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Ventricular Tachycardia: Past, Present & Future

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Take Home Messages

- Ventricular tachycardia (VT) is most commonly a re-entrant circuit sustained by channels of slow conduction in borderzone tissue between dense scar and healthy myocardium.
- Catheter ablation is a guideline recommended therapy for patients with symptomatic monomorphic VT, refractory to antiarrhythmic drugs.
- Recurrence remains common, reflecting limitations in mapping, ablation lesion creation/durability, and patient selection.
- Earlier intervention, improved risk stratification, emerging ablation technologies and machine learning approaches may improve outcomes

Introduction

More than a century after its first description, ventricular tachycardia (VT) remains a challenge, persisting despite—and in part because of—major advances in cardiovascular care. Improvements in coronary revascularisation, heart failure therapies, and use of implantable cardioverter defibrillator have improved survival, but have also resulted in a growing population susceptible to recurrent VT (1). Understanding how VT management has evolved and where limitations persist provides important insight into how future outcomes can be improved.

Past

Our modern understanding of VT began with the advent of the surface electrocardiogram. In 1902, Thomas Lewis reported in *The Lancet* a case of successive ventricular extrasystoles in a seaman suffering from "*precordial pain*, *dropsy* and *shortness of breath*" shortly after myocardial infarction (2). Lewis subsequently established the link between post-infarct scar and ventricular arrhythmia in 1909, after studying canine electrocardiograms following iatrogenic surgical coronary artery ligation (3).

Figure 1: Prominent Welsh cardiologist, Sir Thomas Lewis was the first to record a description of ventricular tachycardia and establish the causal relationship between post-infarct scar and VT. (Source: WikiCommons Creative Commons License).

While drugs like quinidine became widely used to treat VT (4), the first attempt at curative therapy was inspired by Lewis' earlier observation that scar was fundamental to the arrhythmogenic

process (5). In 1953, Charles Bailey treated a patient with frequent paroxysms of drug-resistant VT by ventriculotomy – surgical resection of an aneurysmal region of scar in the left ventricle, from a prior anterior myocardial infarction (6).

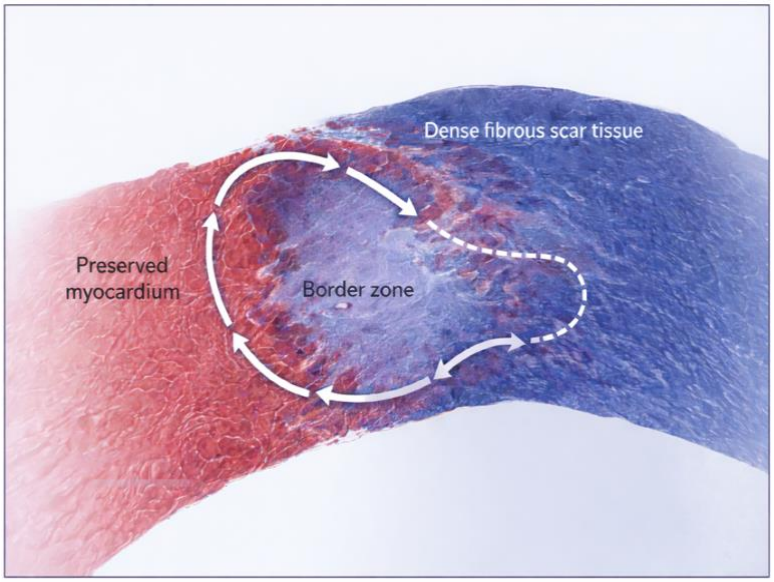


Figure 2: A histological depiction of ventricular tachycardia. The re-entrant circuit (white solid arrows) comprised of 3 distinct tissue types within myocardium: dense fibrotic scar (no conduction), healthy tissue (normal conduction) and areas of surviving 'borderzone' tissue (slow conduction). Within this borderzone, there often exists a narrow channel of conduction (dotted white line), which, if destroyed, breaks the circuit and prevents VT from propagating. (Source: Author)

Longer term follow-up revealed that VT frequently recurred following surgical resections (7). The development of intracardiac electrogram recordings during surgery subsequently enabled Mark Josephson and colleagues, in the late 1970s, to make several seminal observations. First, VT often remained inducible after resection of dense scar, suggesting that critical components of the arrhythmia circuit lay beyond these regions (8). Second, pre-systolic potentials observed while in VT, localised to areas of 'borderzone' – narrow channels (isthmuses) of slow conduction within surviving myocardium, between dense scar and healthy tissue (9). Third, extending surgical resection into this borderzone could terminate VT (10).

Present

Contemporary intracardiac electrograms are recorded by high-density multi-electrode catheters, creating high-resolution 3-dimensional reconstructions of the heart (13) (Figure 3). Extensive surgical resections have been replaced by precise, targeted percutaneous catheter ablation (14).

