Heart, Lung and Circulation (2019) **28,** 76–83
1443-9506/04/$36.00
https://doi.org/10.1016/j.hlc.2018.10.003

REVIEW

Catheter Ablation of Post-Infarct VT: Mechanisms, Strategies and Outcomes

Mehrdad Emami, MD [a*], Joseph B. Morton, MBBS, PhD [b], Kim H. Chan, MBBS, PhD [c], Kurt C. Roberts-Thomson, MBBS, PhD [a]

[a]Centre for Heart Rhythm Disorders, University of Adelaide and the Royal Adelaide Hospital, Adelaide, SA, Australia
[b]University of Melbourne and the Royal Melbourne Hospital, Melbourne, Vic, Australia
[c]University of Sydney and the Royal Prince Alfred Hospital, Sydney, NSW, Australia

Ventricular arrhythmias are one of the leading causes of death in patients with a prior myocardial infarction. Implantable cardioverter-defibrillators (ICDs) are very effective in the prevention of sudden cardiac death but the risk of recurrence remains an issue since defibrillation does not alter the underlying substrate. Recurrent ICD shocks are distressing and are associated with an increase in mortality. Catheter ablation is an effective treatment for recurrent ventricular tachycardia in these patients, particularly when antiarrhythmic therapy produces side effects or is ineffective. This paper reviews the underlying mechanisms of VT in patients with a prior myocardial infarction, and the indications, strategies and outcomes of catheter ablation.

Keywords Ischaemic heart disease • Ventricular tachycardia • Catheter ablation

Introduction

Cardiovascular diseases remain the leading cause of death in developed countries and sudden cardiac death due to cardiac arrhythmia accounts for 50% of cardiovascular mortalities [1,2]. Significant progress has been made over the last few decades, both in understanding the underlying mechanisms and management of ventricular arrhythmias. Implantable cardioverter-defibrillators are highly effective in preventing sudden death when used as primary or secondary prophylactic therapies [3–10]; and, with increasing implantable cardioverter device (ICD) implantation rates, greater numbers of patients are surviving ventricular arrhythmias. However, recurrent ICD shocks are not only distressing for patients, they are associated with an increase in mortality [11–14]. Catheter ablation is being increasingly performed to reduce ICD therapies in patients in whom antiarrhythmic therapy is ineffective or has unwanted side effects. In this review, we will discuss the pathophysiology of ischaemic VT, the different techniques of VT mapping and ablation, and the role of catheter ablation in ventricular tachycardia.

Pathophysiology of Ventricular Tachycardia in Ischaemic Cardiomyopathy

Sustained monomorphic ventricular tachycardia (VT) predominantly arises from the anatomic substrate of a prior, healed myocardial infarction (MI). This generally occurs in patients with large regions of infarcted myocardium, sometimes with aneurysm formation, and poor left ventricular function. Previously, the incidence of sustained monomorphic VT was believed to be up to 3% [15], however, with early intervention and revascularisation, large infarctions are less common and monomorphic VT is less frequent.

Myocardial perfusion is heterogeneous in nature, and varies from sub-epicardial to sub-endocardial layers. Therefore, an interruption in cardiac perfusion will lead to a heterogeneous ischaemic pattern that matures, later, into a heterogeneous scar. This heterogeneous scar is comprised of non-excitable fibrotic tissue, with islands of surviving myocardial cells. These myocytes may form ''channels'' which can traverse the scar. Interstitial fibrosis can separate the

*Corresponding author at: Department of Cardiology, Royal Adelaide Hospital, Port Road, SA 5000.

myocardial bundles within the channels, producing circuitous conduction through the bundles. This, in association with reduced cell-to-cell coupling, produces slow conduction [16,17]. These changes manifest as complex, fractionated electrograms or as late potentials. The combination of channels with slow conduction and fixed anatomical obstacles, formed by dense scar, provides the substrate for re-entry, the commonest mechanism of VT in patients post MI. The initiation of scar-related VT is demonstrated in Figure 1. In regions of myocardial scarring, multiple channels and pathways occur creating numerous potential circuits. This commonly manifests as multiple inducible VT morphologies during programmed stimulation (Figure 2).

Heterogeneous scar provides the substrate for the initiation and maintenance of VT, however, it is not clear why only a small minority of patients develop tachycardia following a MI. It is commonly observed that patients develop their first episode of VT 10 to 20 years post MI. Scar characteristics appear to be different in patients treated with early reperfusion to those with completed myocardial infarctions [18] The factors involved in the transformation and maturation of an acute ischaemic scar to a VT substrate are yet to be completely determined. Haqqani et al. evaluated patients with an ischaemic cardiomyopathy, with and without VT, and demonstrated different scar characteristics. Patients with a history of VT had larger areas of dense scarring, with more frequent late potentials, indicating greater regions of slow conduction in the scar, compared to the patients without VT [19]. Nayyar et al. extended this work by characterising the channels in patients with and without VT. The VT supporting channels were longer, with slower conduction, than channels that did not support VT [20,21]. Arrhythmia triggers such as ischaemia, changes in autonomic tone and heart failure can provide the appropriate milieu for ventricular ectopic beats to initiate sustained VT.

While scar related re-entry is the most common form of VT post MI, re-entry within the His-Purkinje system can be seen. Bundle branch re-entrant VT is the most common form, and predominantly occurs in patients with conduction system disease. The circuit comprises the left and right bundle branches and ventricular myocardium. Normally, the right bundle branch is the antegrade limb with retrograde conduction up the left bundle, producing a left bundle branch block morphology VT. Other focal triggered ventricular arrhythmias, often arising from the Purkinje system, can be observed soon after MI. These are due to delayed after-depolarisations and can initiate polymorphic VT and ventricular fibrillation (VF) [22,23]. Ventricular ectopic beats arising from within the scar or idiopathic beats independent of scar may also be seen.

