



Management of Ventricular Tachycardia

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Abstract

Ventricular tachycardia (VT) is associated with significant morbidity and mortality. Prognosis and management is dependent on the aetiology which can range from a normal heart to inherited cardiac channelopathies to structural heart disease including ischaemic heart disease, dilated cardiomyopathy or cardiac sarcoidosis. Challenges posed by a young patient age in some pathologies and a limited cardiac reserve in others require careful assessment and consideration. This review article provides a summary overview of this large topic for cardiac and non-cardiac physicians with the aim of providing some insight into the more complex invasive management strategies undertaken by electrophysiologists in the treatment of VT.

Introduction

Ventricular tachycardia is a major cause of sudden cardiac death. It accounts for 80% of sudden cardiac deaths worldwide equating to 6 million deaths per year [1]. Underlying ventricular myocardial scar is the most common aetiology, however VT can occur in the presence of a normal heart. Over the last 30 years the expanding use of the implantable cardioverter defibrillator (ICD) has led to improved mortality in patients at risk of sudden arrhythmic death [2-4]. Additionally, improvements in the understanding and visualization of scar using imaging techniques such as cardiac MRI have improved our diagnostic accuracy and treatment strategies of the underlying pathologies. Lack of major advances in anti-arrhythmic drug therapy used to treat ventricular tachycardia has been offset by development and advances in VT ablation tools and techniques for scar-related VT in recent years. The aim of this review is to discuss the management of VT, focusing on catheter ablation of scar related VT.

Myocardial Scar

Structural heart disease includes all aetiologies with underlying myocardial scar. The most common cause of structural heart disease is ischaemic cardiomyopathy (ICM), with non-ischaemic cardiomyopathy (NICM) constituting a large group including arrhythmogenic right ventricular cardiomyopathy (ARVC), dilated cardiomyopathy (DCM), post-infective scar (myocarditis) and infiltrative disorders (e.g. sarcoidosis) to name a few. There is a variation in the distribution and location of scar in differing aetiologies which is crucial to appreciate. The majority of the aforementioned aetiologies lead to predominantly left ventricular scar excluding ARVC which is characterised by fibrofatty infiltration of the right ventricle (RV) leading to abnormal RV function and abnormal structural appearance on cardiac imaging although patchy involvement can make it difficult to detect at times. Arrhythmogenic tissue has been noted in the RV outflow tract, the RV inflow tract and the free wall near the apex of the right ventricle. Left ventricle involvement, both in isolation or with RV involvement, has been described. Coronary ischemia leads to predominately endocardial scar that may be transmural whereas scar in non-ischemic cardiomyopathies tends to be isolated to the mid-wall or epicardium. One challenge in catheter ablation of ventricular tachycardia, especially in the setting of non-ischemic cardiomyopathy, is that origin of VT is more often intramural or epicardial than it is in patients with coronary artery disease. Bogun et al. [5] identified that myocardial scarring with delayed-enhancement on cardiac MRI was associated with ventricular arrhythmias and extended this observation to patients with NICM. They also suggested that scar location could help guide ablation. They identified that while 48% of their study population of NICM had scar by DE-CMR, all patients referred with sustained VT had some evidence of scar [5]. In their cohort the critical site of VT occurred within areas of scar in all cases. In patients with predominantly intramural delayed enhancement, catheter ablation was uniformly ineffective. Two patients had DE-CMR scar limited to the epicardial surface; neither of these patients had VTs that could be ablated from the endocardium. In contrast, in patients that had predominantly endocardial enhancement on DE-CMR, 71% underwent successful catheter

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ablation of all VTs via an endocardial approach; the remaining 29% had the majority of their VTs eliminated. The primary finding of this study was that DE-CMR can be used as a guide for VT ablation, even in patients considered to have non-ischemic cardiomyopathies. VT, in the absence of structural heart disease, can occur in inherited cardiac channelopathies (eg. LQT syndrome, catecholaminergic polymorphic VT and Brugada syndrome) and is often treated as a malignant arrhythmia however a more benign form can be found in normal hearts which includes outflow tract ventricular tachycardias and idiopathic fascicular VTs.

