l and > 2 meq/l, respectively. Agents, for example, digoxin, that may induce arrhythmia should be withheld.

For chronic management of VT, optimization of guideline-directed medical treatment is very important especially in patients with HFREF. Until recently, these treatments consisted of angiotensin converting enzyme inhibitors (ACEis) or angiotensin receptor blockers (ARBs), beta blockers (BBs), and mineralocorticoid receptor antagonists (MRAs). Of these guideline-directed medical treatments, BB and MRA have been proved to prevent sudden cardiac death [13, 14]. These drugs have the ability to improve reverse modeling which reduces VT. BBs are the

Figure 1.
Slow VT at a rate of approximately 125/bpm in a patient on high dose of beta blocker and amiodarone.

first-line therapy for the management of VT in HF patients. In MADIT-II trial (the Multicenter Automatic Defibrillator Implantation Trial II), patients with ICD treated with the highest dose of BB experienced less ICD treatment compared to patients not taking BB [15].

A meta-analysis compared medical treatment with ICD preventing SCD in patients with HF and left ventricular systolic dysfunction. MRAs were found to be the most effective drug when added to ACEi and BB, in preventing SCD [16]. Zannad et al. also showed that MRAs were equally effective in preventing SCD in patients with ICD as without ICD [17].

A newly emerged drug in HFREF, angiotensin receptor neprilysin inhibitor (ARNi), was compared with enalapril in PARADIGM-HF trial (prospective comparison of angiotensin neprilysin inhibitor (ARNI) with ACE-i to determine impact on global morbidity and mortality in heart failure) [18]. ARNi was shown to be superior in reducing cardiovascular death and hospitalization compared to enalapril. ARNi also reduced SCD by 20% compared to enalapril. European Society of Cardiology 2016 HF guideline has made a class 1 recommendation regarding the use of BB, MRA, and ARNi in patients with HFREF and VT [19].

Optimum use of guideline-directed medical treatment prevents development of VT to some extent. If the patient continues to be at risk of VT because of low ejection fraction, non-sustained or sustained VT, antiarrhythmic drugs, ICD implantation, and VT ablation are the subsequent treatment options for chronic management of VT. General use of antiarrhythmic drugs in HF is not recommended for VT since these drugs, except amiodarone, have been shown to increase mortality in patients with HF due to proarrhythmic or negative inotropic effects.

Notorious CAST trial (Cardiac Arrhythmia Suppression Trial) showed that class 1C agents, encainide, and flecainide increases mortality and non-fatal cardiac arrest when used to suppress VT post-myocardial infarction [20]. CAST trial was planned to answer the question of whether suppressing ventricular premature beats (VPB) also aid in reducing mortality. Patients who had myocardial infarction within the preceding 2 years and >6 VPBs on holter recording were enrolled. Those who had MI within 90 days were required to have EF < 55%, and those who had MI after this period were required to have EF < 40%. Patients were randomly assigned to class1C agents (encainide, flecainide, or moricizine). Patients whose PVBs were suppressed were allocated to the treatment with one of the class 1C agent or placebo. The trial was prematurely stopped based on the in-term analysis that showed that encainide and flecainide used to suppress VPBs increased the mortality by 2.5 times. It is likely that mortality excess can be attributed to the proarrhythmic effects of encainide and flecainide.

Amiodarone is the sole agent that can be used safely for suppression of VT in HF patients. Amiodarone has been studied extensively in patients with left ventricular dysfunction. Its efficacy for decreasing mortality in patients with VT and LV dysfunction has not been shown in SCD-HeFT trial (the Sudden Cardiac Death in Heart Failure Trial) [21]. However, a meta-analysis including 8522 patients post-myocardial infarction or with systolic HF showed that amiodarone reduced SCD and cardiovascular mortality [22]. Its safety, unlike class 1 antiarrhythmic agents, has been confirmed in this patient population. In patients with more severe HF, amiodarone use is associated with adverse prognosis [21]. Amiodarone cannot be used for a long period of time because it is associated with multiple side effects, primarily affecting thyroid, lung, liver, skin, and eye [23]. Therefore, regular monitoring of lung, liver, and thyroid function is required. Due to these side effects, discontinuation rates of amiodarone have been noted to be high [22].

Sotalol, a group III antiarrhythmic drug, with BB properties, is highly effective in suppressing VT but it is contraindicated in HF patients since increased mortality was demonstrated when D-sotalol was used in patients with left ventricular dysfunction after myocardial infarction in SWORD trial [24]. Dofetilide, another class III antiarrhythmic drug, failed to reduce arrhythmic death in patients with HF [25]. If VT occurs despite amiodarone therapy, mexiletine can be used as an adjunct to amiodarone.

Electrophysiologic study was once used for identification of successful antiarrhythmic therapy and also the patients who require other advanced therapies. Patients were given certain antiarrhythmic drugs after VT was induced at programmed stimulation. Patients on chronic oral antiarrhythmic drug were then assessed whether VT could be induced again [26].

