



Ablation of ischemic ventricular tachycardia: evidence, techniques, results, and future directions

Samuel H. Baldinger, William G. Stevenson, and Roy M. John

Purpose of review

This article summarizes current understanding of the arrhythmia substrate and effect of catheter ablation for infarct-related ventricular tachycardia, focusing on recent findings.

Recent findings

Clinical studies support the use of catheter ablation earlier in the course of ischemic disease with moderate success in reducing arrhythmia recurrence and shocks from implantable defibrillators, although mortality remains unchanged. Ablation can be lifesaving for patients presenting with electrical storm. Advanced mapping systems with image integration facilitate identification of potential substrate, and several different approaches to manage hemodynamically unstable ventricular tachycardia have emerged. Novel ablation techniques that allow deeper lesion formation are in development.

Summary

Catheter ablation is an important therapeutic option for preventing or reducing episodes of ventricular tachycardia in patients with ischemic cardiomyopathy. Present technologies allow successful ablation in the majority of patients, even when the arrhythmia is hemodynamically unstable. Failure of the procedure is often because of anatomic challenges that will hopefully be addressed with technological progress.

Keywords

catheter ablation, ischemic heart disease, ventricular tachycardia

INTRODUCTION

Infarct-related monomorphic ventricular tachycardia typically arises from regions of poorly coupled surviving myofibers within scar tissue characteristically showing slow electrical conduction. In ischemic cardiomyopathy, such arrhythmia substrates are most commonly located subendocardially but can also occur intramurally and subepicardially. Areas of slow conduction can be large or widely separated within scar tissue, giving rise to multiple and potentially interconnected circuits that can result in multiple ventricular tachycardia morphologies. Progressive fibrosis and ventricular remodeling can lead to onset of ventricular arrhythmias late after myocardial infarction. A small proportion (approximately 10%) of monomorphic ventricular tachycardia in ischemic cardiomyopathy is because of automaticity or reentry involving the Purkinje system.

The occurrence of sustained monomorphic ventricular tachycardia is a marker for increased mortality in patients with structural heart disease. Implantable cardioverter defibrillators (ICDs) are effective for prevention of sudden death, but

recurrent shocks reduce quality of life, cause post-traumatic stress syndrome, and are associated with worsened cardiac outcomes [1,2,3]. Interrupting areas of slow conduction by catheter ablation aims to decrease the risk for ventricular tachycardia recurrence and thereby seeks to improve quality of life and potentially reduce the risk of death. Ablation for ventricular tachycardia storm (defined as three or more episodes in 24 h) or incessant ventricular tachycardia can be lifesaving [4].

Effective ablation requires identification of the ventricular tachycardia origin during mapping. Ablation is typically performed during ventricular tachycardia if hemodynamic stability can be maintained. If induced ventricular tachycardias are unstable or the clinical ventricular tachycardia

Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston, Massachusetts, USA

Correspondence to Roy M. John, MD, PhD, Department of Medicine, Brigham and Women's Hospital, 75 Francis Street Boston, MA 02115, USA. E-mail: rjohn2@partners.org

Curr Opin Cardiol 2016, 31:29–36

DOI:10.1097/HCO.0000000000000237

KEY POINTS

- Catheter ablation for sustained monomorphic ventricular tachycardia associated with coronary artery disease can significantly reduce ventricular tachycardia recurrence and ICD shock burden for most patients.
- Advances in mapping and ablation technology coupled with evolving techniques for substrate modification hold promise for improved outcomes for ventricular tachycardia ablation.
- Late ventricular tachycardia recurrence is common, and the effect of ablation on overall mortality has not been defined in prospective randomized clinical studies.
- Ablation should be considered earlier in the course of ventricular tachycardia without waiting to exhaust all pharmacological therapies but with due considerations to risks and benefits.

cannot be induced, ablation aims to modify the arrhythmia substrate identified during stable sinus or paced rhythms (substrate-guided ablation). Recent studies have advanced both strategies. The review will address the recent advances in techniques, clinical experience, and outcomes for ablation for monomorphic ventricular tachycardia in ischemic cardiomyopathy. Polymorphic ventricular tachycardia and ventricular fibrillation that occur more often in the acute or subacute period of myocardial ischemia or infarction are not discussed.

CURRENT EVIDENCE, RECENT DEVELOPMENTS, AND ONGOING TRIALS

Indications and timing for postinfarct ventricular tachycardia ablation

With increasing experience and expertise, catheter ablations are being increasingly employed earlier in the course of ventricular tachycardia without waiting to exhaust all pharmacological options. Table 1 summarizes current recommendations for the use of catheter ablation according to the 2009 European Heart Rhythm Association/Heart Rhythm Society (EHRA/HRS) Expert Consensus on Catheter Ablation of Ventricular Arrhythmias [5].