Electrophysiologic Mechanism

The three common mechanisms for most cardiac arrhythmias are also accountable for VT. This categorisation provides the understanding to appreciate the rationale behind patient selection for electrophysiology studies and subsequent ablation, however an overlap in the mechanisms often exists. Certain mechanisms have a predilection to specific aetiologies. Re-entry is the most common mechanism encountered in the presence of scar. Re-entrant arrhythmias can be induced in the electrophysiology laboratory with the aim of identification and ablation of the underlying substrate. It relies on the presence of anisotropic conduction due to a mixture of healthy myocardial tissue interspersed with connective scar tissue both of which have different conduction and refractory properties. However, it is also the cause of idiopathic fascicular VTs associated with a normal heart. Post-myocardial infarction scar is a complex heterogeneous admixture of viable myocardial cells interspersed with fibrous scar [6]. Fibrosis creates areas of conduction block and surviving myocytes display slow conduction surrounded by healthy myocardium. Similarly NICM scar constitutes a combination of interstitial and replacement fibrosis, myocyte atrophy/hypertrophy and myofibre disarray interspersed with normal myocardial tissue leading to regions of abnormal conduction [7]. Scar in these pathologies provides the anisotropic conduction necessary for re-entry with slowly conducting channels (VT isthmus) within scar exiting at the scar border to capture rapidly conducting surrounding myocardium creating re-entrant tachycardia circuits [6]. Multiple viable conduction channels can therefore be responsible for several VT morphologies. This complex architecture, in often large scars, makes the location of all potential VT isthmuses challenging accounting for a high recurrence rate after VT ablation. Idiopathic fascicular VT usually occurs secondary to a focal reentry circuit due to areas of slow decremental conduction within the Purkinje fibres of the left ventricle. The origin of VT is most commonly localized to a small region in the postero-inferior left ventricle close to the posterior fascicle of the left bundle branch. Radiofrequency ablation of this tachycardia is less complex compared with ablation in scarred substrate and associated with a higher success rate. Increased automaticity often leads to polymorphic VT and occurs when abnormal early depolarisations lead to acceleration of phase 4 of the action potential. Metabolic causes such as acute ischaemia, drugs, hypoxia, electrolyte abnormality and acid-base disorders are all triggers. Treatment of the underlying cause is the mainstay of management. Triggered activity shares features with both of the aforementioned mechanisms. A new action potential is generated during phase 3 or phase 4 of the action potential as occurs with increased automaticity. However a premature beat can provoke the arrhythmia. Triggered activity is thought to be the underlying mechanism for Torsades de Pointes. Triggered activity is also the underlying mechanism in outflow tract VT with catecholamine mediated after-depolarisations playing a crucial role.

This is supported by the initiation of tachycardia with emotional stress and exercise and termination with Adenosine. Radiofrequency ablation carries a high success rate.

Aetiology and Management

Establishing the underlying cause is a key step in the management of ventricular tachycardia. A combination of therapy targeted to the underlying cause and disease specific risk assessment for sudden cardiac death can be implemented. Ischaemic heart disease is the leading cause of both polymorphic and monomorphic VT. In recent decades early revascularization of acute coronary syndrome especially ST elevation myocardial infarction has reduced the burden of ventricular arrhythmia and sudden cardiac death. In spite of this a significant number of sudden cardiac deaths due to acute coronary syndrome occur prior to hospital admission. This highlights the importance of prevention of ischaemic heart disease in the population through risk factor modification such as smoking cessation. Patients presenting with out of hospital cardiac arrest, angiography and revascularization is recommended when the ECG is consistent with ST elevation myocardial infarction and early (within 2 hours) angiography is recommended in patients with other ECG markers of acute coronary syndrome. Coronary angiography should be considered in all other survivors of out of hospital cardiac arrest to exclude coronary occlusion particularly when a ventricular arrhythmia was the captured rhythm peri-arrest. In addition to prompt revascularization optimal medical therapy including b-blockers improves mortality. The most optimal timing of ICD therapy after a myocardial infarction or coronary revascularization is still debated but there is consensus that early ICD placement rewards no mortality benefit. Therefore in these patients assessment of LV function and ICD should be performed 6 weeks post infarction. The mainstay of sudden cardiac death prevention in patient with left ventricular dysfunction includes consideration of a primary prevention ICD in the presence of a left ventricular ejection fraction (LVEF) < 35% and NYHA functional class II-III symptoms following optimization of heart failure medications (for at least 3 months). This holds true for both ischaemic and non-ischaemic cardiomyopathies. In patients with hypertrophic cardiomyopathy (HCM) relief of left ventricular outflow tract obstruction has not been shown to reduce the risk of sudden cardiac death. Guidance for ICD implantation is taken from stratification of risk factors which have been used in the development of a HCM risk-SCD calculator [8,9]. Arrhythmogenic right ventricular cardiomyopathy is associated with a high incidence of sudden cardiac death. High risk features include family history of sudden death, history of syncope, NSVT on telemetry and right and left ventricular dysfunction and specific late gadolinium enhancement pattern on MRI. Due to the naturally progressive nature of the disease primary prevention ICD should be considered in all patients.