Of the therapies currently available to manage VT, ICD is by far the most effective one and has the best supported safety and efficacy data from the trials and registries. An ICD has two important components: an ICD generator and a lead for sensing, pacing, and shock delivery (**Figure 2**). ICD improves the survival of patients who had VT and syncope, patients who had VT and LVEF<40%, and hemodynamic compromise [27]. ICD has been shown to prevent sudden cardiac death prophylactically in patents with LVEF <35% resulting both from ischemic or non-ischemic cardiomyopathy [21, 28, 29]. The important issue in these primary prevention groups is that they should have already received guideline-directed medical treatment for at least 3 months before ICD implantation is planned. Electrophysiologic study is no longer a required procedure before planning ICD for primary prevention.
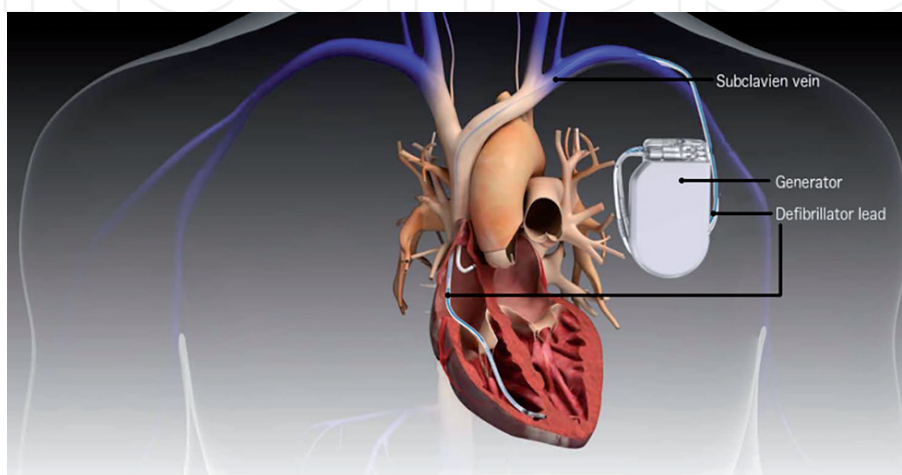

Figure 2.
A schematic representation of an intracardiac defibrillator implanted to right ventricle of heart failure patient.

ICD has antitachycardia pacing (ATP) treatment in addition to defibrillator shock and also programs which can discriminate supraventricular tachycardia from VT which aids to minimize inappropriate shocks. ATP consists of one or more sequence of pacing stimuli, generally expressed as a percentage of tachycardia cycle length for a given RR interval. In case of burst ATP, pacing stimuli is delivered at constant coupling intervals, whereas ramp ATP consists of pacing stimuli with decrement coupling interval (**Figure 3**). Once VT is confirmed, first therapy in the form of ATP was given, and if ATP does not work, then shock is delivered. Generally ICD's VT detection zone is programmed to >167 beats/min and ventricular fibrillation detection zone to >185–200 beats/min. Antiarrhythmic drugs commonly prolong VT cycle length and hence cause slow VT, a condition which may require to lower the detection zone for VT (**Figure 1**). In secondary prevention patients with HF, the programming of detection zone depends on the cycle length of the VT occurred. Generally, the detection zone is programmed 20 bpm slower than the rate of the VT occurred before. ATP for faster VT (188–250 bpm) may also be programmed with the aim for reducing shocks. ICD shocks are related with poor prognosis and quality of life. For this reason, every effort should be made to reduce shocks. It was shown that reducing defibrillator shocks was associated with increased survival [30].

Cardiac resynchronization therapy (CRT) is also an important milestone in the management of moderate to severe HF patients with prolonged QRS duration (>150 msn and LBBB morphology). CRT without defibrillator (CRT-P) can prevent SCD by improving reverse remodeling. CARE-HF Trial (Cardiac Resynchronization—Heart Failure) showed that CRT-P prevents SCD by 46% in the long term follow-up [31]. Although CRT was shown to reduce new onset VT, it had no effect on recurrent VTs [32].

In patients with HF who are refractory to antiarrhythmic therapy, radiofrequency catheter ablation has emerged as an important therapeutic option. The success rate of this technique varies according to the type of cardiomyopathy. The American Heart Association(AHA)/the Heart Rhythm Society (HRS) recommends the use of VT ablation in patients with prior myocardial infarction and recurrent VT, unresponsive or intolerant to antiarrhythmic treatment [8]. Electrophysiologic study (EPS) with programmed electrical stimulation is recommended before ablation in case of sustained monomorphic VT in patients with prior MI [33]. Catheter ablation can be effective, but acute complications and long-term VT recurrence risk necessitating repeat ablation should be recognized. And worth notifying, procedure
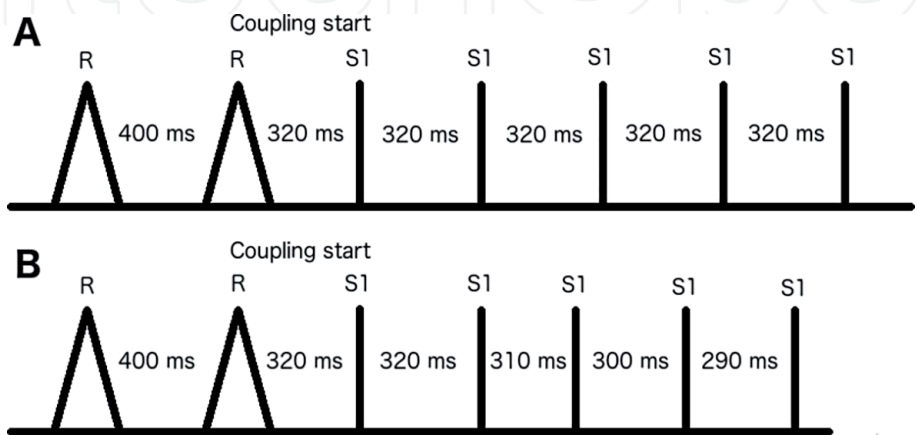