Two randomized trials [ventricular tachycardia ablation in coronary heart disease (VTACH) and Substrate Mapping and Ablation in Sinus Rhythm to Halt Ventricular Tachycardia (SMASH-VT)] explored the use of catheter ablation in ICD recipients with prior myocardial infarction, ventricular tachycardia, and impaired left ventricular function [6,7]. Both studies found a significant reduction in ventricular tachycardia recurrence during follow-up of approximately 2 years. However, neither of these trials was sufficiently powered to examine mortality. In a retrospective study, Bunch *et al.* [8] reported a lower risk of death and heart failure hospitalizations in patients treated with ventricular tachycardia ablation after an ICD shock compared with patients managed medically only. Recent publications support an early invasive approach. In a single-center,

Table 1. Indications for catheter ablation of ventricular tachycardia in patients with structural heart disease (adapted from [5])

Catheter ablation of VT is recommended for

- 1 Patients with SMVT, including VT terminated by an ICD, that recurs despite antiarrhythmic drug therapy or when antiarrhythmic drugs are not tolerated or not desired
- 2 Control of incessant SMVT or VT storm that is not because of a transient reversible cause
- 3 Patients with frequent PVCs, NSVTs, or VT that is presumed to cause ventricular dysfunction
- 4 Bundle branch reentrant or interfascicular VTs
- 5 Recurrent sustained polymorphic VT and VF that is refractory to antiarrhythmic therapy when there is a suspected trigger that can be targeted for ablation

Catheter ablation should be considered for

- 1 Patients who have one or more episodes of SMVT despite therapy with one or more class I or III antiarrhythmic drugs
- 2 Patients with recurrent SMVT because of prior MI who have LV ejection fraction >0.30 and expectation for 1 year of survival, and it is an acceptable alternative to amiodarone therapy
- 3 Patients with hemodynamically tolerated SMVT due to prior MI who have reasonably preserved LV ejection fraction (>0.35) even if they have not failed antiarrhythmic drug therapy

VT catheter ablation is contraindicated

- 1 In the presence of a mobile ventricular thrombus (epicardial ablation may be considered)
- 2 For asymptomatic PVCs and/or NSVT that are not suspected of causing or contributing to ventricular dysfunction
- 3 When VT is because of transient, reversible causes, such as acute ischemia, hyperkalemia, or drug-induced torsade de pointes

ICD, implantable cardioverter defibrillator; LV, left ventricle; MI, myocardial infarction; NSVT, nonsustained ventricular tachycardia; PVCs, premature ventricular contractions; SMVT, symptomatic sustained monomorphic ventricular tachycardia; VF, ventricular fibrillation; VT, ventricular tachycardia.

observational study, Dinov *et al.* [9^{***}] reported better acute and long-term success if catheter ablation of scar-related ventricular tachycardia was performed within 30 days after the first documented ventricular tachycardia. Once again, a mortality benefit was not evident in patients who underwent early ventricular tachycardia ablation compared with those who had their ablation late (>1 year) after their first presentation with ventricular tachycardia.

The benefits of early catheter ablation and its superiority to medical therapy are the subject of ongoing randomized trials. The PARTITA trial (NCT01547208) is randomizing patients who experience an appropriate ICD shock to immediate catheter ablation or waiting until the occurrence of a ventricular tachycardia storm. The Substrate Targeted Ablation Using the FlexAbility™ Ablation Catheter System for the Reduction of Ventricular Tachycardia (STAR-VT) trial (NCT02130765) aims to show a benefit of ventricular tachycardia ablation as first line therapy compared with medical therapy in patients with documented monomorphic ventricular tachycardia (either spontaneous or induced by programmed stimulation) who have an implanted ICD. The Ventricular Tachycardia (VT) Ablation Versus Enhanced Drug Therapy (VANISH) trial (NCT00905853) aims to compare aggressive antiarrhythmic therapy to catheter ablation for ventricular tachycardia recurrence after receiving ICD therapy despite antiarrhythmic drugs.

Mapping strategies and image integration

Electroanatomic mapping systems are routinely used in most centers for voltage and activation mapping and for navigation of catheters within the cardiac chambers. Automated multipoint acquisition from multipolar catheters permit fast, high-resolution mapping to identify abnormal tissue that typically demonstrates low voltages, fractionation, and late potentials [10]. The judicious use of unipolar and bipolar voltage maps enables the identification of border zones. When combined with analysis of electrograms and pacing maneuvers, conducting channels that support reentry circuits within scars can be identified during stable sinus or paced rhythm [11,12].

Mapping systems also allow integration of real-time intracardiac ultrasound images, fluoroscopy, and previously acquired MRI or computer tomography. Contrast enhanced MRI may be used to identify ventricular tachycardia substrate and areas that are likely to contain critical isthmus sites [13,14]. Prospective studies are required to assess whether such imaging guidance will improve the efficiency and efficacy of ventricular tachycardia

ablation. Image integration is potentially useful to minimize risks of phrenic nerve and coronary artery injury during epicardial ventricular tachycardia ablation [15]. MRI-guided interventions with intraprocedural imaging may facilitate substrate identification, navigation, lesion formation, and early detection of complications [16]. However, the incorporation of an MRI scanner in the electrophysiology laboratory is expensive and raises multiple logistical issues.

Contact force-sensing catheters

Radiofrequency ablation lesion creation is critically dependent on adequate contact force between the catheter and tissue [17]. Newer ablation catheters incorporate contact force sensors [18]. In an ovine model, contact force monitoring improved lesion formation in the ventricles when ablating both from the endocardium and the epicardium [19]. Contact force monitoring during mapping increases specificity for identifying low-voltage area and abnormal electrograms [20]. The clinical benefits of contact force mapping and ablation for ventricular tachycardia are yet to be fully elucidated.