Catheter Ablation of VT

Indications

Implantable cardioverter device shocks are highly effective in terminating VT but the underlying substrate for the arrhythmia remains and patients are prone to recurrent VT. About 15% of recipients of ICDs are on concomitant anti-arrhythmic drugs (AAD) at the time of ICD implantation and up to 38% receive an appropriate shock for ventricular tachycardia in the first 5 years [24,25]. Sotalol and amiodarone are the main antiarrhythmic medications used in this population. Sotalol can cause QT prolongation and its use is limited in patients with renal impairment. Amiodarone is more effective but has numerous multi-organ toxicities, with significant discontinuation rates [26,27]. Whilst effective at reducing ICD therapies, neither have been shown to reduce mortality, with a suggestion that amiodarone may increase mortality in patients with heart failure NYHA class III [27]. Several trials have compared catheter ablation to medical therapy in patients with ventricular tachycardia and prior myocardial infarction. The SMASH-VT (Substrate Mapping and Ablation in Sinus Rhythm to Halt Ventricular Tachycardia) study randomised patients presenting with VT or VF, to ICD alone or ICD with catheter ablation [28]. Catheter ablation reduced the rate of appropriate ICD therapies by 65% (33% vs 12%, $p = 0.007$). The VTACH (Ventricular Tachycardia Ablation in Coronary Heart Disease) trial included patients with haemodynamically stable VT and left ventricular ejection fraction (LVEF)$\leq$50%, with patients randomised to ICD alone or in combination with catheter ablation [29]. The time to first VT/VF recurrence was significantly longer in the catheter ablation group compared to the control group (18.6 months vs 5.9 months), with a significant reduction in VT free survival (47% vs 29%, $p = 0.045$) at 2 years. The SMS (Substrate Modification Study) randomised patients with haemodynamically unstable VT or VF and LVEF$\leq$40% to ICD alone or in combination with catheter ablation [30]. There was no significant difference in the time to first ventricular arrhythmia, but there was a reduction in the number of ICD therapies by more than 50%. Recently, the VANISH (Ventricular Tachycardia Ablation versus Escalated Antiarrhythmic Drug Therapy in Ischemic Heart Disease) trial randomised patients with VT, already on antiarrhythmic medication, to catheter ablation with continuation of the medication or escalated antiarrhythmic drug therapy [31]. This trial demonstrated a 28% reduction in the primary endpoint (composite of death, VT storm, or appropriate ICD therapy) with catheter ablation compared to escalated drug therapy.

The exact time-point at which catheter ablation should be performed has yet to be clearly determined. However, a recent sub-analysis of the VANISH study showed that patients with VT refractory to amiodarone had higher recurrence rates and mortality and derived a greater benefit from catheter ablation. Several trials [PARTITA (ClinicalTrials.gov identifier: NCT01547208), BERLIN VT (ClinicalTrials.gov Identifier: NCT02501005), STAR VT (ClinicalTrials.gov Identifier: NCT02130765)] are currently underway which may help to delineate the time to undertake catheter ablation. Currently, there is no data demonstrating a reduction in mortality with catheter ablation. Therefore, in patients with ischaemic VT, catheter ablation should be considered to reduce ICD therapies and to avoid the long-term toxic side