Figure 3.
Antitachycardia pacing (ATP) therapy of intracardiac defibrillator. (A) Burst ATP; pacing stimuli at lower than VT and constant coupling interval. (B) Ramp ATP; pacing stimuli starting with lower than VT cycle length and coupling intervals decreasing at each stimuli.

of ablation lasts for long hours with extended recovery times. If VT remains refractory to catheter ablation, repeat ablation may be tried. If the first ablation was done by endocardial mapping, repeat ablations may be carried by epicardial mapping. Surgical ablation is indicated in patients with VT refractory to antiarrhythmic drugs whose catheter ablation has failed [12]. It was shown that surgical cryoablation guided by endocardial and epicardial mapping along with aneurysmectomy when indicated was a successful way of terminating VT in patients who underwent bypass operation [34].

Due to multiple mechanisms of VT in idiopathic dilated cardiomyopathy, the success rate of catheter ablation is less than in ischemic cardiomyopathy. It has been shown that ablation in this type of cardiomyopathy results in higher recurrence rate of VT than ischemic cardiomyopathy [35]. Catheter ablation of VT in dilated cardiomyopathy should only be done in patients with clear mechanism of VT (e.g., bundle branch reentry) only in experienced centers. Despite these shortcomings, successful VT ablation in non-ischemic dilated cardiomyopathy has increased. Predictors of recurrence after VT ablation in non-ischemic dilated cardiomyopathy were found to be inducibility of sustained VT in the programmed stimulation study, poor systolic function (EF < 35%), and delayed intervention time [36].

Worth mentioning, there are some types of VTs occurring in the structurally normal heart, termed idiopathic VT. Idiopathic VT is further categorized according to the anatomic location in the heart. Most of them originate from the right ventricular outflow tract (RVOT) and have left bundle branch block (LBBB) pattern on the electrocardiogram. The second most common idiopathic VT originating from the conduction system is termed as fascicular VT. The other idiopathic VT originates from the mitral or tricuspid annulus and termed as annular VT. The clinical course of idiopathic VT is usually benign; however, if they occur in the form of incessant VT, they may cause LV systolic dysfunction, termed as arrhythmia-induced cardiomyopathy (AIC). It is important to differentiate AIC from non-ischemic dilated cardiomyopathy because RF ablation is the first line treatment and curative in the former [8]. VT originating from left ventricular outflow tract (LVOT) is rare compared to VT originating from RVOT. Some form of VTs originating from LVOT cannot be ablated by using conventional approach. This unique type of VT with LBBB inferior axis and early precordial transition can successfully be ablated from the aortic root, using either the left or non-coronary aortic sinus of valsalva [37]. VTs can also originate from papillary muscle of left or right ventricle. Ablation of papillary muscle VT is difficult compared to other idiopathic VTs. However, there is a case report showing successful ablation of incessant VT originating from posterior papillary muscle of right ventricle [38]. EPS is highly recommended before ablation of VT in structurally normal hearts which are suspected to be originated from RVOT, LVOT, aortic cusps, and epicardial VT [33]. EPS has also a role in case of sustained monomorphic VT in patients with arrhythmogenic right ventricular dysplasia (ARVD). It was shown that inducibility of sustained monomorphic VT during EPS highly predicts SCD, heart transplantation, VT with hemodynamic compromise, or syncope in patients with ARVD [39].

Another form of VT occurring in the structurally normal heart is catecholaminergic polymorphic VT. This type of VT should be suspected when syncope triggered by emotion or physical effort occurs in young patients with normal heart and QT interval. First line treatment is BBs. Hypertrophic cardiomyopathy (HCM), a common cause of SCD in young athletes, is a heterogenous group of cardiomyopathy with increased wall thickness. HCM with mid-ventricular obstruction and apical aneurysm is a rare form of HCM which is associated with frequent occurrence of VT. Prophylactic ICD is the main treatment, but RF ablation is required for repetitive VTs [40].