Substrate-based ablation

The critical isthmus of a specific monomorphic ventricular tachycardia can be identified by activation or entrainment mapping during ventricular tachycardia, if the ventricular tachycardia is hemodynamically stable or can be induced and terminated reproducibly to allow mapping during short episodes [21–23]. In clinical practice, the majority of induced ventricular tachycardias are hemodynamically unstable and multiple morphologies of ventricular tachycardia, often reflecting multiple reentry circuits, are induced with repeated attempts to initiate ventricular tachycardia. Therefore, ablation is often limited to targeting the arrhythmia substrate during sinus or paced rhythm. Areas of slow conduction and late potentials within areas of low voltage have been correlated with critical isthmus sites for reentry [24–26].

Based on these observations, several strategies for modifying the ventricular tachycardia substrate have been described recently (Table 2). These have included the targeting of sites with late potentials or fractionated electrograms that can be shown to be poorly coupled to the surrounding myocardium [27,28], circumferential ablation of the scar area [29], circumferential core isolation around critical ventricular tachycardia circuit elements [30], or ‘homogenization’ of the scar with extensive overlapping ablation lesions [31]. de Chillou *et al.* [32]

Table 2. Recently published data on substrate-based ablation approaches for ablation of ischemic ventricular tachycardia

Technique	Patient selection	Patients	Men	Age (years)	Ischemic heart disease	LVEF (%)	No. of VTs induced at baseline	Epicardial ablation	RF time (min)	Procedure time (min)	Adverse events	Noninducible	Mean or median follow-up (months)	VT recurrence	Death
Jais <i>et al.</i> [27]	Unselected	70	90%	67 ± 11	80%	35 ± 10	2 (1–3)	30%	23 ± 11	148 ± 73	9%	70%	22 (14–27)	46%	19%
Vergara <i>et al.</i> [28]	Selected	50	94%	66 ± 10	72%	32 ± 9	2.8 (1–10)	42%	N/A	N/A	N/A	80%	13 ± 4	20%	5%
Tilz <i>et al.</i> [29]	Selected	12	100%	54 ± 8	100%	32 ± 13	3 ± 2	0%	53 ± 15	195 ± 64	N/A	92%	16 (10–26)	30%	0%
Tzou <i>et al.</i> [30]	Selected	44	95%	63 ± 14	73%	31 ± 13	3 ± 2	11%	N/A	326 ± 121	2%	82%	17.5 ± 9	14%	N/A
Di Biase <i>et al.</i> [31]	Unselected	43	75%	62 ± 8	100%	24 ± 8	2 (2–5)	100%	74 ± 21	288 ± 90	2%	100%	21 (19–25)	19%	2%
de Chillou <i>et al.</i> [32]	Selected	10	80%	71 ± 11	100%	37 ± 14	N/A	0%	10 ± 9	208 ± 62	0%	80%	65 ± 6	33%	40%
Berruezo <i>et al.</i> [33]	Unselected	101	91%	65 ± 12	74%	36 ± 13	N/A	27%	28 ± 16	227 ± 69	7%	78%	21 (11–29)	26%	9%

LAVA, local abnormal ventricular activity; LVEF, left ventricular ejection fraction; N/A, not available; RF, radiofrequency; VT, ventricular tachycardia.

located the critical isthmus by pace-mapping in sinus rhythm in a selected population with hemodynamically stable ventricular tachycardia, and Berruezo *et al.* [33] recently suggested that ‘dechanneling’ of the scar specifically targets channels relevant to the ventricular tachycardia. These investigators targeted the earliest of all recorded ‘late’ potentials in the scar border zone during sinus rhythm under the assumption that these areas represent entrance sites of conduction channels. Ablation at the entrance sites eliminated the conducting channels, thereby limiting the total ablations necessary for abolishing conducting channels in the scar. Tung *et al.* demonstrated that local ablation can modify electrical activity in scar regions remote from the ablation site [34]. However, the optimal strategy resulting in effective ablation but at the same time minimizing unnecessary radiofrequency application and procedure time is still not known. Anatomically ablating unexcitable scar might not be necessary, and abnormal electrograms may arise from tissue remote from the ablation catheter [35].

At our center, substrate modification of the scar is performed, targeting regions of slow conduction evident as late or fractionated potentials. We aim for sites where pace-mapping approximates the morphology of an induced clinical ventricular tachycardia and has a stimulus to QRS delay >40 ms (Fig. 1). Ablation is usually performed with a contact-sensing catheter aiming for 10–20 g of contact force and impedance drop of 10–15 Ω during ablation. As an acute endpoint, we aim to achieve noninducibility of all sustained monomorphic ventricular tachycardia by rendering the whole scar or scar areas felt to be involved in ventricular tachycardia circuits unexcitable to pacing (10 mA at 2 ms pulse width) or to disconnect them from healthy myocardium.

Hemodynamic support during ventricular tachycardia ablation

Hemodynamic support using percutaneous left ventricular assist devices (pLVADs) with an Impella Recover 2.5 (Abiomed, Inc., Danvers, Massachusetts, USA), a TandemHeart device (Cardiac Assist, Inc., Pittsburgh Pennsylvania, USA) or extracorporeal membrane oxygenators may offer an alternative for selected high-risk patients and may allow mapping of fast ventricular tachycardia in the setting of poor ventricular function or significant coronary artery disease.