Yet, the fundamental principles remain unchanged: VT is sustained by slow conduction through isthmuses of viable myocardium within scar borderzone, and interruption of these pathways can abolish it (15,16).

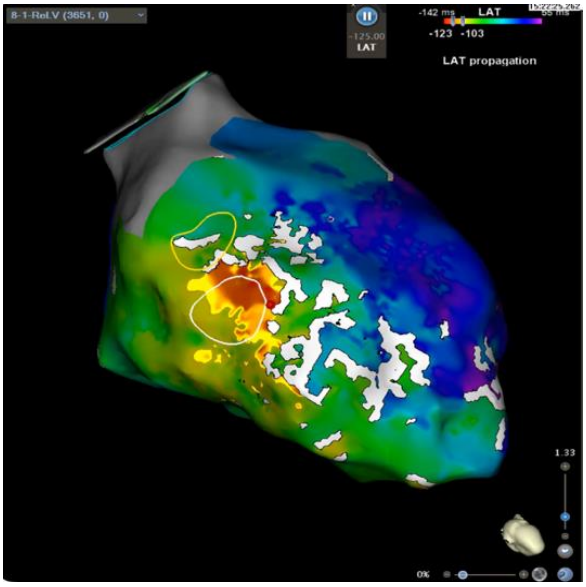
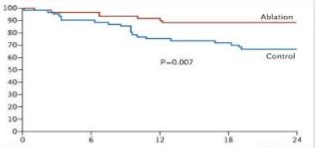
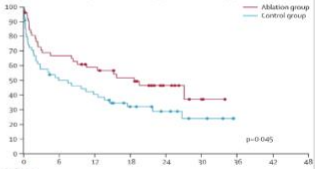
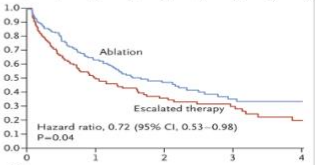
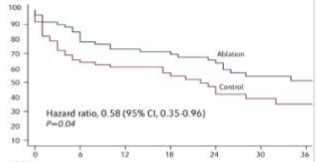
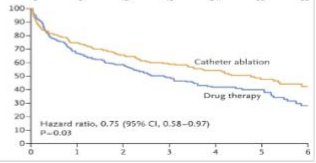


Figure 3: Example of a modern electroanatomical map of a patient in ventricular tachycardia during catheter ablation. (Source: Author)

Catheter ablation is now a guideline-recommended (Class I) therapy for VT in patients with structural heart disease refractory to antiarrhythmic drugs (17). Randomised trials consistently demonstrate reductions in VT recurrence, arrhythmia burden, and ICD therapies, with a gradual shift from reactive ablation towards earlier first-line intervention *(11,12,18–20)* (Table 1). However, these findings must be interpreted in the context of important limitations. Trial populations were predominantly restricted to stable ischaemic cardiomyopathy cohorts, limiting generalisability to routine clinical practice. Effect sizes may also be influenced, for instance in VANISH, by high crossover rates and challenges in standardising adherence, dose optimisation and addition of adjunctive antiarrhythmic drug therapy. Moreover, benefits were primarily driven by arrhythmic rather than mortality endpoints and despite this, remain modest, with approximately one-third of patients experiencing VT recurrence within one year of ablation (12).

Table 1: A comparison of trials evaluating catheter ablation of ventricular tachycardia

Trial (Year)	Population	Ablation Strategy	Comparator	Primary Outcome	Key Findings	
SMASH-VT (Reddy, NEJM 2007)	Prior MI, ICD indicated for secondary prevention	Prophylactic substrate ablation before or soon after ICD implantation	ICD alone	ICD therapies for VT/VF	Prophylactic ablation significantly reduced ICD therapies	
VTACH (Kuck, Lancet 2010)	Ischaemic cardiomyopathy, ICD planned	Early ablation prior to ICD implantation	ICD alone	Time to first VT/VF recurrence	Early ablation prolonged time to VT recurrence and reduced VT burden	
VANISH (Sapp, NEJM 2016)	Ischaemic cardiomyopathy, ICD, recurrent VT despite AADs	Ablation after VT recurrence on AADs	Escalation of AAD	Composite: death, VT storm, or ICD shock	Ablation superior to drug escalation, driven by reduced VT storm and ICD shocks	
PAUSE-SCD (Tung, Circ 2022)	Structural heart disease, secondary-prevention ICD planned	Early ablation at time of ICD implantation	ICD with deferred ablation	VT recurrence or ICD therapies	Early ablation reduced VT recurrence and ICD therapies across diverse substrates	
VANISH-II (Sapp, NEJM 2024)	Ischaemic cardiomyopathy, sustained VT, ICD eligible	First-line catheter ablation (before long-term AADs)	AAD therapy	Composite: death, VT storm, appropriate ICD shock, or sustained VT requiring intervention	First-line ablation reduced the composite endpoint, driven by fewer VT episodes and ICD therapies	

MI = myocardial infarction, ICD = implantable cardiac defibrillator, VT = ventricular tachycardia, AAD = antiarrhythmic drugs Source: adapted from Reddy (16), Kuck

Why do recurrence rates remain high?