Implantable Cardioverter-Defibrillator

Over the years, multiple treatment options have evolved including pharmacotherapy, ICDs and catheter ablation. Combinations of these therapies are often used when structural heart disease is present. Patients with monomorphic VT who have structurally normal hearts in the absence of a channelopathy are at a low risk of sudden death. Consequently, ICDs are rarely necessary in this setting; these patients are almost always managed with medications or ablation. The current treatment of VT in structural heart disease and channelopathies revolves around ICDs for the prevention of sudden death (Table 1). These electronic devices are implanted transvenously although

Table 1: Pathologies, VT mechanism and management.

AETIOLOGY			VT MORPHOLOGY	EP MECHANISM	Management
STRUCTURAL	Ischaemic heart disease	ACS	Polymorphic	Increased automaticity	Early revascularization Optimal medical therapy
		Chronic stable CAD	Monomorphic	Re-entry	Optimal medical therapy Revascularisation if indicated Icd depending on LVEF
	Cardiomyopathy	Dilated	Monomorphic	Re-entry	Icd depending on LVEF
		ARVC	Monomorphic	Re-entry	Medical therapy Icd Genetic Screening
		HCM	Monomorphic	Re-entry	Medical therapy Icd depending on risk stratification Genetic screening
Non-Structural	Channelopathies	LQTS	Polymorphic	Triggered activity	Medical therapy Avoidance of QT prolonging medications (www.crediblemeds.org) Correction of electrolyte
		SQTS	Polymorphic	Triggered activity	Genetic Screening ICD depending on risk stratification
	Outflow tract	Brugada	Polymorphic	Re-entry	ICD depending on risk stratification Avoidance of drugs (www.brugadadrugs.org) Avoid and aggressively treat pyrexia Genetic screening
		Idiopathic fascicular left ventricular VT	Monomorphic	Re-entry	Medical therapy Radiofrequency ablation
Normal Structure			Monomorphic	Triggered activity	Medical therapy Radiofrequency ablation
Non-cardiac	Electrolyte		Polymorphic	Increased automaticity	Correct electrolytes Avoid QT prolonging drugs
	hypoxia		Polymorphic	Increased automaticity	treat cause of hypoxia
	Acidosis		Polymorphic	Increased automaticity	treat cause of hypoxia

ACS: Acute Coronary Syndrome; CAD: Coronary Artery Disease; ARVC: Arrhythmogenic Right Ventricular Cardiomyopathy; HCM: Hypertrophic Cardiomyopathy; LQTS: Long QT Syndrome; SQTS: Short QT Syndrome; SCD: Sudden Cardiac Risk; NYHA: New York Heart Association; LVEF: Left Ventricular Ejection Fraction; LV: Left Ventricle; RV: Right Ventricle.

an entirely subcutaneous option exists in those who meet specific criteria. Briefly, the device is programmed with pre-set criteria for the detection of ventricular arrhythmias. When these criteria are met the device automatically delivers programmed therapies which include anti-tachycardia pacing or defibrillation for arrhythmia termination. Both European and American guidelines recommend implantation of an ICD in cardiac arrest survivors or patients experiencing a ventricular arrhythmia in the absence of a reversible cause [10,11]. In the three major randomized controlled trials comparing ICD to anti-arrhythmic medication in survivors of cardiac arrest or patients experiencing haemodynamically compromising ventricular arrhythmias demonstrated a mortality benefit in favour of ICDs [12-14]. The population groups were similar in all three studies however, the mean left ventricular ejection fraction was higher in CASH than in CIDS or AVID. These studies have formed the basis of current secondary prevention guidelines. It is important to appreciate their limitations especially as demographics and technology have significantly changed in the last 15 years. Octogenarians were underrepresented in all three studies yet they form a significant proportion of our current patient population. There was lack of data regarding the timing of ICD insertion post myocardial infarction and post revascularization whilst thoracotomy performed in a significant majority of the original cohort is rare in current day practice.