Recent small retrospective series showed that pLVAD support is feasible during ablation [36–38]. Ventricular tachycardias could be mapped

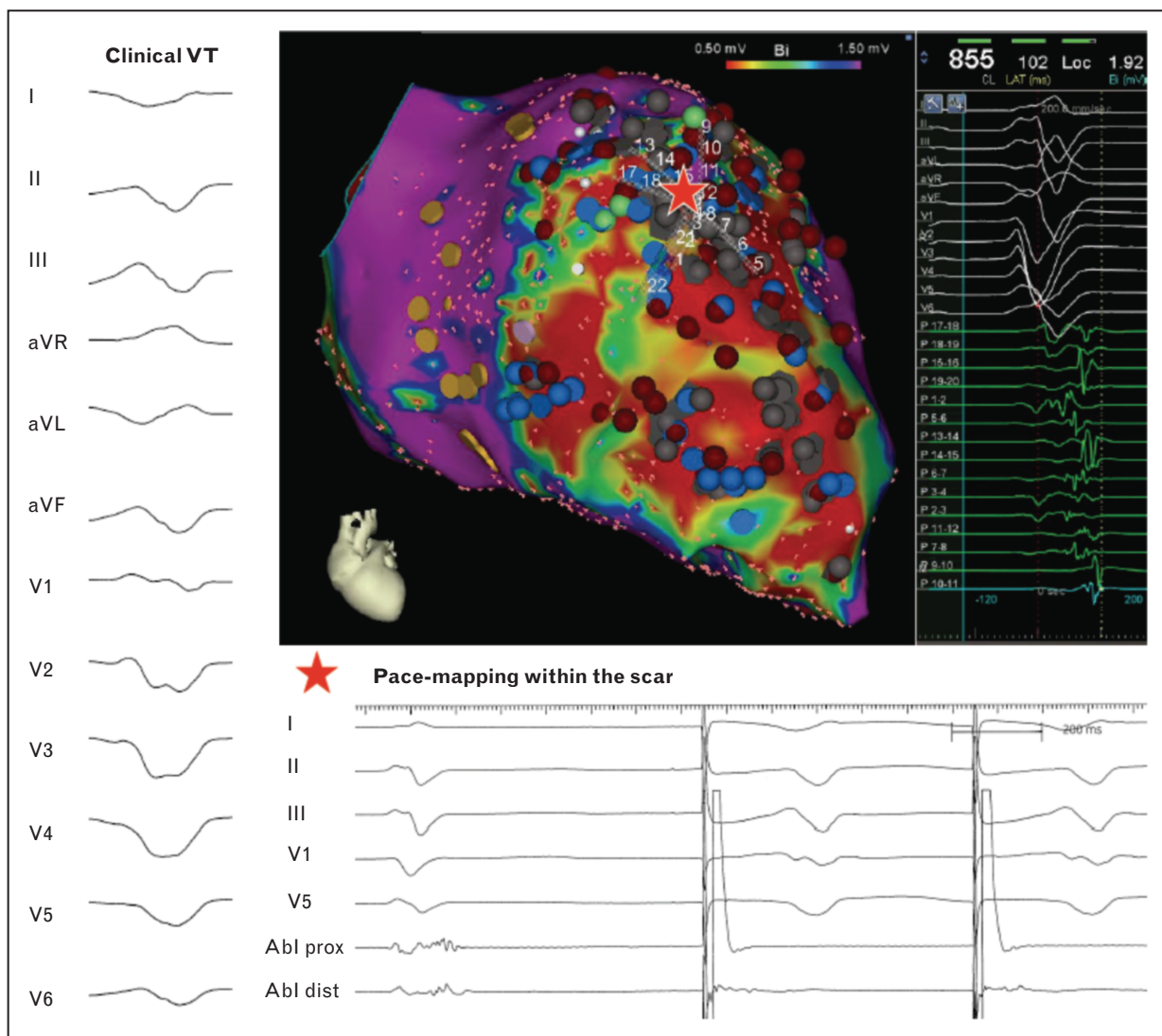


FIGURE 1. Example of an endocardial voltage map of the left ventricle that shows a large anteroseptal scar. Areas in purple represent bipolar voltages of 1.5 mV or greater. Blue, green, and yellow represent decreasing gradients of voltage, with red representing areas with voltage less than 0.5 mV. Late and fractionated electrograms are recorded from a multispline catheter in the scar area (electrograms in green in the right panel of figure). Pacing shows a good match (bottom panel) to the clinical VT (left panel) with a long stimulus-to-QRS interval, consistent with a slowly conducting channel. Blue dots represent areas where late potentials were recorded and red dots represent points on ablation. Abl, ablation catheter; dist, distal; prox, proximal; VT, ventricular tachycardia.

for relatively longer periods of time and were terminated by radiofrequency ablation more often compared with a control group without hemodynamic support or with an intraaortic balloon pump. However, there was no difference in acute procedural success or ventricular tachycardia recurrence rates during follow-up. In addition, the magnetic motor of the Impella system can interfere with the electroanatomic mapping, especially during mapping in the ventricular outflow tracts. Extracorporeal membrane oxygenation offers biventricular support and does not interfere with the mapping systems. Extracorporeal membrane oxygenation does not

unload the left ventricle as compared with pLVAD but offers better hemodynamic support (4–6 l/min).

Epicardial ablation

When endocardial ablation fails, it is often because of the location of critical components of a ventricular tachycardia circuit deep in the myocardium or in the subepicardium. Epicardial ablation has become an essential component of ablation strategies, especially for ventricular tachycardia because of nonischemic cardiomyopathies, although proximity of major coronary vessels or the phrenic nerve

to critical sites may preclude adequate ablation [39]. In ischemic heart disease, epicardial ablation is usually performed after failed endocardial ablation. An initial endo-epicardial approach might be beneficial in selected patients. A recent small single-center study showed an association between an initial combined approach and less hospitalization and reablation during follow-up, but failed to show any differences in ventricular tachycardia recurrence or mortality [40].