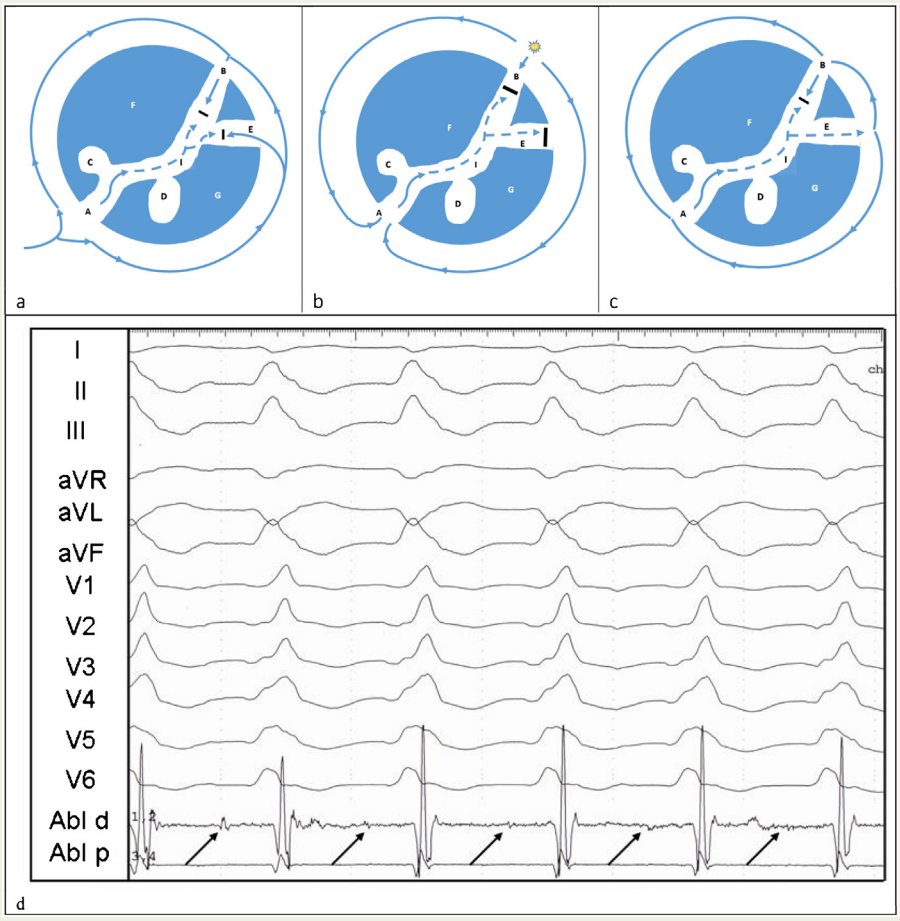

Figure 1 The initiation of re-entrant ventricular tachycardia. Scar is represented in blue. During sinus rhythm (a), activation of the channel (I) within scar occurs from both ends, with a collision of wavefronts. With a premature ventricular ectopic (b), there is conduction block at one end of the channel with subsequent activation from the other end. Due to the slow conduction within the channel, the regions of block (B, E) have time to recover and this initiate ventricular tachycardia (c). In this illustration there is a figure-of-8 re-entry with circuits around both the superior and inferior scar. (d) Example of electrogram recorded during VT from the isthmus of the scar with arrows pointing at the late diastolic potentials. Abbreviations: Abl d, ablation distal; Abl p, ablation proximal; VT, ventricular tachycardia

effects of anti-arrhythmic drugs. It is the authors' belief that catheter ablation is currently under-utilised and should be considered early, and in preference to antiarrhythmic drug escalation.

There are several circumstances in which early catheter ablation should be considered. Patients who present with electrical storm (three or more episodes of ventricular arrhythmias in a 24-hour period) have a high mortality rate and a high ventricular arrhythmia recurrence rate. Successful catheter ablation appears to reduce mortality in this population [32,33]. Slow ventricular tachycardia, often below ICD detection rates, can cause difficulties with device programming and the addition of antiarrhythmic drug therapy can potentiate this problem. Following catheter ablation, patients with haemodynamically stable VT and a relatively preserved ejection fraction appear to have a low incidence of sudden death without ICD implantation, but this has not been assessed in a randomised trial [34].

The most recent guidelines from American Heart Association/American College of Cardiology/Heart Rhythm Society (AHA/ACA/HRS) and European Society of Cardiology (ESC) outline the following recommendations for catheter ablation of VT [1,35]:

Class I indications:

1 In patients with scar-related heart disease who presented with incessant VT or electrical storm
2 In patients with scar-related heart disease and recurrent ICD shocks due to sustained VT

Class II indications

3 After the first episode of sustained VT in a patient with ischaemic heart disease and an ICD
4 In patients with scar-related heart disease and ICD shocks for sustained monomorphic VT or symptomatic sustained monomorphic VT that is recurrent, or haemodynamically tolerated

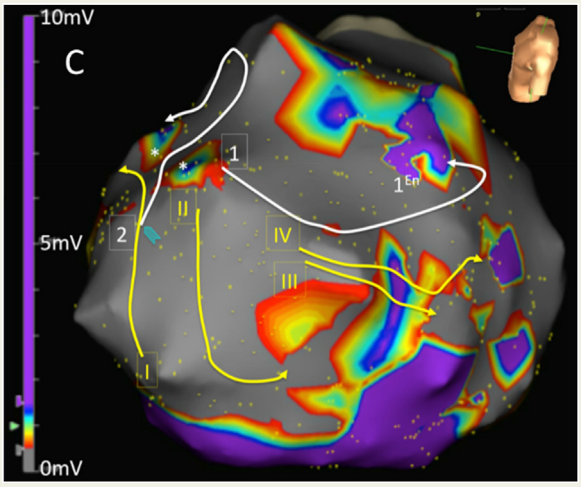

Figure 2 This is an electroanatomic map of the left ventricle in the LAO view in a patient with a prior anterior MI. The grey area represents dense scar and purple represents normal myocardium. The six arrows represent VT channels. Two white arrows represent channels responsible for two different morphologies of clinical VT. Four non-VT channels identified by pace-mapping are illustrated in this figure as yellow arrows. Note the circuitous routes of propagation of the channels.
Abbreviations: LAO, left anterior oblique; MI, myocardial infarction; VT, ventricular tachycardia

Pre-Procedural Considerations and Evaluation of the Substrate

Ventricular tachycardia ablation requires careful planning ahead of the procedure. A 12-lead electrocardiograph (ECG) of the VT is very useful in the diagnosis of site of origin of VT, as well as the site of the prior myocardial infarction [36]. The QRS morphology of the VT is determined by the exit site of the circuit and will guide the appropriate ventricle to access. If obtaining a 12-lead ECG is difficult due to prompt termination of VT by an ICD, the recorded intracardiac electrograms by the device can be used as a surrogate for the surface ECG, mainly to determine the cycle length of the clinical VT, and post pacing intervals following ATP from the ICD lead may help estimate the site of origin of VT [37,38].

Cardiac imaging has an increasing role in the management of ischaemic ventricular arrhythmias with the identification of the underlying substrate. A transthoracic echocardiogram is essential prior to electrophysiology study and ablation of VT to assess the left ventricular size and function, location of prior myocardial infarction, exclusion of ventricular thrombus, planning the method of left ventricular access, and preparation of haemodynamic support if necessary.