5. Management of ICD repetitive shocks

Despite ICD can effectively terminate ventricular tachycardia either by anti-tachycardia pacing or defibrillation shock, it cannot prevent VT recurrences. In patients with ICD, prevention of VT recurrence is required to minimize ICD shocks which can not only be quite uncomfortable for the patient leading to poor quality of life but also cause early battery depletion. Apart from these, recurrent shocks lead to HF progression, frequent hospitalization, and mortality. Use of antiarrhythmic drugs, particularly amiodarone can reduce ICD appropriate shocks by 34% [41]. In the OPTIC study (the Optimal Pharmacological Therapy in Cardioverter Defibrillator Patients), beta blocker and amiodarone combination were shown to be superior in suppression of VT recurrence compared to BB alone or sotalol [42]. Drug discontinuation rate at 1 year was found to be 18.2% for amiodarone, 23.5% for sotalol, and 5.3% for BB. Mexiletine, a class 1b antiarrhythmic drug, was shown to reduce VT recurrence as an adjunct to amiodarone in amiodarone-refractory VT in patients with ICD [43]. Ranolazine, a late Na channel inhibitor, was also shown to reduce VT burden and ICD shocks in patients with drug refractory VT and ICD [44].

Radiofrequency catheter ablation can be lifesaving in patients with ICD and repetitive shocks. In ischemic cardiomyopathy, some trials such as SMASH-VT (Substrate Mapping and Ablation in Sinus Rhythm to Halt Ventricular Tachycardia), VTACH (Ventricular Tachycardia Ablation in Coronary Heart Disease), and VANISH trials have shown the superiority of ablation for reducing ICD shocks [45–47]. The SMASH-VT trial compared ICD implantation plus prophylactic ablation to ICD implantation alone in patients with recent VT. Ablation reduced ICD shocks significantly from 31 to 9% and reduced VT from 33 to 12%. The VTACH trial assessed the effect of catheter ablation in patients with ischemic cardiomyopathy and mappable VT. Ablation significantly prolonged time to recurrent VT. The VANISH trial compared catheter ablation to escalation of antiarrhythmic therapy on top of first-line antiarrhythmic therapy in patients with VT. Ablation significantly reduced composite outcome of death, appropriate ICD shocks, and VT storm. Repetitive ICD shocks should also warrant referral to an advanced heart failure unit, capable for left ventricular assist device (LVAD) implantation and transplantation [48]. Catheter ablation of VT has risk of complication like all other invasive procedures. Complications related to these procedures are cardiac perforation, systemic embolism including myocardial infarction/stroke, vascular complications, and mortality.

Autonomic modulation procedures may also be applied for VT refractory to ablation. It was shown that videoscopic surgical cardiac sympathetic denervation may reduce the number of ICD shocks in refractory cases [49]. The surgery involves removal of the lower half of the stellate ganglion and T2-T4 stellate ganglia. This technique is especially effective when sympathetic denervation was made bilaterally. Renal denervation was also shown to reduce VT recurrences [50].

Stereotactic body radiation therapy (SBRT) for VT in HF patients has recently emerged as a new way of suppression of VT. SBRT is a technique that delivers high dose of radiation (25 gray) to target tissues with reduced exposure to normal adjacent tissues. SBRT has been used for decades to target various cancers. First, Cuculich et al. showed a 99.9% reduction in VT burden with cardiac SBRT in a case series of five patients with a high burden of drug-refractory VT, who had been suffering through repeated ICD shocks [51]. And recently, ENCORE VT trial showed that SBRT reduced VT and premature ventricular contraction episodes 94% at 6 months among 18 patients with treatmentrefractory VT, over half of whom presented with VT storm [52].