Techniques for ablation of intramural substrate

Although the advent of cooled tip radiofrequency catheters and contact force monitoring has improved lesion formation, safe delivery of deep intramyocardial lesions remains a problem. Bipolar radiofrequency energy between two separate ablation catheters positioned on the septum from both ventricles has proven to be beneficial in isolated cases and in preliminary studies [41–43].

Transcoronary ethanol ablation is an approach that is used in the setting of malignant refractory ventricular tachycardia when standard approaches fail [44]. Important considerations including coronary anatomy, risk of heart block, and risk of hemodynamic deterioration from the loss of functioning myocardium.

Needle electrodes for the creation of deeper intramural lesions show promise in early human studies where conventional catheter ablation techniques have been unsuccessful [45,46]. The use of a warm saline-enhanced needle electrode where radiofrequency energy is applied through the saline stream into deep myocardial layers shows promise in ablation of viable tissue in large myocardial infarct scars [47].

Procedural endpoints and outcome

Reported ventricular tachycardia recurrence rates after postinfarct ventricular tachycardia ablation procedures vary widely, but typically range between 30 and 50% in the larger studies. The most common origin of recurrent ventricular tachycardia is from areas adjacent to prior ablation lesions suggesting that the ventricular tachycardia exit points may have been altered by ablation [48].

The only endpoint criterion endorsed by current guidelines is noninducibility of ventricular tachycardia by programmed stimulation [5]. Absence of inducible ventricular tachycardia has been associated with better outcome [49], but has the limitation of the probabilistic nature of ventricular tachycardia inducibility. The predictive accuracy of

programmed stimulation has also been disappointing [50]. Ventricular tachycardia recurrence has been observed in 29% of patients who were rendered noninducible acutely [51]; this may reflect healing and contraction of initial radiofrequency lesions [52]. The various techniques of substrate-based ablation described above have introduced additional acute endpoints, such as elimination of all late or abnormal electrograms [27,31,53]. Development of more reliable endpoints for ventricular tachycardia ablation procedures remains a high priority.

The impact of ventricular tachycardia ablation on mortality is not well defined. A recent meta-analysis found a lower mortality in patients in whom ventricular tachycardia was rendered noninducible by programmed stimulation [49]. The analysis, however, did not adjust for differences in other risk predictors. A retrospective analysis performed by Yokokawa *et al.* [54] involving 1064 patients from seven centers suggested noninducibility as an independent predictor of survival. The study could not, however, evaluate the independent impact of ventricular tachycardia recurrence on mortality. A strong association between freedom from ventricular tachycardia and transplant-free survival during follow-up was demonstrated in a retrospective multicenter study involving 2061 patients [55], and patients with early ventricular tachycardia recurrence, within seven days after ablation, had a greater than two-fold increase in mortality in one series [56].

Complications of ventricular tachycardia ablation

The most common complications are related to vascular access and usually resolve spontaneously [57]. Cardiac tamponade occurs in about 1% of patients [58]. From a nationwide inpatient sample database, Palaniswamy *et al.* [59] reported major complication rates of 11.2% (including vascular: 6.9%, cardiac: 4.3%, and neurologic: 0.5%) and in-hospital mortality rates (1.6%) in the United States during 2002–2011. Most larger trials, however, reported lower complication rates [4,6,7], and at our center, complications related to the ablation are seen in about 5%. The majority of ablation-related mortality is because of uncontrolled ventricular arrhythmia and associated heart failure.

Future directions

Incorporating pathophysiological considerations beyond anatomic scar assessment in mapping of ventricular tachycardia substrate may prove useful. In a recent pilot study, Klein *et al.* [60] described a

novel mapping approach for postinfarction ventricular tachycardia ablation by integrating iodine-123-labeled-metaiodobenzylguanidine scintigraphy with voltage mapping, enabling identification of viable but denervated myocardial tissue.

The quality and durability of ablation lesions will continue to be a major determinant of clinical outcome. Technology allowing the monitoring of lesion size will continue to play an important role in improving outcomes. Electroporation is a nonthermal ablation modality capable of creating deep myocardial lesions. Animal studies have suggested its potential application in epicardial ablation [61].

CONCLUSION

Catheter ablation for ventricular tachycardia associated with ischemic cardiomyopathy can significantly reduce ventricular tachycardia recurrences, and this should translate into an improved quality of life for patients who are having recurrent ventricular tachycardia terminated by ICD shocks and should be considered early in the management of these patients. Complication rates are acceptable, but recurrence rates remain significant. Technological advances in mapping and ablation are expected to further improve success rates.

Acknowledgements

None.

Financial support and sponsorship

S.M.B. received educational grants from the University Hospital of Bern, Switzerland, and the Swiss Foundation for Pacemakers and Electrophysiology.

Conflicts of interest

R.M.J. receives consulting fees from St Jude Medical, Boston Scientific, and Biosense Webster, Inc.; W.G.S. is coholder of a patent for needle ablation that is assigned to Brigham and Women's Hospital.