The first limitation lies in 'mapping'. Successful ablation depends on VT being both inducible, and haemodynamically tolerable for sufficient time to accurately delineate the re-entrant circuit (14). General anaesthesia, and antiarrhythmic therapy may suppress inducibility (21,22), while comorbidity and frailty often limits haemodynamic tolerance (23). In such cases, operators often rely on 'substrate mapping', using voltage surrogates to infer scar and borderzone location, which can be imprecise (24).

Secondly, even when identified, the critical isthmus sustaining the VT circuit may be inaccessible. Radiofrequency ablation (RFA) is the commonest energy modality used in VT ablation (14). Where arrhythmogenic tissue lies deep within myocardium (for instance, in non-ischaemic cardiomyopathy), endocardial RFA lesions can be insufficiently deep or durable (25,26) (Figure 4). Recovery of conduction through partially injured tissue may contribute to early recurrence (27).

Finally, timing is crucial. Early VT ablation is associated with higher rates of freedom from arrhythmia because intervention occurs before progressive remodelling transforms a discrete, targetable arrhythmogenic substrate into one that is diffuse and poorly defined (28,29). Recurrent VT itself may accelerate this process by imposing haemodynamic stress, exacerbating myocardial ischaemia, and triggering sympathetic activation (30). ICD shocks, while lifesaving, are also associated with myocardial injury and arrhythmogenesis (31,32). Collectively, these insults create a self-perpetuating cycle in which VT begets VT.

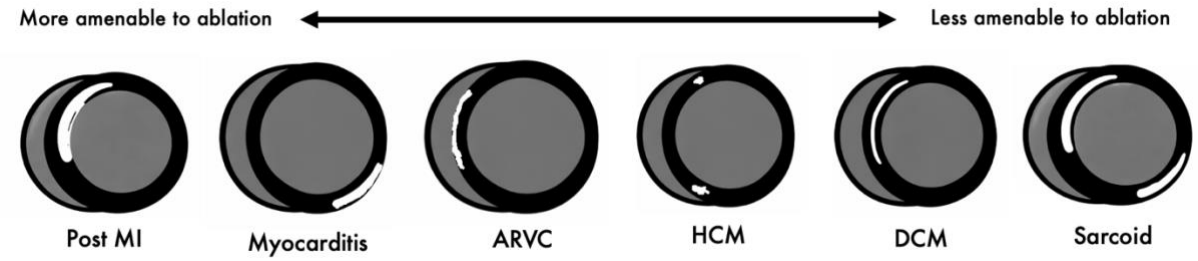


Figure 4: Examples of scar types in a range of conditions, arranged by order of ablation success likelihood. MI = myocardial infarction. ARVC = arrhythmogenic right ventricular cardiomyopathy. HCM = hypertrophic cardiomyopathy. DCM = dilated cardiomyopathy. (Source: adapted from Marholdt. et al (32), Designed by Author).

Future

Advances in ablation technology, imaging, and computational analysis offer the potential to address several enduring limitations of VT ablation. Novel energy sources such as pulsed field ablation may enable deeper, homogeneous, and potentially more durable lesions (33). While experience in VT remains early, these technologies may be particularly relevant for intramural substrates that are poorly treated by conventional RFA (Figure 4).

For patients with refractory VT who are unsuitable for, or have failed, catheter-based approaches, alternative strategies such as stereotactic radiotherapy and stellate ganglion block are emerging. In select patients with VT, early studies show meaningful reductions in arrhythmia burden, although longer-term efficacy and late toxicity require further evaluation (34,35).

Parallel developments in procedural risk stratification and peri-procedural support are also reshaping VT ablation. Predictive scoring systems may allow earlier identification of patients at

risk of haemodynamic compromise, facilitating pre-emptive mechanical circulatory support, and enabling mapping and ablation in otherwise prohibitive cases (23).

Artificial intelligence and machine-learning approaches may further refine VT management by integrating surface electrocardiograms, imaging data, and intracardiac electrograms to identify critical isthmuses without reliance on sustained VT induction (34). If validated, such approaches could reduce procedural complexity, time, and potentially extend the benefits of ablation to a broader patient population.

Conclusion

Our conceptual understanding of VT has remained consistent, even as therapeutic strategies have greatly evolved. Despite substantial technological advances, VT treatment remains limited by challenges in mapping, ablation, and patient selection. Emerging ablation technologies, shifting towards earlier intervention, and integration with artificial intelligence, offer a path toward more precise, and effective treatment.

Disclosures

None.

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