Pre-procedural cardiac magnetic resonance imaging with late gadolinium enhancement (cMRI-LGE) delineates the myocardial scar and determines its transmurality. Identification of patchy scars supporting re-entrant VT circuits and marking the scar edge housing the exit point of the re-entrant circuit can be achieved by pre-procedural cMRI-LGE [39]. Computerised tomography may also be used for scar delineation. The modern electro-anatomical mapping systems (Biosense Webster Irvine California/Abbott Medical, Abbott Park Illinois/Boston Scientific, Marlborough, MA, USA) permit the integration of real time three-dimensional electro-anatomical maps with pre-procedurally acquired cardiac imaging to facilitate identification of the underlying VT substrate.

Mapping and Ablation of VT

The catheter ablation procedure in patients post MI generally involves three components: (1) inducing and characterisation of the VTs; (2) defining the arrhythmia substrate; and, (3) radiofrequency ablation of the critical tachycardia components. Patients often have one predominant clinical VT morphology but with programmed stimulation other VT morphologies are frequently induced. Programmed stimulation is usually performed at two sites, at two cycle lengths with up to three extra stimuli at progressively shorter coupling intervals. All VTs induced are recorded and assessed for morphology, cycle length and haemodynamic stability. This helps localise the VT exit site and plan the ablation strategy. Following induction, the induced VTs are mostly terminated with pacing or external cardioversion. There are institutional variations in programmed stimulation protocols and more recently, many centres have stopped performing this at the start of the procedure and only undertake it after catheter ablation has been completed. The rationale behind this change is the belief that the morphology of the induced VT does not alter the ablation strategy.

Identification of the VT substrate involves the use of three-dimensional (3D) electro-anatomical mapping systems. This allows recording of local electrograms which are incorporated into a 3D geometry of the ventricle. The voltage amplitude of the electrograms differentiate areas of scarring from regions of healthy myocardium (Figure 3). Normal myocardium is characterised by a bipolar voltage of >1.5 mV, the border zone by voltages of 0.5 to 1.5 mV and scar by voltages of <0.5 mV. The location of abnormal electrograms, such as late potentials and fractionated electrograms, can be marked on the geometry.

As discussed previously, surviving myocyte channels within regions of scarring can have slow, circuitous routes of propagation and provide the substrate for VT. This anatomy produces electrogram abnormalities such as fractionated electrograms and late potentials. Fractionated electrograms have multiple intrinsic deflections with prolonged activation, representing zigzag routes of propagation. Late potentials are discrete electrograms occurring after the end of the QRS during sinus rhythm or pacing, and represent regions of slow conduction (Figure 3). Pacing at sites within the scar may demonstrate slow conduction out to the rest of the myocardium. This is represented by latency from the pacing stimulus to the onset of the QRS. Sites with long latencies, with a stimulus to QRS time of >80 ms, and matching paced QRS to the VT morphology can indicate VT isthmuses [40–42].

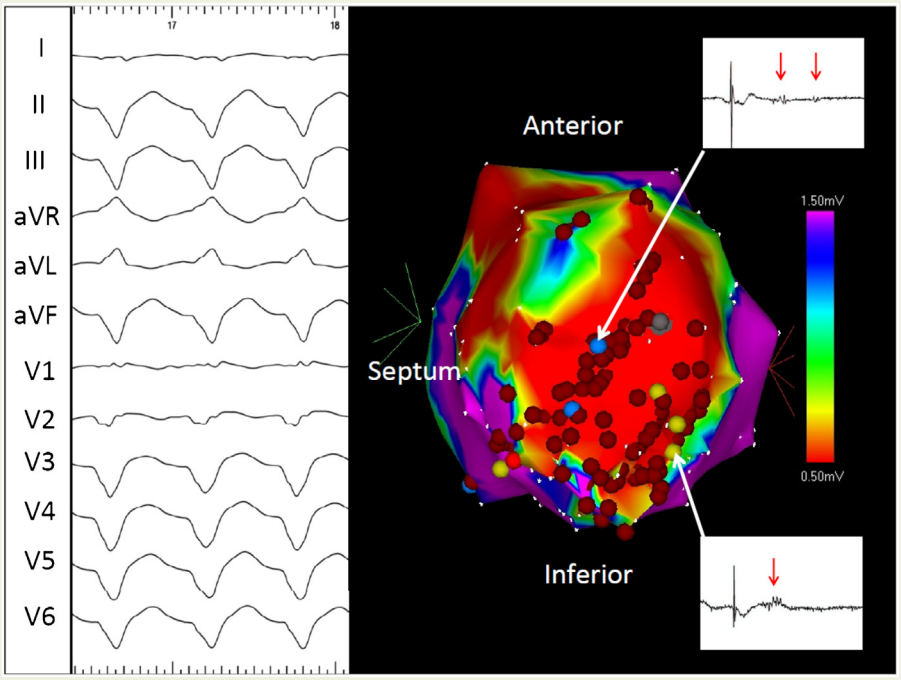

Figure 3 This figure represents the electrophysiological and electroanatomical characteristics of a patient with a prior anterior MI and ventricular tachycardia. The left panel demonstrates a 12-lead ECG of the clinical sustained monomorphic VT. This is a right bundle branch block morphology VT with a superior axis, consistent with a left ventricular apical exit. The voltage mapping of the left ventricle (LAO view) is shown in the right panel, red representing scar and purple representing normal myocardium. The voltage map (right) has late potentials marked with blue dots and fractionated electrograms marked with yellow dots. The red dots represent sites of catheter ablation.
Abbreviations: LAO, left anterior oblique; MI, myocardial infarction; VT, ventricular tachycardia; ECG, electrocardiograph

After VT induction and identification of the substrate, catheter ablation typically involves a combination of substrate modification, pace mapping and entrainment mapping. A great majority of patients undergoing VT ablation are haemodynamically unstable during VT, requiring radiofrequency ablation to occur predominantly during sinus or a paced rhythm. In this situation, fractionated electrograms and late potentials are targeted with radiofrequency ablation, in combination with pace mapping. It is possible to map during haemodynamically unstable VT with the use of left ventricular assist devices (LVADs) or extracorporeal membrane oxygenation (ECMO), and these may be useful in certain high risk patients. However, at this stage, the benefit of catheter ablation with haemodynamic support is yet to be determined in randomised trials.