REFERENCES AND RECOMMENDED READING

Papers of particular interest, published within the annual period of review, have been highlighted as:

- of special interest
- of outstanding interest

1. Moss AJ, Greenberg H, Case RB, *et al.* Long-term clinical course of patients after termination of ventricular tachyarrhythmia by an implanted defibrillator. *Circulation* 2004; 110:3760–3765.
 2. Poole JE, Johnson GW, Hellkamp AS, *et al.* Prognostic importance of defibrillator shocks in patients with heart failure. *N Engl J Med* 2008; 359:1009–1017.
 3. Sood N, Ruwald AC, Solomon S, *et al.* Association between myocardial substrate, implantable cardioverter defibrillator shocks and mortality in MADIT-CRT. *Eur Heart J* 2014; 35:106–115.
- The study showed an increased risk of mortality in patients who received appropriate shock after adjustment for relevant clinical variables. The association was not evident for antiarrhythmia pacing alone.
4. Stevenson WG, Wilber DJ, Natale A, *et al.* Irrigated radiofrequency catheter ablation guided by electroanatomic mapping for recurrent ventricular tachycardia after myocardial infarction: the multicenter thermocool ventricular tachycardia ablation trial. *Circulation* 2008; 118:2773–2782.
 5. Aliot EM, Stevenson WG, Almendral-Garrote JM, *et al.* EHRA/HRS expert consensus on catheter ablation of ventricular arrhythmias: developed in a partnership with the European Heart Rhythm Association (EHRA), a Registered Branch of the European Society of Cardiology (ESC), and the Heart Rhythm Society (HRS); in collaboration with the American College of Cardiology (ACC) and the American Heart Association (AHA). *Heart Rhythm* 2009; 6:886–933.
 6. Reddy VY, Reynolds MR, Neuzil P, *et al.* Prophylactic catheter ablation for the prevention of defibrillator therapy. *N Engl J Med* 2007; 357:2657–2665.
 7. Kuck KH, Schaumann A, Eckardt L, *et al.* Catheter ablation of stable ventricular tachycardia before defibrillator implantation in patients with coronary heart disease (VTACH): a multicentre randomised controlled trial. *Lancet* 2010; 375:31–40.
 8. Bunch TJ, Weiss JP, Crandall BG, *et al.* Patients treated with catheter ablation for ventricular tachycardia after an ICD shock have lower long-term rates of death and heart failure hospitalization than do patients treated with medical management only. *Heart Rhythm* 2014; 11:533–540.
 9. Dinov B, Arya A, Bertagnolli L, *et al.* Early referral for ablation of scar-related ventricular tachycardia is associated with improved acute and long-term outcomes: results from the Heart Center of Leipzig Ventricular Tachycardia Registry. *Circ Arrhythm Electrophysiol* 2014; 7:1144–1151.
- This important study suggests that catheter ablation early after the first documented ventricular tachycardia is associated with lower recurrence rates and improved survival and shows an association between ventricular tachycardia recurrence and mortality.
10. Tanaka Y, Genet M, Chuan Lee L, *et al.* Utility of high-resolution electroanatomic mapping of the left ventricle using a multispline basket catheter in a swine model of chronic myocardial infarction. *Heart Rhythm* 2015; 12:144–154.
 11. Hsia HH, Lin D, Sauer WH, *et al.* Anatomic characterization of endocardial substrate for hemodynamically stable reentrant ventricular tachycardia: identification of endocardial conducting channels. *Heart Rhythm* 2006; 3:503–512.
 12. Chopra N, Tokuda M, Ng J, *et al.* Relation of the unipolar low-voltage penumbra surrounding the endocardial low-voltage scar to ventricular tachycardia circuit sites and ablation outcomes in ischemic cardiomyopathy. *J Cardiovasc Electrophysiol* 2014; 25:602–608.
 13. Andreu D, Ortiz-Perez JT, Boussy T, *et al.* Usefulness of contrast-enhanced cardiac magnetic resonance in identifying the ventricular arrhythmia substrate and the approach needed for ablation. *Eur Heart J* 2014; 35:1316–1326.
 14. Wijnmaalen AP, van der Geest RJ, van Huls van Taxis CF, *et al.* Head-to-head comparison of contrast-enhanced magnetic resonance imaging and electroanatomical voltage mapping to assess post-infarct scar characteristics in patients with ventricular tachycardias: real-time image integration and reversed registration. *Eur Heart J* 2011; 32:104–114.
 15. Yamashita S, Sacher F, Mahida S, *et al.* Role of high-resolution image integration to visualize left phrenic nerve and coronary arteries during epicardial ventricular tachycardia ablation. *Circ Arrhythm Electrophysiol* 2015; 8:371–380.
 16. Nazarian S, Bluemke DA, Halperin HR. Applications of cardiac magnetic resonance in electrophysiology. *Circ Arrhythm Electrophysiol* 2009; 2:63–71.
 17. Ikeda A, Nakagawa H, Lambert H, *et al.* Relationship between catheter contact force and radiofrequency lesion size and incidence of steam pop in the beating canine heart: electrogram amplitude, impedance, and electrode temperature are poor predictors of electrode-tissue contact force and lesion size. *Circ Arrhythm Electrophysiol* 2014; 7:1174–1180.
 18. Hoffmayer KS, Gerstenfeld EP. Contact force-sensing catheters. *Curr Opin Cardiol* 2015; 30:74–80.
 19. Sacher F, Wright M, Derval N, *et al.* Endocardial versus epicardial ventricular radiofrequency ablation: utility of in vivo contact force assessment. *Circ Arrhythm Electrophysiol* 2013; 6:144–150.
 20. Mizuno H, Vergara P, Maccabelli G, *et al.* Contact force monitoring for cardiac mapping in patients with ventricular tachycardia. *J Cardiovasc Electrophysiol* 2013; 24:519–524.
 21. Stevenson WG, Khan H, Sager P, *et al.* Identification of reentry circuit sites during catheter mapping and radiofrequency ablation of ventricular tachycardia late after myocardial infarction. *Circulation* 1993; 88:1647–1670.
 22. Stevenson WG, Friedman PL, Sager PT, *et al.* Exploring postinfarction reentrant ventricular tachycardia with entrainment mapping. *J Am Coll Cardiol* 1997; 29:1180–1189.
 23. Morady F, Kadish A, Rosenheck S, *et al.* Concealed entrainment as a guide for catheter ablation of ventricular tachycardia in patients with prior myocardial infarction. *J Am Coll Cardiol* 1991; 17:678–689.
 24. Kocovic DZ, Harada T, Friedman PL, Stevenson WG. Characteristics of electrograms recorded at reentry circuit sites and bystanders during ventricular tachycardia after myocardial infarction. *J Am Coll Cardiol* 1999; 34:381–388.