In selected cases with recurrent VT which cannot be managed with the treatment recommendation given above, implantation of LVAD could temporarily stabilize patient hemodynamically, as well as improve reverse remodeling. LVAD is a battery-operated mechanical pump, which takes the blood from failed LV and pumps it to the aorta to be transmitted to the rest of the body (**Figure 4**). There are not many heart failure patients with LVAD. However, the management of VT in this patient population requires mention since it is somewhat different than HF patients without LVAD. LVAD may be able to continue maintaining cardiac output in spite of sustained VT, and most of the LVAD patients have ICD in place. When such patients present to the emergency department, first patient hemodynamic status should be assessed. If the blood pressure checked by Doppler ultrasonography is okay, it is reasonable to transfer the patient to a tertiary center where there is LVAD specialist and electrophysiologist. If there is hemodynamic compromise, then the patient should be immediately converted to normal sinus rhythm with electrical shock [53]. If the patient is a candidate neither for transplantation nor LVAD, end-of-life care should be applied for palliation. Shared decision making with the patient and relatives should be done, and discussion regarding measures such as ICD deactivation may be applied in these patients.
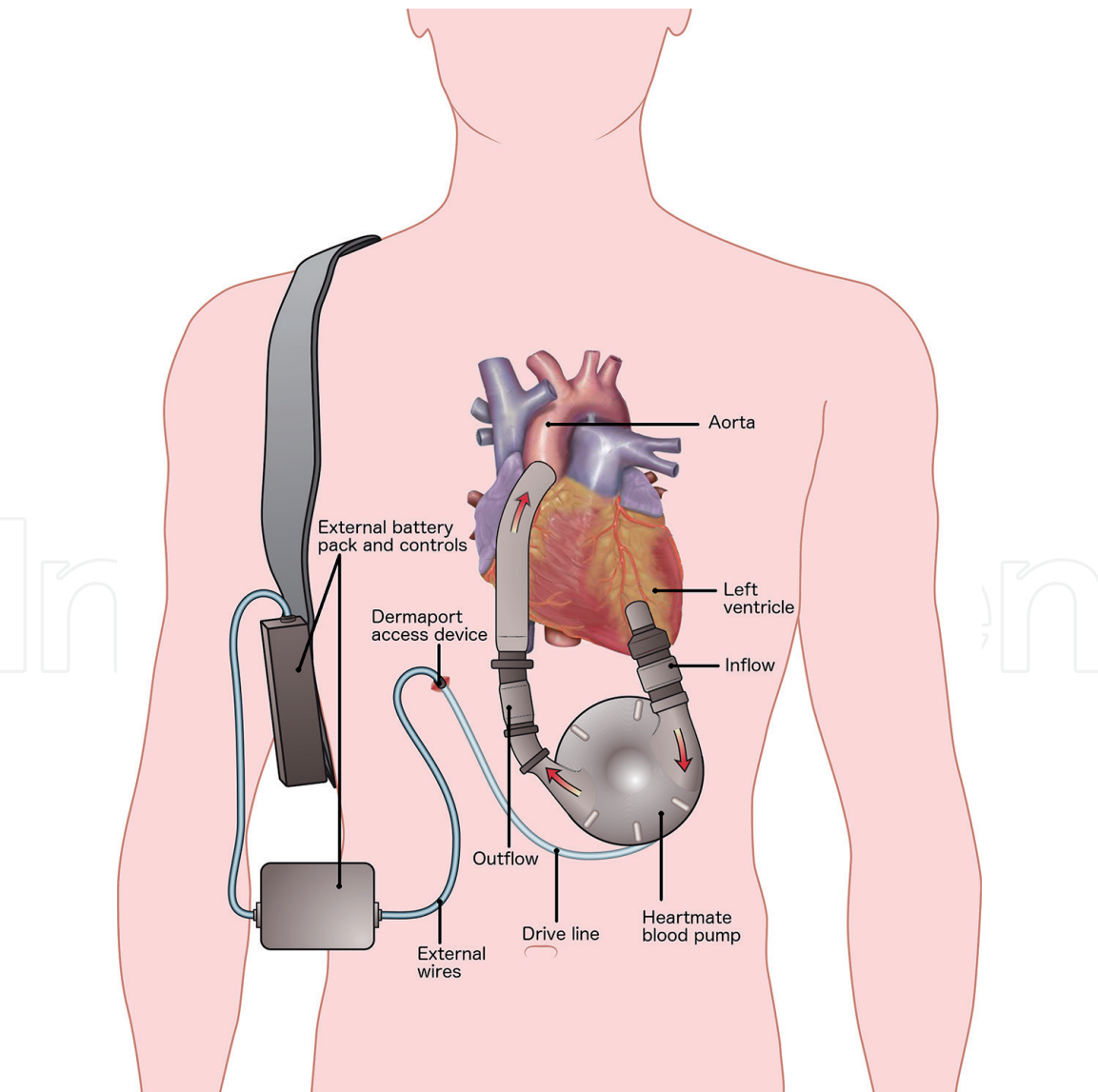

Figure 4.
A schematic representation of a left ventricle assist device (LVAD) showing battery-operated mechanical pump taking blood from left ventricle and pumping it to aorta.

VT storm is a medical emergency requiring prompt intervention. Reversible causes of VT, such as hypokalemia, hypomagnesemia, ischemia, and hypoxia should be sought and corrected where applicable. Beta blocker dose should be uptitrated to decrease the sympathetic tone. Another intervention to reduce sympathetic drive is sedation. Radiofrequency catheter ablation has been shown to be effective in controlling VT storm [54].

6. Conclusion

Ventricular tachycardia is a frequent event in HF population and is one of the poor prognostic factors related with HF. Management of VT is important because it is associated with SCD which is the responsible cause of death in 50% of patients with HF. Optimization of guideline-directed treatment is the most important step to prevent occurrence of VT in these patients. ICD has resulted marked improvement in survival of patients with HF and VT. However, repetitive ICD shocks due to recurrent VT poses a great problem and decreases survival. Antiarrhythmic therapy and VT ablation generally offer a complementary treatment in patients with ICD. Patients with VT who have failed standard therapy (antiarrhythmic therapy and catheter ablation) have limited options, with one-year survival below 20%. Autonomic modulation procedures and stereotactic body radiation therapy could be applied in patients with refractory VT. Patients with recurrent VT despite all other measures should be referred to tertiary centers where they are evaluated in respect of indications for LVAD implantation and transplantation.