Mortality benefit of primary prevention ICDs has been investigated in several studies. A recent meta-analysis, including studies of patients with both ischaemic and non-ischaemic cardiomyopathy, demonstrated a substantial mortality benefit [15] See (Table 2) for studies included. There was a 50% relative risk reduction in arrhythmic deaths and a 27% risk reduction in all-cause mortality. It is important to highlight that two studies were excluded from this meta-analysis as they recruited patients early after a myocardial infarction [16] or at the time of coronary bypass surgery [17]. The current European and American guidelines recommend a primary prevention ICD in patients with a NYHA functional class II-III and left ventricular ejection fraction <35% unless patients are within

40 days of a myocardial infarction or have a life expectancy of less than a year [10,11]. Although appropriate ICD shock therapy for ventricular arrhythmias saves lives, it is associated with significant negative physical, psychological and social impact on the patient [18]. Additionally ICDs do not prevent arrhythmia occurrence, thus adjunctive treatment with pharmacological agents and/or ablation may be necessary.

Anti-arrhythmic Pharmacotherapy

Pharmacotherapy for more benign VTs eg outflow tract and idiopathic fascicular VT is routinely employed in symptomatic patients or those with a significant arrhythmia burden on monitoring. Commonly these include beta-blocker and non-dihydropyridine calcium-channel blockers (Verapamil and Diltiazem). Verapamil is particularly effective in idiopathic fascicular VT and should be the first line agent in the absence of significant LV impairment.

Beta-blockers are well established first line therapeutic agents with randomized controlled trial data confirming their efficacy in the treatment of ventricular tachycardia and prevention of sudden cardiac death [19-21]. These drugs are very well tolerated by most patients with their use limited predominantly by hypotension or bronchospasm. Recurrence of VT despite these agents requires consideration of additional options. Antiarrhythmic drug trials have been disappointing, particularly in patients with left ventricular dysfunction. Earlier studies confirmed the inferiority of Class I agents for secondary VT/VF prevention in post-MI patients compared to both Amiodarone and Sotalol [22,23]. Class IC antiarrhythmic agents slow propagation and reduce tissue excitability through sodium-channel blockade and were associated with excess mortality in the Cardiac Arrhythmia Suppression Trial (CAST) in post-MI patients with impaired LVEF [24]. As such, the use of Class IC antiarrhythmic agents are not recommended for patients with ischemic heart disease or left ventricular dysfunction from any cause. The most commonly used Class I agent in the management of scar-related VT

Table 2: Studies included in meta-analysis by Theuns et al. [15].

Study	Year	patient numbers total / (lcd)	Patient group	Control	Result
MADIT (42)	1996	196/ (95)	ICM \leq 35% NYHA I-III + asymptomatic NSVT or inducible VT/VF	Conventional medical therapy including both heart failure medications and antiarrhythmic	56% relative risk reduction in all-cause mortality in the lcd group
CAT (43)	2002	104 / (50)	DCM <9 months LVEF <30%, NYHA class II-III	No information given	No significant difference in mortality
MADIT II (42)	2002	1232 / (742)	ICM EF <30%	Conventional medical therapy	31% relative risk reduction in all-cause mortality lcd group
AMIOVERT (44)	2003	103 / (51)	DCM EF <35% NYHA I-III + asymptomatic NSVT	No control group. Amiodarone vs. lcd All patients received medical therapy	No significant difference in mortality
DEFINITE (45)	2004	458 / (229)	DCM EF <36%+ ventricular ectopics or NSVT	Conventional medical therapy alone. Amiodarone use was discouraged in both groups	Significant reduction in arrhythmic death but not all cause mortality in the lcd group.
SCD-HeFT (46)	2005	2521 / (829)	ICM + DCM EF \leq 35% + NYHA II-III	Conventional medical therapy plus placebo	Risk reduction of 23% in the lcd group.