25. Bogun F, Bahu M, Knight BP, *et al.* Response to pacing at sites of isolated diastolic potentials during ventricular tachycardia in patients with previous myocardial infarction. *J Am Coll Cardiol* 1997; 30:505–513.
26. Harada T, Stevenson WG, Kocovic DZ, Friedman PL. Catheter ablation of ventricular tachycardia after myocardial infarction: relation of endocardial sinus rhythm late potentials to the reentry circuit. *J Am Coll Cardiol* 1997; 30:1015–1023.
27. Jais P, Maury P, Khairy P, *et al.* Elimination of local abnormal ventricular activities: a new end point for substrate modification in patients with scar-related ventricular tachycardia. *Circulation* 2012; 125:2184–2196.
28. Vergara P, Trevisi N, Ricco A, *et al.* Late potentials abolition as an additional technique for reduction of arrhythmia recurrence in scar related ventricular tachycardia ablation. *J Cardiovasc Electrophysiol* 2012; 23:621–627.
29. Tiltz RR, Makimoto H, Lin T, *et al.* Electrical isolation of a substrate after myocardial infarction: a novel ablation strategy for unmappable ventricular tachycardias: feasibility and clinical outcome. *Europace* 2014; 16:1040–1052.
30. Tzou WS, Frankel DS, Hegeman T, *et al.* Core isolation of critical arrhythmia elements for treatment of multiple scar-based ventricular tachycardias. *Circ Arrhythm Electrophysiol* 2015; 8:353–361.
31. Di Biase L, Santangeli P, Burkhardt DJ, *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; 60:132–141.
32. de Chillou C, Groben L, Magnin-Poull I, *et al.* Localizing the critical isthmus of postinfarct ventricular tachycardia: the value of pace-mapping during sinus rhythm. *Heart Rhythm* 2014; 11:175–181.
33. Berrueto A, Fernandez-Armenta J, Andreu D, *et al.* Scar dechanneling: new method for scar-related left ventricular tachycardia substrate ablation. *Circ Arrhythm Electrophysiol* 2015; 8:326–336.
34. Tung R, Mathuria NS, Nagel R, *et al.* Impact of local ablation on interconnected channels within ventricular scar: mechanistic implications for substrate modification. *Circ Arrhythm Electrophysiol* 2013; 6:1131–1138.
35. Baldinger SH, Nagashima K, Kumar S, *et al.* Electrogram analysis and pacing are complimentary for recognition of abnormal conduction and far-field potentials during substrate mapping of infarct-related ventricular tachycardia. *Circ Arrhythm Electrophysiol* 2015; 8:874–888.
36. Aryana A, Gearoid O'Neill P, Gregory D, *et al.* Procedural and clinical outcomes after catheter ablation of unstable ventricular tachycardia supported by a percutaneous left ventricular assist device. *Heart Rhythm* 2014; 11:1122–1130.
37. Miller MA, Dukkupati SR, Mittnacht AJ, *et al.* Activation and entrainment mapping of hemodynamically unstable ventricular tachycardia using a percutaneous left ventricular assist device. *J Am Coll Cardiol* 2011; 58:1363–1371.
38. Reddy YM, Chinitz L, Mansour M, *et al.* Percutaneous left ventricular assist devices in ventricular tachycardia ablation: multicenter experience. *Circ Arrhythm Electrophysiol* 2014; 7:244–250.
39. Baldinger SH, Kumar S, Barbhaiya CR, *et al.* Epicardial radiofrequency ablation failure during ablation procedures for ventricular arrhythmias: reasons and implications for outcomes. *Circ Arrhythm Electrophysiol* (in press).
40. Izquierdo M, Sanchez-Gomez JM, Ferrero de Loma-Osorio A, *et al.* Endo-epicardial versus only-endocardial ablation as a first line strategy for the treatment of ventricular tachycardia in patients with ischemic heart disease. *Circ Arrhythm Electrophysiol* 2015; 8:882–889.
41. Gizurason S, Spears D, Sivagangabalan G, *et al.* Bipolar ablation for deep intra-myocardial circuits: human ex vivo development and in vivo experience. *Europace* 2014; 16:1684–1688.
42. Teh AW, Reddy VY, Koruth JS, *et al.* Bipolar radiofrequency catheter ablation for refractory ventricular outflow tract arrhythmias. *J Cardiovasc Electrophysiol* 2014; 25:1093–1099.
43. Berte B, Sacher F, Mahida S, *et al.* Impact of septal radiofrequency ventricular tachycardia ablation: insights from magnetic resonance imaging. *Circulation* 2014; 130:716–718.
44. Kumar S, Barbhaiya CR, Sobieszczyk P, *et al.* Role of alternative interventional procedures when endo- and epicardial catheter ablation attempts for ventricular arrhythmias fail. *Circ Arrhythm Electrophysiol* 2015; 8:606–615.