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References

[1] Antiarrhythmics versus Implantable Defibrillators (AVID) Investigators. A comparison of antiarrhythmic-drug therapy with implantable defibrillators in patients resuscitated from near-fatal ventricular arrhythmias. NEJM. 1997;**337**:1576-1583

[2] Chan MM, Lam CS. How do patients with heart failure with preserved ejection fraction die? European Journal of Heart Failure. 2013;**15**:604-613

[3] Moss AJ, Greenberg H, Case RB, et al. Long-term clinical course of patients after termination of ven-tricular tachyarrhythmia by an implanted defibrillator. Circulation. 2004;**110**(25):3760-3765

[4] Kunz R, Burnand B, Schünemann HJ, et al. Das GRADE-system. Der Internist. 2008;**49**:673-680

[5] Klein L, Hsia H. Sudden cardiac death in heart failure. Cardiology Clinics. 2014;**32**:135-144

[6] Smer A, Saurav A, Azzouz MS, Salih M, Ayan M, Abuzaid A, et al. Meta-analysis of risk of ventricular arrhythmias after improvement in left ventricular ejection fraction during follow-up in patients with primary prevention implantable cardioverter defibrillators. The American Journal of Cardiology. 2017;**120**(2):279-286. DOI: 10.1016/j.amjcard.2017.04.020. Epub 2017 Apr 27

[7] Baldinger SH, Stevenson WG, John RM. Ablation of ischemic ventricular tachycardia: Evidence, techniques, results, and future directions. Current Opinion in Cardiology. 2016;**31**:29-36

[8] Al-Khatib SM, Stevenson WG, Ackerman MJ, Bryant WJ, Callans DJ, Curtis AB, et al. 2017 AHA/ACC/HRS guideline for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: Executive summary: A Report of the American College of Cardiology/ American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. Heart Rhythm. 2018;**15**(10):e190-e252. DOI: 10.1016/j. hrthm.2017.10.035. Epub 2017 Oct 30

[9] Tisdale JE, Patel R, Webb CR, Borzak S, Zarowitz BJ. Electrophys-iologic and prodysrhythmic effects of intravenous inotropic agents. Progress in Cardiovascular Diseases. 1995;**38**:167-180

[10] Nakahara S, Chien C, Gelow J, et al. Ventricular dysrhythmias after left ventricular assist device. Circulation: Arrhythmia and Electrophysiology. 2013;**6**:648-654

[11] Yalagudri S, Zin Thu N, Devidutta S, Saggu D, Thachil A, Chennapragada S, et al. Tailored approach for management of ventricular tachycardia in cardiac sarcoidosis. Journal of Cardiovascular Electrophysiology. 2017;**28**(8):893-902. DOI: 10.1111/jce.13228

[12] Priori SG, Blomström-Lundqvist C, Mazzanti A, Blom N, Borggrefe M, Camm J, et al. Task Force for the Management of Patients with Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death of the European Society of Cardiology (ESC) 2015 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: The Task Force for the Management of Patients with Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death of the European Society of Cardiology (ESC)Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPC). Europace. 2015;**17**(11):1601-1687. DOI: 10.1093/europace/euv319. Epub 2015 Aug 29

[13] Al-Gobari M, El Khatib C, Pillon F, Gueyffier F. β-Blockers for the prevention of sudden cardiac death in heart failure patients: a meta-analysis of randomized controlled trials. BMC Cardiovascular Disorders. 2013;**13**(52):1-9. DOI: 10.1186/1471-2261-13-52

[14] Pitt B, Remme W, Zannad F, Neaton J, Martinez F, Roniker B, et al. Eplerenone Post-Acute Myocardial Infarction Heart Failure Efficacy and Survival Study Investigators. Epub 2003 Mar 31. Eplerenone, a selective aldosterone blocker, in patients with left ventricular dysfunction after myocardial infarction. The New England Journal of Medicine. 2003;**348**(14):1309-1321

[15] Moss AJ, Zareba W, Hall WJ, Klein H, Wilber DJ, Cannom DS, et al. Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. The New England Journal of Medicine. 2002;**346**:877-883

[16] Peck KY, Lim YZ, Hopper I, Krum H. Medical therapy versus implantable cardioverter-defibrillator in preventing sudden cardiac death in patients with left ventricular systolic dysfunction and heart failure: A meta-analysis of >35,000 patients. International Journal of Cardiology. 2014;**173**(2):197-203. DOI: 10.1016/j.ijcard.2014.02.014. Epub 2014 Feb 22

[17] Zannad F, McMurray JJ, Krum H, van Veldhuisen DJ, Swedberg K, Shi H, et al. Eplerenone in patients with systolic heart failure and mild symptoms. The New England Journal of Medicine. 2011;**364**:11-21

[18] McMurray JJ, Packer M, Desai AS, et al. Angiotensin-neprilysin inhibition versus enalapril in heart failure. The New England Journal of Medicine. 2014;**371**:993-1004

[19] Ponikowski P, Voors AA, Anker SD, Bueno H, Cleland JG, Coats AJ, et al. Authors/task force members; document reviewers.2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: The task force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC). Developed with the special contribution of the Heart Failure Association (HFA) of the ESC. European Journal of Heart Failure. 2016;**18**(8):891-975. DOI: 10.1002/ejhf.592. Epub 2016 May 20

[20] Cardiac Arrhythmia Suppression Trial (CAST) Investigators. Preliminary report: effect of encainide and flecainide on mortality in a randomized trial of arrhythmia suppression after myocardial infarction. The New England Journal of Medicine. 1989;**321**:406-412