Studies included in meta-analysis by Theuns et al. [15] EF ejection fraction, NYHA New York heart association, ICD implantable cardioverter defibrillator, VF ventricular fibrillation.

is Mexiletine, which was used in 20% of patients who received adjuvant antiarrhythmic treatment in the ICD arm of the AVID trial [14]. As a Class IB antiarrhythmic agent, it does not seem to carry the increased mortality risk associated with the Class IC drugs, based on observational data with the Class IB drug Lidocaine from the Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries [25,26]. Amiodarone and Sotalol are therefore the principal agents used in the chronic treatment of VT. In the Optimal Pharmacological Therapy in Cardioverter Defibrillator Patients trial, Amiodarone and Sotalol were each more effective in preventing ICD shocks compared to beta-blockers alone, however the 1-year shock rates were still 10% in the Amiodarone arm and 24% in the Sotalol arm, with drug-related adverse effects leading to discontinuation in 20% of patients [27]. The study also demonstrated the superiority of Amiodarone in conjunction with B-blocker compared to monotherapy with Sotalol or Beta-blockers alone at reducing the number of shocks received in patients with an ICD with structural heart disease. Therefore for most patients with structural heart disease, current clinical practice favours class III anti-arrhythmics, which prolong myocardial repolarization through potassium-channel blockade. It is important to appreciate that in the era of the primary prevention ICD, Amiodarone did not confer a survival benefit over placebo in the Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT) [28]. While the lower efficacy and poor tolerability of the Class I agents has relegated them to third-line therapy for the prevention and treatment of ventricular arrhythmia, there is evidence that combination of Class I and a Class III agent may be more effective than monotherapy with either agent [29]. Despite the availability of multiple pharmacological agents for the management of ventricular arrhythmias, treatment with pharmacotherapy alone can be limited due to their adverse side-effects, poor tolerability or patient and/or physician preference to avoid the use of such agents.

Radiofrequency Ablation

Most of our knowledge and understanding of VT ablation in scarred substrate is derived from historical surgical studies [30,31]. Majority of subsequent non-surgical ablation data comes from non-randomised single centre studies or observational studies, predominantly in ICM patients. Only 3 randomised controlled trials

exist and none confirm a mortality benefit from VT ablation [32-34]. However, ablation has been shown to provide a mortality benefit in patients experiencing VT storm [35]. Prophylactic VT ablation has been shown to reduce VT recurrence and hence device therapy however the numbers needed to treat to avoid one patient receiving therapy was deemed too high to adopt this in practice [33,34]. The VTACH trial recruited patients with ischaemic cardiomyopathy, an EF<55% and stable monomorphic VT and showed a delay to recurrence of VT in patients who underwent ablation before ICD placement [34]. Similarly, the SMASH VT study was a prophylactic VT ablation study in ICM patients with monomorphic VT who were randomly assigned to defibrillator implantation alone or defibrillator implantation with adjunctive catheter ablation. Patients did not receive anti-arrhythmic drugs. A statistically significant reduction in appropriate ICD therapy observed in the ablation group [33]. More recently, Sapp et al. recruited 259 patients with recurrent VT in the ventricular tachycardia ablation versus escalation of antiarrhythmic drugs (VANISH) trial. Patient with ICM and an ICD in-situ were randomized to ablation or escalation of medical therapy. The latter involved initiation of Amiodarone, a dose increment of Amiodarone or addition of Mexiletine in those already established on maximum doses of Amiodarone [32]. Over a mean follow-up of 28 months, there was a significant difference in the overall combined endpoint of death, VT storm, and ICD shocks. Although there was no statistically significant difference in mortality rates, the ablation arm did show significant reductions in ICD shocks and VT storm, in comparison with the patients on drug therapy alone. Patients undergoing ablation who received device therapy despite treatment with Amiodarone were found to have a statistically significant increase in event free survival compared with those escalated to additional antiarrhythmic agents eg Mexiletine. Despite the lack of mortality benefit demonstrated in these randomized controlled trials it is important to mention one observational study which reported otherwise. In 2015, Tung et al. published a multicenter experience of patients undergoing VT ablation. A total of 2,061 ICM and DCM patients from 12 centers were studied. The one-year freedom from VT recurrence was 70%. Furthermore, that freedom from VT was associated with reduced overall mortality rates, as well as with transplant-free survival. Study of different New York Heart Association (NYHA) classes of patients

Table 3: Electrophysiology Mapping.