45. Sapp JL, Cooper JM, Zei P, Stevenson WG. Large radiofrequency ablation lesions can be created with a retractable infusion-needle catheter. *J Cardiovasc Electrophysiol* 2006; 17:657–661.
46. Sapp JL, Beeckler C, Pike R, *et al.* Initial human feasibility of infusion needle catheter ablation for refractory ventricular tachycardia. *Circulation* 2013; 128:2289–2295.
47. John RM, Connell J, Termin P, *et al.* Characterization of warm saline-enhanced radiofrequency ablation lesions in the infarcted porcine ventricular myocardium. *J Cardiovasc Electrophysiol* 2014; 25:309–316.
48. Yokokawa M, Desjardins B, Crawford T, *et al.* Reasons for recurrent ventricular tachycardia after catheter ablation of postinfarction ventricular tachycardia. *J Am Coll Cardiol* 2013; 61:66–73.
49. Ghanbari H, Baser K, Yokokawa M, *et al.* Noninducibility in postinfarction ventricular tachycardia as an end point for ventricular tachycardia ablation and its effects on outcomes: a meta-analysis. *Circ Arrhythm Electrophysiol* 2014; 7:677–683.
50. Santangeli P, Frankel DS, Marchlinski FE. End points for ablation of scar-related ventricular tachycardia. *Circ Arrhythm Electrophysiol* 2014; 7:949–960.
51. Della Bella P, Baratto F, Tsiachris D, *et al.* Management of ventricular tachycardia in the setting of a dedicated unit for the treatment of complex ventricular arrhythmias: long-term outcome after ablation. *Circulation* 2013; 127:1359–1368.
52. Frankel DS, Mountantonakis SE, Zado ES, *et al.* Noninvasive programmed ventricular stimulation early after ventricular tachycardia ablation to predict risk of late recurrence. *J Am Coll Cardiol* 2012; 59:1529–1535.
53. Silberbauer J, Oloriz T, Maccabelli G, *et al.* Noninducibility and late potential abolition: a novel combined prognostic procedural end point for catheter ablation of postinfarction ventricular tachycardia. *Circ Arrhythm Electrophysiol* 2014; 7:424–435.
54. Yokokawa M, Kim HM, Baser K, *et al.* Predictive value of programmed ventricular stimulation after catheter ablation of postinfarction ventricular tachycardia. *J Am Coll Cardiol* 2015; 65:1954–1959.
- The study showed that noninducibility after ventricular tachycardia ablation in patients with postinfarction ventricular tachycardia – the only endpoint criterion endorsed by current guidelines – is independently associated with lower mortality.
55. Tung R, Vaseghi M, Frankel DS, *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; 12:1997–2007.
- The study showed that noninducibility after ventricular tachycardia ablation in patients with postinfarction ventricular tachycardia – the only endpoint criterion endorsed by current guidelines – is independently associated with lower mortality.
56. Nagashima K, Choi EK, Tedrow UB, *et al.* Correlates and prognosis of early recurrence after catheter ablation for ventricular tachycardia due to structural heart disease. *Circ Arrhythm Electrophysiol* 2014; 7:883–888.
57. Bohnen M, Stevenson WG, Tedrow UB, *et al.* Incidence and predictors of major complications from contemporary catheter ablation to treat cardiac arrhythmias. *Heart Rhythm* 2011; 8:1661–1666.
58. Tokuda M, Kojodjojo P, Epstein LM, *et al.* Outcomes of cardiac perforation complicating catheter ablation of ventricular arrhythmias. *Circ Arrhythm Electrophysiol* 2011; 4:660–666.
59. Palaniswamy C, Kolte D, Harikrishnan P, *et al.* Catheter ablation of postinfarction ventricular tachycardia: ten-year trends in utilization, in-hospital complications, and in-hospital mortality in the United States. *Heart Rhythm* 2014; 11:2056–2063.
60. Klein T, Abdulghani M, Smith M, *et al.* Three-dimensional 123I-meta-iodobenzylguanidine cardiac innervation maps to assess substrate and successful ablation sites for ventricular tachycardia: feasibility study for a novel paradigm of innervation imaging. *Circ Arrhythm Electrophysiol* 2015; 8:583–591.
- The study describes a novel mapping approach by integrating molecular imaging of sympathetic denervation in ventricular tachycardia ablation procedures. Functional imaging beyond anatomic scar assessment in mapping of ventricular tachycardia substrate may prove useful.
61. Neven K, van Driel V, van Wessel H, *et al.* Safety and feasibility of closed chest epicardial catheter ablation using electroporation. *Circ Arrhythm Electrophysiol* 2014; 7:913–919.