[21] Bardy GH, Lee KL, Mark DB, Poole JE, Packer DL, Boineau R, et al. Sudden cardiac death in heart failure trial (SCD-HeFT) Investigators. Amiodarone or an implantable cardioverter-defibrillator for congestive heart failure. The New England Journal of Medicine. 2005;**352**:225-237. DOI: 10.1056/NEJMoa043399

[22] Piccini JP, Berger JS, O'Connor CM. Amiodarone for the prevention of sudden cardiac death: a meta-analysis of randomized controlled trials. European Heart Journal. 2009;**30**:1245-1253

[23] Bokhari F, Newman D, Greene M, Ko-rley V, Mangat I, Dorian P. Long-term comparison of the implantable cardioverter defibrillator versus amiodarone: Eleven-year follow-up of a subset of patients in the Canadian Implantable Defibrillator Study (CIDS). Circulation. 2004;**110**:112-116

[24] Waldo AL, Camm AJ, deRuyter H, Friedman PL, MacNeil DJ, Pauls JF, et al. Effect of d-sotalol on mortality in patients with left ventricular dysfunction after recent and remote myocardial infarction. The SWORD

Investigators. Survival With Oral d-Sotalol. Lancet. 1996;**348**:7-12

[25] Kober L, Bloch Thomsen PE, Moller M, Torp-Pedersen C, Carlsen J, Sandoe E, et al. Effect of dofetilide in patients with recent myocardial infarction and left-ventricular dysfunction: A randomised trial. Lancet. 2000;**356**:2052-2058

[26] Horowitz LN, Josephson ME, Farshidi A, Spielman SR, Michelson EL, Greenspan AM. Recurrent sustained ventricular tachycardia 3. Role of the electrophysiologic study in selection of antiarrhythmic regimens. Circulation. 1978;**58**(6):986-997

[27] Connolly SJ, Hallstrom AP, Cappato R, Schron EB, Kuck KH, Zipes DP, et al. Meta-analysis of the implantable cardioverter defibrillator secondary prevention trials. AVID, CASH and CIDS studies. Antiar-rhythmics vs Implantable Defibrillator study. Cardiac Arrest Study Hamburg. Canadian Implantable Defibrillator Study. European Heart Journal. 2000;**21**: 2071-2078. DOI: 10.1053/euhj.2000.2476

[28] Moss AJ, Zareba W, Hall WJ, Klein H, Wilber DJ, Cannom DS, et al. Multicenter automatic defibrillator implantation trial II investigators. Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. The New England Journal of Medicine. 2002;**346**:877-883. DOI: 10.1056/ NEJMoa013474

[29] Kadish A, Dyer A, Daubert JP, Quigg R, Estes NA, Anderson KP, et al. Defibrillators in non-ischemic cardiomy-opathy treatment evaluation (DEFINITE) Investigators. Prophylactic defibrillator implantation in patients with nonischemic dilated. NEJM. 2004;**350**(21):2151-2158

[30] Sweeney MO, Sherfesee L, DeGroot PJ, Wathen MS, Wilkoff BL. Differences in effects of electrical therapy type for ventricular arrhythmias on mortality in implantable cardioverter-defibrillator patients. Heart Rhythm. 2010;**7**:353-360. DOI: 10.1016/j. hrthm.2009.11.027

[31] Cleland JG, Daubert JC, Erdmann E, Freemantle N, Gras D, Kappenberger L, et al. Longer-term effects of cardiac resynchronization therapy on mortality in heart failure [the CArdiac REsynchronization-Heart Failure (CARE-HF) trial ex- tension phase]. European Heart Journal. 2006;**27**:1928-1932

[32] Sapp JL, Parkash R, Wells GA, Yetisir E, Gardner MJ, Healey JS, et al. Cardiac resynchronization therapy reduces ven-tricular arrhythmias in primary but not secondary prophy-lactic implantable cardioverter defibrillator patients: Insight from the resynchronization in ambulatory heart failure trial. Circulation. Arrhythmia and Electrophysiology. 2017;**10**. pii: 004875

[33] Muresan L, Cismaru G, Martins RP, Bataglia A, Rosu R, Puiu M, et al. Recommendations for the use of electrophysiological study: Update 2018. Hellenic Journal of Cardiology. 2018S;**1109-9666**(18)30352-X:1-19. DOI: 10.1016/j. hjc.2018.09.002

[34] Pagé PL, Cardinal R, Shenasa M, Kaltenbrunner W, Cossette R, Nadeau R. Surgical treatment of ventricular tachycardia. Regional cryoablation guided by computerized epicardial and endocardial mapping. Circulation. 1989;**80**(3 Pt 1):I124-I134

[35] Dinov B, Fiedler L, Schönbauer R, et al. Outcomes in catheter ablation of ventricular tachycardia in dilated non-ischemic cardiomyopathy in comparison to ischemic cardiomyopathy: Results from the Prospective HEart Centre of LeiPzig VT (HELP-VT) Study. Circulation. 2014;**129**:728-736

[36] Della Bella P, Trevisi N. Catheter ablation of ventricular tachycardia in nonischemic dilated cardiomyopathy: A difficult field where we should focus our efforts. Circulation. Arrhythmia and Electrophysiology 2016;**9**(10). pii: e004649