While initial studies utilising substrate ablation techniques targeted clinically relevant electrograms, more recent studies have advocated extensive ablation within the scar, targeting all abnormal electrograms. This has produced improved success rates compared with traditional techniques. Another technique termed ''Core isolation'', recently described by Tzou et al. involves electrically isolating the critical region of scar, identified by entrainment, late potentials and pace mapping [43]. Demonstration of isolation led to an improvement of VT-free outcomes.

While abnormal electrograms have a high sensitivity for identifying critical components of the VT circuit, the majority represent slow, passive activation unrelated to the VT [20]. The best method for determining the relevance of a location to the VT circuit is by using entrainment manoeuvres during VT. This can only be performed during haemodynamically stable VT, or for very short periods of time in patients with unstable VT. It is beyond the scope of this paper to provide a detailed description of entrainment, and these can be found elsewhere. Briefly, this involves overdrive pacing, slightly faster than the VT cycle length, to determine whether the site of pacing is a critical component of the VT circuit. Catheter ablation, at sites fulfilling criteria for VT isthmuses, has a high success rate terminating VT (Figure 4).

In contrast to some other conditions like dilated cardiomyopathy, the vast majority of VT circuits in patients post MI are subendocardial. However, a proportion require epicardial access and ablation. Patients with recurrent VT and prior endocardial ablation attempts should be considered for epicardial ablation. The epicardial space is predominantly accessed via a percutaneous sub-xiphoid puncture with a Tuohy needle, allowing placement of a wire into the space with the subsequent introduction of a sheath. In patients with prior cardiac surgery or pericarditis, adhesions may prevent access and in this situation, limited surgical access is required [44]. Care needs to be taken to avoid damage to coronary arteries and the phrenic nerve.

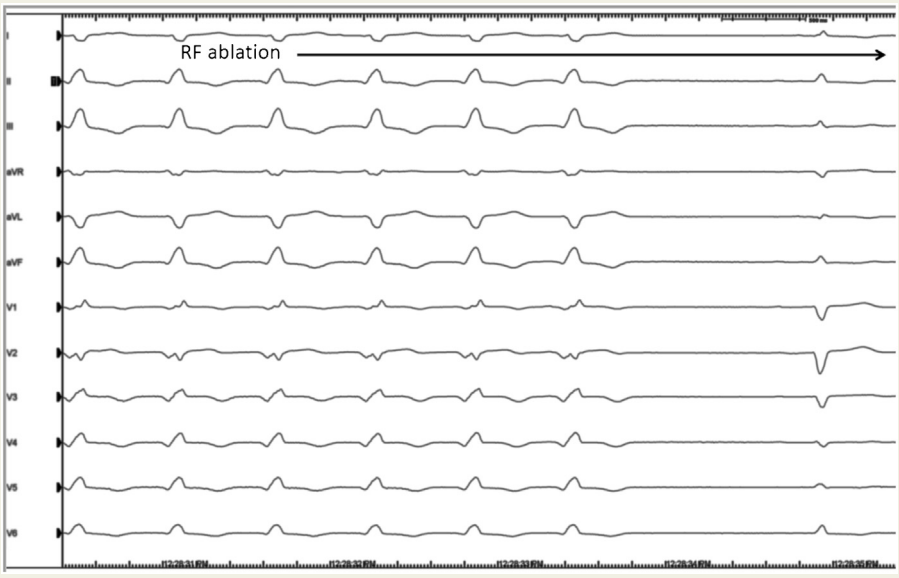

Figure 4 This figure shows a 12-lead ECG recorded during catheter ablation. Ablation at the isthmus terminated VT in seconds into the radiofrequency ablation.
Abbreviations: VT, ventricular tachycardia; ECG, electrocardiograph

Bundle branch re-entry is unusual in patients post MI but may occur. Typically, this VT has a left bundle branch block morphology with antegrade conduction down the right bundle. A His deflection precedes the QRS complex, and variation in the His-His interval precedes the subsequent VT cycle length variation. Catheter ablation of the right bundle is curative.

Focal ventricular arrhythmias, due to triggered activity or enhanced automaticity, may also be seen in patients post MI. These may arise from damaged Purkinje fibres early in the post MI period, triggering polymorphic VT or VF. They may also be seen in the longer term where they can arise from sites within the infarcted region or sites unrelated to the prior MI. Catheter ablation can abolish these arrhythmias and, in some cases, improve left ventricular function. Activation mapping is the predominant method of determining the optimal site of ablation in these cases (Figure 5).

Procedural Endpoints and Outcomes

Following the completion of ablation, programmed stimulation is repeated to assess the effectiveness of the ablation lesions. The aim is non-inducibilty of any sustained monomorphic ventricular tachycardia. The VT free success rates at 12 months is 49–72% [45–47]. However, despite recurrences, many patients have a significant reduction in the number of VT episodes [46]. These results have mostly been obtained with entrainment and pace mapping for haemodynamically tolerated VT and limited substrate ablation for poorly tolerated VT. However, recent studies have evaluated extensive substrate ablation techniques (targeting all abnormal electrograms within scar) against traditional techniques and have demonstrated lower recurrence rates with more extensive ablation [48–50]. No difference in mortality has been observed.