Mapping technique	Definition
Substrate Mapping	Recording of intra-cardiac electrograms during sinus rhythm to identify areas of slow conduction (fractionation, late potentials and double potentials). Once identified, these regions are potential targets for ablation.
Pacemapping	Intracardiac pacing during SR from multiple catheter positions at a rate close to the patients VT rate. Reproduction of a QRS morphology resembling the clinical VT in all 12 ECG leads suggests proximity to the exit site of the VT circuit.
Activation Mapping	Recording of intra-cardiac electrograms from multiple locations during VT. The site of earliest ventricular activation is usually the exit site of the tachycardia. Presystolic (diastolic) signals occurring before the surface QRS complex indicate the location of the critical isthmus of the mapped VT.
Entrainment Mapping	Intracardiac pacing during VT at a rate marginally faster than the clinical VT rate. If the pacing site is within the VT circuit the surface QRS morphology will resemble the VT morphology in all 12 leads at the faster paced rate. Once pacing is terminated the measurement of the return time of the next intracardiac VT beat will be short

revealed that freedom from VT was associated with improved survival rates in all NYHA-class patients particularly in NYHA class III and IV patients [36]. This dataset implies that the “sickest” patients have the most to gain from successful VT ablation. Current guidelines from the American College of Cardiology, the American Heart Association, and the Heart Rhythm Society as well as the European Society of Cardiology recommend catheter ablation for recurrent VT or ICD therapy in drug refractory cases [37]. The ESC guidelines recommend urgent catheter ablation in patients with incessant VT which is associated with a high morbidity and is an independent predictor of cardiac death [10].

Planning

VT ablation requires careful planning with a thorough review of the clinical history; including symptoms, patient demographic, drug history, previous cardiac surgery or prior ablation, and clinical assessment; end-organ perfusion, haemodynamic stability, signs of heart failure and vascular access. Assessment of haemodynamic stability during the arrhythmia is crucial as VT induction may be required during VT mapping and ablation. Availability of the electrocardiogram (ECG) of the clinical VT can be invaluable to assess the location of the VT exit which highlights the vicinity of the VT isthmus.

Non-invasive imaging not only helps to ascertain the underlying aetiology but identifies the region, distribution and extent of scar whether endocardial, mid-myocardial, epicardial or a combination. This is key operator information for procedural planning, assessing potential ablation risks/complications and ablation outcome to guide patient counseling. Access of the pericardial space for percutaneous epicardial mapping and ablation is now well established following work by Sosa et al in the 1990s and has become an important technique in ablation of VT associated with epicardial scar [38].

Mapping and Ablation

Early techniques of mapping and ablation revolved around electrophysiological data gathered during the clinical VT. However, due to challenges faced with data gathering during haemodynamically unstable VTs in, often sick, patients, multiple VT morphologies and an understandably high VT recurrence rate due to circuits not targeted during the initial ablation procedure, a paradigm shift in VT ablation has led to substrate ablation strategies with the aim of identifying and modifying the underlying substrate with radiofrequency ablation rather than just targeting the identified or clinical VT. Current endocardial VT ablation strategies routinely involve access to the left ventricle retrogradely, via the femoral artery, and anterogradely, via a trans-septal puncture across the inter-atrial septum. A 3-dimensional geometry of the chamber of interest is created using 3-D mapping systems with the ability to display voltage and activation timing data on the geometric shell. This allows

visualisation of scar based on historically tested voltage criteria (<0.5 mV=scar, 0.5-1.5 mv=scar border zone and >1.5 mV=healthy myocardium) [39]. A combination of mapping techniques is required to locate the arrhythmogenic substrate within scar which is dependent upon the rhythm during mapping (sinus rhythm versus VT). Intracardiac recording of abnormal electrograms and pace mapping are performed during sinus rhythm whilst activation mapping and entrainment are performed during VT. A simple explanation of these techniques can be found in (Table 3). Slowly conducting channels of viable myocardium embedded within electrically unexcitable scar are often identified by the presence of abnormal electrograms (fractionated, late potentials, double potentials) which can be displayed as activation maps reflecting regions of slow conduction occurring late beyond the surface QRS or tagged on the map with colour-coded balls. Despite agreement for the need of identification of such areas to guide scar-related VT ablation amongst experts several challenges, limitations and caveats exist which will not be discussed here. This has led to a lack of globally accepted mapping or ablation algorithm with variations found between centres and amongst operators performing this procedure. Several ablation strategies for scar modification have been described. Creation of linear lesions across presumed areas of arrhythmogenic scar, targeting late potentials/local abnormal ventricular electrograms, lesions that encircle the scar (core isolation) and complete scar homogenization are described ablation strategies [40-45]. The optimal VT ablation endpoint currently relies on a historically utilised but limited test; the programmed ventricular stimulation study. It is, however, a non-standardised test which is limited by patient stability/induction of ischaemia. The reproducibility of the test can be influenced by external factors such as general anaesthesia as well as patient factors such as autonomic tone. Non-inducibility of VT is used by many operators as a positive endpoint as supported by some studies however data supporting a favourable longterm outcome is lacking in others [46-52]. Large scale randomized controlled studies are required to test alternative endpoints such as the abolition of late potentials and/or local abnormal voltage activity (LAVA) post ablation. Vergara et al. [42] and Jais et al. [43] demonstrated using a combined endpoint of VT stimulation and/or elimination of abnormal electrograms correlated with VT recurrence and death more accurately compared with VT stimulation alone [42,43]. For now the ESC guidelines recommend the use of programmed ventricular stimulation alone to assess VT ablation endpoint [10]. Despite advances in mapping and ablation tools and techniques for scar related VT, freedom from medium to long-term recurrence requires improvement [34,36].