[37] Kanagaratnam L, Tomassoni G, Schweikert R, Pavia S, Bash D, Beheiry S, et al. Ventricular tachycardias arising from the aortic sinus of valsalva: An under-recognized variant of left outflow tract ventricular tachycardia. Journal of the American College of Cardiology. 2001;**37**(5):1408-1414

[38] Fragakis N, Karamitsos TD, Vassilikos V. Successful catheter ablation of an incessant ventricular tachycardia originating from the posterior papillary muscle in a structurally normal right ventricle. Hellenic Journal of Cardiology. 2016;**57**:286-288

[39] Saguner AM, Medeiros-Domingo A, Schwyzer MA, On CJ, Haegeli LM, Wolber T, et al. Usefulness of inducible ventricular tachycardia to predict long-term adverse outcomes in arrhythmogenic right ventricular cardiomyopathy. The American Journal of Cardiology. 2013;**111**(2):250-257. DOI: 10.1016/j. amjcard.2012.09.025

[40] Lim KK, Maron BJ, Knight BP. Successful catheter ablation of hemodynamically unstable monomorphic ventricular tachycardia in a patient with hypertrophic cardiomyopathy and apical aneurysm. Journal of Cardiovascular Electrophysiology. 2009;**20**(4):445-447. DOI: 10.1111/j.1540-8167.2008.01366.x

[41] Antangeli P, Muser D, Maeda S, et al. Comparitive effectiveness of antiarrhythmic drugs and catheter ablation for the prevention of recurrent ventricular tachycardia in patients with implantable cardioverter-defibrillators: A systematic review and meta-analysis of randomized contolled trials. Heart Rhythm. 2016;**13**:1552-1559. DOI: 10.1016/j. hrthm.2016.03.004. PMID: 26961297

[42] 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**:165-171

[43] Gao D, Van Herendael H, Alshengeiti L, et al. Mexiletine as an adjunctive therapy to amiodarone reduces the frequency of ventricular tachyarrhythmia events in patients with an implantable defibrillator. Journal of Cardiovascular Pharmacology. 2013;**62**:199-204. DOI: 10.1097/ FJC.0b013e31829651fe. PMID: 23609328

[44] Bunch TJ, Mahapatra S, Murdock D, Molden J, Weiss JP, May HT, et al. Ranolazine reduces ventricular tachycardia burden and ICD shocks in patients with drug-refractory ICD shocks. Pacing and Clinical Electrophysiology. 2011;**34**:1600-1606

[45] Reddy VY, Reynolds MR, Neuzil P, et al. Prophylactic catheter ablation for the prevention of defibrillator therapy. The New England Journal of Medicine. 2007;**357**:2657-2665. DOI: 10.1056/ NEJMoa065457. PMID: 18160685

[46] Kuck KH, Schaumann A, Eckhardt 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. DOI: 10.1111/jce.12073. PMID: 23350967

[47] Sapp JL, Wells GA, Parkash R, Stevenson WG, Blier L, Sarrazin JF, et al. Ventricular tachycardia ablation versus escalation of antiarrhythmic drugs. The New England Journal of Medicine. 2016;**375**(2):111-121

[48] Yancy CW, Januzzi JL Jr, Allen LA, Butler J, Davis LL, Fonarow GC, et al. 2017 ACC expert consensus decision pathway for optimization of heart failure treatment: Answers to 10 pivotal issues about heart failure with reduced ejection fraction: A report of the American College of Cardiology Task Force on Expert Consensus Decision Pathways. Journal of the American College of Cardiology. 2018;**71**(2): 201-230. DOI: 10.1016/j.jacc.2017.11.025. Epub 2017 Dec 22

[49] Vaseghi M, Barwad P, Malavassi Corales FJ, et al. Cardiac sympathetic denervation for refractory ventricular arrhythmias. Journal of the American College of Cardiology. 2017;**69**: 3070-3080. DOI: 10.1016/j. jacc.2017.04.035. PMID: 28641796

[50] Bradfield JS, Ajijola OA, Vaseghi M, et al. Mechanisms and management of refractory ventricular arrhythmias in the age of autonomic modulation. Heart Rhythm. 2018;**15**:1252-1260. DOI: 10.1016/j.hrthm.2018.02.015. PMID: 29454137

[51] Cuculich PS, Schill MR, Kashaniand R, et al. Noninvasive cardiac radiation for ablation of ventricular tachycardia. The New England Journal of Medicine. 2017;**377**:2325-2336. (Abstract)

[52] Robinson CG, Samson PP, Moore KMS, Hugo GD, Knutson N, Mutic S, et al. Phase I/II trial of electrophysiology-guided noninvasive cardiac radioablation for ventricular tachycardia. Circulation. 2019;**139**(3):313-321

[53] Fitzgibbon J, Kman NE, Gorgas D. Asymptomatic sustained polymorphic ventricular tachycardia in a patient with a left ventricular assist device: Case report and what the emergency physician should know. The Journal of Emergency Medicine. 2016;**50**(3):e135-e141

[54] Carbucicchio C, Santamaria M, Trevisi N, Maccabelli G, Giraldi F, Fassini G, 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**:462-469