At this stage, catheter ablation has not been shown to reduce mortality; however, successful catheter ablation, with the associated non-inducibility post ablation, has been associated with improved survival. A meta-analysis, including data from 928 patients, demonstrated that non-inducibility post ablation was associated with a significant reduction in the recurrence of ventricular arrhythmias, and a significant reduction in mortality compared to partial success (OR 0.59; 95% CI: 0.36 to 0.98; p = 0.04) and failed ablation (OR 0.32; 95% CI: 0.10 to 0.99; p = 0.049). This is supported by other data from specialist centres, and the mortality reductions appear to be independent of heart failure severity [45].

Catheter ablation of VT post MI can be a challenging procedure and the positive results of published trials have been with experienced operators in high volume centres. Whether these results can be replicated in low volume centres is not known. Complications occur in up to 7% of patients [31,45,46]. Procedure related mortality can be up to 3%, often due to uncontrolled arrhythmia. Other complications include stroke and transient ischaemic attacks in up 0.5%, cardiac perforation in up to 1%, vascular complications in up to 5%, and acute haemodynamic decompensation in up to 2%. Whilst these complications can be significant, patients undergoing these procedures often have multiple medical problems, including poor left ventricular function, and are undergoing the procedure for recurrent life threatening arrhythmias.

Conclusion

In summary, a small proportion of patients with a history of myocardial infarction develop ventricular tachycardia after scar maturation. Myocardial re-entry is the most common

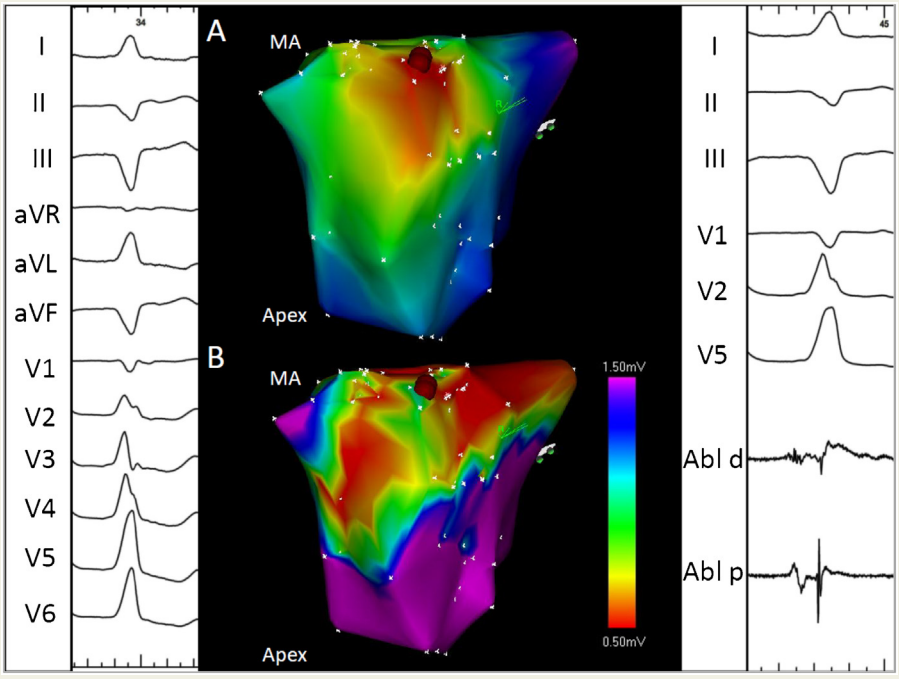

Figure 5 This figure demonstrates a focal VT arising from the region of a prior MI. The left panel shows a 12-lead ECG of the VT morphology, which shows a left bundle branch block morphology and left superior axis. The central panel shows an activation map of the left ventricular inferior wall, with the site of earliest activation shown in red (top). A voltage map of this region with scar represented in red and normal myocardium in purple (bottom). The red dot shows the site of catheter ablation. The right panel shows the local electrogram at the site of ablation. This is fractionated and early compared to the QRS.
Abbreviations: Abl d, ablation distal; Abl p, ablation proximal; MA, mitral annulus; VT, ventricular tachycardia; ECG, electrocardiograph

mechanism, due to surviving myocyte channels within regions of myocardial scarring. Implantable cardioverter devices effectively reduce sudden death but shocks are associated with significant adverse effects. Anti-arrhythmic drugs have a modest effect on the recurrence of VT and are limited by significant long-term side effects. Catheter ablation of VT is effective, and can be performed successfully in patients with both haemodynamically stable and haemodynamically unstable VT.