Complications

Major complication rates are low despite patients often having a poor cardiac reserve. Impressively a 0% procedural mortality rate was observed by Peicl et al. [53] after 473 VT ablation procedures [53]. Similarly there were no deaths within 30 days of the procedure

in both the SMASH VT and VTACH trials [33,34]. The SMASH-VT and VTACH authors reported 4.9% and 3.7% procedure related complications respectively which included 1 pericardial effusion without tamponade managed conservatively, 1 cardiac decompensation requiring prolonged hospitalisation, 1 deep vein thrombosis requiring anticoagulation, 1 transient ischaemic attack and 1 transient ST elevation. A meta-analysis of studies on VT ablation for ventricular arrhythmia storm (447 patients) found a 2% complication rate [54]. Of these, procedure-related mortality was 0.6% (1 myocardial infarction, 1 cardiac tamponade, and 1 electromechanical dissociation), cerebrovascular events in 0.6%, heart block in 0.6%, and cardiac tamponade in 0.2%. For patients requiring epicardial ablation percutaneous access of the pericardium is difficult in the presence of prior cardiac surgery due to adhesions. Sacher et al. [55] reported failure to obtain epicardial access or adequate mapping in 85% post cardiac surgery patients [55]. There is a risk of damage to coronary artery bypass grafts and the authors quoted a 20% right ventricular puncture rate however only 3% resulted in significant bleeding. Coronary angiography is necessary prior to ablation delivery to ensure a 1cm distance from coronary arteries to avoid arterial damage. The risk of downstream constrictive pericarditis is unclear but there were no cases identified in the 2-year follow up period of this study [55]. Peichl et al. [53] performed univariate analysis on their patient group and reported an older age, reduced LV systolic function, and elevated serum creatinine to be the most significant risk factors for complications. Interestingly they also noted that procedures that started after 2pm were associated with twice the complication risk (10% vs. 5%). Procedure-related mortality rate across publications by 19 experienced centers averaged 0.5% [56-60].

Conclusion

The treatment of VT has been transformed by the utilisation of ICDs, ability to access the pericardium percutaneously and developments in cardiac mapping systems as well as ablation catheters and techniques. In addition to advances in ablation tools and techniques the availability of mechanical circulatory support devices such as extracorporeal membrane oxygenation (ECMO) and left ventricular assist devices has facilitated ablation in a cohort of patients with unstable arrhythmia and sick hearts. Despite these advances several questions remain unanswered. The optimal timing of ablation is unclear. Benefit is derived from ablation during VT storm with ablation not recommended as a prophylactic treatment option however the practice of offering ablation after the first or third ICD shock in patients on optimal drug therapy is largely driven by physician choice. There is no consensus on the ideal VT mapping and ablation protocol which is partially still due to the inability to optimally locate all potential VT circuits. Acute outcomes of VT ablation are currently ascertained by a historically tested and debated endpoint.

Recurrence rates in patients with scar related VT remain sub-optimal. Whether this is due to the inability to reach the substrate with ablation therapy, failure to identify and eliminate all VT sustaining channels, or the development of new substrate due to ongoing myocardial remodeling remains unclear. Ongoing research to clarify some of these questions is underway however it is crucial to appreciate the need for early referral of such patients to specialist centres for management of their complex arrhythmias.

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