References

[1] 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: 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):e73–189.
[2] Goldberger JJ, Buxton AE, Cain M, Costantini O, Exner DV, Knight BP, et al. Risk stratification for arrhythmic sudden cardiac death: identifying the roadblocks. Circulation 2011;123(21):2423–30.
[3] Connolly SJ, Gent M, Roberts RS, Dorian P, Roy D, Sheldon RS, et al. Canadian implantable defibrillator study (CIDS): a randomized trial of the implantable cardioverter defibrillator against amiodarone. Circulation 2000;101(11):1297–302.
[4] Kuck KH, Cappato R, Siebels J, Ruppel R. Randomized comparison of antiarrhythmic drug therapy with implantable defibrillators in patients resuscitated from cardiac arrest: the Cardiac Arrest Study Hamburg (CASH). Circulation 2000;102(7):748–54.
[5] A comparison of antiarrhythmic-drug therapy with implantable defibrillators in patients resuscitated from near-fatal ventricular arrhythmias. N Engl J Med 1997;337(22):1576–83.
[6] Moss AJ, Hall WJ, Cannom DS, Daubert JP, Higgins SL, Klein H, et al. Improved survival with an implanted defibrillator in patients with coronary disease at high risk for ventricular arrhythmia. Multicenter Automatic Defibrillator Implantation Trial Investigators. N Engl J Med 1996;335(26):1933–40.
[7] 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. N Engl J Med 2002;346(12):877–83.
[8] Buxton AE, Josephson ME, Marchlinski FE, Miller JM. Polymorphic ventricular tachycardia induced by programmed stimulation: response to procainamide. J Am Coll Cardiol 1993;21(1):90–8.
[9] Burke MC, Gold MR, Knight BP, Barr CS, Theuns D, Boersma LVA, et al. Safety and efficacy of the totally subcutaneous implantable defibrillator: 2-year results from a pooled analysis of the IDE study and EFFORTLESS registry. J Am Coll Cardiol 2015;65(16):1605–15.
[10] Weiss R, Knight BP, Gold MR, Leon AR, Herre JM, Hood M, et al. Safety and efficacy of a totally subcutaneous implantable-cardioverter defibrillator. Circulation 2013;128(9):944–53.
[11] Moss AJ, Greenberg H, Case RB, Zareba W, Hall WJ, Brown MW, et al. Long-term clinical course of patients after termination of ventricular tachyarrhythmia by an implanted defibrillator. Circulation 2004;110(25):3760–5.
[12] Hegel MT, Griegel LE, Black C, Goulden L, Ozahowski T. Anxiety and depression in patients receiving implanted cardioverter-defibrillators: a longitudinal investigation. Int J Psychiatry Med 1997;27(1):57–69.
[13] 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(3):353–60.

[14] Schron EB, Exner DV, Yao Q, Jenkins LS, Steinberg JS, Cook JR, et al. Quality of life in the antiarrhythmics versus implantable defibrillators trial: impact of therapy and influence of adverse symptoms and defibrillator shocks. Circulation 2002;105(5):589–94.
[15] Volpi A, Cavalli A, Turato R, Barlera S, Santoro E, Negri E. Incidence and short-term prognosis of late sustained ventricular tachycardia after myocardial infarction: results of the Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico (GISSI-3) Data Base. Am Heart J 2001;142(1):87–92.
[16] de Bakker JM, Coronel R, Tasseron S, Wilde AA, Opthof T, Janse MJ, et al. Ventricular tachycardia in the infarcted, Langendorff-perfused human heart: role of the arrangement of surviving cardiac fibers. J Am Coll Cardiol 1990;15(7):1594–607.
[17] de Bakker JM, van Capelle FJ, Janse MJ, Tasseron S, Vermeulen JT, de Jonge N, et al. Slow conduction in the infarcted human heart: 'Zigzag' course of activation. Circulation 1993;88(3):915–26.
[18] Wijnmaalen AP, Schalij MJ, von der Thusen JH, Klautz RJ, Zeppenfeld K. Early reperfusion during acute myocardial infarction affects ventricular tachycardia characteristics and the chronic electroanatomic and histological substrate. Circulation 2010;121(17):1887–95.
[19] Haqqani HM, Kalman JM, Roberts-Thomson KC, Balasubramaniam RN, Rosso R, Snowdon RL, et al. Fundamental differences in electrophysiologic and electroanatomic substrate between ischemic cardiomyopathy patients with and without clinical ventricular tachycardia. J Am Coll Cardiol 2009;54(2):166–73.
[20] Nayyar S, Wilson L, Ganesan AN, Sullivan T, Kuklik P, Chapman D, 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(1):90–8.
[21] Nayyar S, Wilson L, Ganesan A, Sullivan T, Kuklik P, Young G, et al. Electrophysiologic features of protected channels in late postinfarction patients with and without spontaneous ventricular tachycardia. J Interv Card Electrophysiol 2018;51(1):13–24.
[22] Pogwizd SM, Corr PB. Mechanisms underlying the development of ventricular fibrillation during early myocardial ischemia. Circ Res 1990;66(3):672–95.
[23] Xing D, Martins JB. Triggered activity due to delayed afterdepolarizations in sites of focal origin of ischemic ventricular tachycardia. Am J Physiol Heart Circ Physiol 2004;287(5):H2078–84.
[24] Dev S, Peterson PN, Wang Y, Curtis JP, Varosy PD, Masoudi FA. Prevalence, correlates, and temporal trends in antiarrhythmic drug use at discharge after implantable cardioverter defibrillator placement (from the National Cardiovascular Data Registry [NCDR]). Am J Cardiol 2014;113(2):314–20.
[25] Saxon LA, Hayes DL, Gilliam FR, Heidenreich PA, Day J, Seth M, et al. Long-term outcome after ICD and CRT implantation and influence of remote device follow-up: the ALTITUDE survival study. Circulation 2010;122(23):2359–67.
[26] Connolly SJ, Dorian P, Roberts RS, Gent M, Bailin S, Fain ES, 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(2):165–71.
[27] Bardy GH, Lee KL, Mark DB, Poole JE, Packer DL, Boineau R, et al. Amiodarone or an implantable cardioverter-defibrillator for congestive heart failure. N Engl J Med 2005;352(3):225–37.
[28] Reddy VY, Reynolds MR, Neuzil P, Richardson AW, Taborsky M, Jongnarangsin K, et al. Prophylactic catheter ablation for the prevention of defibrillator therapy. N Engl J Med 2007;357(26):2657–65.
[29] Kuck KH, Schaumann A, Eckardt L, Willems S, Ventura R, Delacretaz E, 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(9708):31–40.
[30] Kuck KH, Tilz RR, Deneke T, Hoffmann BA, Ventura R, Hansen PS, et al. Impact of substrate modification by catheter ablation on implantable cardioverter-defibrillator interventions in patients with unstable ventricular arrhythmias and coronary artery disease: results from the multicenter randomized controlled SMS (substrate modification study). Circ Arrhythm Electrophysiol 2017;10(3).
[31] Sapp JL, Wells GA, Parkash R, Stevenson WG, Blier L, Sarrazin JF, et al. Ventricular tachycardia ablation versus escalation of antiarrhythmic drugs. N Engl J Med 2016;375(2):111–21.
[32] 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(8):560–71.
[33] Vergara P, Tung R, Vaseghi M, Brombin C, Frankel D, Di Biase L, et al. Successful ventricular tachycardia ablation in patients with electrical storm reduces recurrences and improves survival. Heart Rhythm 2018;15(1):48–55.
[34] Maury P, Baratto F, Zeppenfeld K, Klein G, Delacretaz E, Sacher F, et al. Radio-frequency ablation as primary management of well-tolerated sustained monomorphic ventricular tachycardia in patients with structural heart disease and left ventricular ejection fraction over 30%. Eur Heart J 2014;35(22):1479–85.
[35] Priori SG, Blomstrom-Lundqvist C, Mazzanti A, Blom N, Borggrefe M, Camm J, et al. 2015 ESC 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). Eur Heart J 2015;36(41):2793–867.
[36] Miller JM, Marchlinski FE, Buxton AE, Josephson ME. Relationship between the 12-lead electrocardiogram during ventricular tachycardia and endocardial site of origin in patients with coronary artery disease. Circulation 1988;77(4):759–66.
[37] Yoshida K, Liu TY, Scott C, Hero A, Yokokawa M, Gupta S, et al. The value of defibrillator electrograms for recognition of clinical ventricular tachycardias and for pace mapping of post-infarction ventricular tachycardia. J Am Coll Cardiol 2010;56(12):969–79.
[38] Haqqani HM, Roberts-Thomson KC. Radiofrequency catheter ablation for ventricular tachycardia. Heart Lung Circ 2012;21(6-7):402–12.
[39] Sasaki T, Miller CF, Hansford R, Yang J, Caffo BS, Zviman MM, et al. Myocardial structural associations with local electrograms: a study of postinfarct ventricular tachycardia pathophysiology and magnetic resonance-based noninvasive mapping. Circ Arrhythm Electrophysiol 2012;5(6):1081–90.
[40] Brunckhorst CB, Delacretaz E, Soejima K, Jackman WM, Nakagawa H, Kuck KH, et al. Ventricular mapping during atrial and right ventricular pacing: relation of electrogram parameters to ventricular tachycardia reentry circuits after myocardial infarction. J Interv Card Electrophysiol 2004;11(3):183–91.
[41] Brunckhorst CB, Delacretaz E, Soejima K, Maisel WH, Friedman PL, Stevenson WG. Identification of the ventricular tachycardia isthmus after infarction by pace mapping. Circulation 2004;110(6):652–9.
[42] Brunckhorst CB, Stevenson WG, Soejima K, Maisel WH, Delacretaz E, Friedman PL, et al. Relationship of slow conduction detected by pace-mapping to ventricular tachycardia re-entry circuit sites after infarction. J Am Coll Cardiol 2003;41(5):802–9.
[43] Tzou WS, Frankel DS, Hegeman T, Supple GE, Garcia FC, Santangeli P, et al. Core isolation of critical arrhythmia elements for treatment of multiple scar-based ventricular tachycardias. Circ Arrhythm Electrophysiol 2015;8(2):353–61.
[44] Sacher F, Roberts-Thomson K, Maury P, Tedrow U, Nault I, Steven D, et al. Epicardial ventricular tachycardia ablation a multicenter safety study. J Am Coll Cardiol 2010;55(21):2366–72.
[45] Tung R, Vaseghi M, Frankel DS, Vergara P, Di Biase L, Nagashima K, et al. Freedom from recurrent ventricular tachycardia after catheter ablation is associated with improved survival in patients with structural heart disease: An International VT Ablation Center Collaborative Group study. Heart Rhythm 2015;12(9):1997–2007.
[46] Stevenson WG, Wilber DJ, Natale A, Jackman WM, Marchlinski FE, Talbert T, 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(25):2773–82.
[47] Tanner H, Hindricks G, Volkmer M, Furniss S, Kuhlkamp V, Lacroix D, et al. Catheter ablation of recurrent scar-related ventricular tachycardia using electroanatomical mapping and irrigated ablation technology: results of the prospective multicenter Euro-VT-study. J Cardiovasc Electrophysiol 2010;21(1):47–53.
[48] Sacher F, Lim HS, Derval N, Denis A, Berte B, Yamashita S, et al. Substrate mapping and ablation for ventricular tachycardia: the LAVA approach. J Cardiovasc Electrophysiol 2015;26(4):464–71.
[49] Di Biase L, Burkhardt JD, Lakkireddy D, Carbucicchio C, Mohanty S, Mohanty P, et al. Ablation of stable VTs versus substrate ablation in ischemic cardiomyopathy: the VISTA randomized multicenter trial. J Am Coll Cardiol 2015;66(25):2872–82.
[50] Di Biase L, Santangeli P, Burkhardt DJ, Bai R, Mohanty P, Carbucicchio C, et al. Endo-epicardial homogenization of the scar versus limited substrate ablation for the treatment of electrical storms in patients with ischemic cardiomyopathy. J Am Coll Cardiol 2012;60(2):132–41.