EP Lab Digest

20 YEARS

A product, news & clinical update for the electrophysiology professional

EP Lab Spotlight

Morristown Medical Center

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In this issue we welcome back Morristown Medical Center, the first EP program to be featured in *EPLD*'s Spotlight Interview, in September 2001.

When was the EP program started at your institution? By whom?

Stephen Winters, MD, initiated a full-time, hospital-based EP program at Atlantic Health System's Morristown Medical Center (MMC) in November 1991. He came from the Mount Sinai Medical Center in New York where he was co-director of the EP program and director of the arrhythmia clinic.

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In This Issue

Cover Story

Lead Extraction: From Traction to Technology

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As we celebrate the 20th year of *EP Lab Digest*, the age-old expression "need necessitates invention" could not be more accurate, and in no field of cardiology is this more evident than the evolution of lead extraction. Within a decade of the initial pacemaker implant in October 1958[1], the need to remove these devices was realized and the concept of lead extraction was born. Case reports of pacemaker lead extraction, for indications such as infection and lead malfunction, began to appear in the literature in the late 1960s.[2] The earliest methods available were surgical intervention and manual traction. Surgical techniques ranged from limited thoracotomy to open chest procedures requiring cardiopulmonary bypass.[3] The surgical approach was usually seen as a final solution given the significant morbidity and prolonged recovery times associated with these procedures.

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Cover Story

History of Ventricular Tachycardia Ablation

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Ventricular arrhythmias can be responsible for worsening heart failure, painful shocks from implanted defibrillators, and sudden death in patients with structural heart disease and inherited channelopathies. Antiarrhythmic drugs often provide incomplete control of ventricular tachycardia (VT), worsen underlying heart failure, and expose the patient to risk of toxicities. For these reasons, catheter ablation for VT has evolved into a critically important part of arrhythmia management for patients with structural heart disease. Figure 1 demonstrates the critical points in the history of VT catheter ablation related to the historical and technological limitations in delivery of ablative strategies to the target tissue of interest, and shows the progress in understanding the underlying mechanisms of arrhythmogenesis.

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HMP Global

History of Ventricular Tachycardia Ablation

Uyanga Batnyam, MD, and Usha Tedrow, MD, MSc

Surgical VT Ablation

The first reported left ventricular aneurysmectomy was performed in 1956 in a patient with drug-refractory VT; the VT remained controlled despite discontinuation of quinidine after the surgery.[1] LV aneurysmectomy with or without coronary bypass grafting and infarctectomy was performed for patients with ischemic cardiomyopathy for over a decade, but carried high perioperative mortality rates, and VT recurrence was frequent.[2]

In the 1970s, Josephson and colleagues described ventricular subendocardial resection.[3] Operative epicardial mapping with a handheld electrode with adjunctive endocardial mapping showed tissue important to the VT circuit at the border of the aneurysm, often in areas where complete resection was not surgically feasible. Therefore, endocardial resection of these areas was performed, and for areas not easily resected, adjunctive surgical cryoablation was introduced. Although this technique was more successful in eliminating VT when compared to aneurysmectomy alone,[4,5] the perioperative mortality remained high.

Catheter Ablation and DC Shock Ablation

After Scheinman and colleagues reported the first successful catheter-based direct current (DC) shock ablation in humans for supraventricular tachycardia in 1982,[6] Hartzler performed the first in-human DC shock ablations in 3 patients with drug-refractory VT. One patient had idiopathic VT and the other 2 patients had infarct-related VT, which continued despite subendocardial resection.[7] Hartzler used a 7 French quadripolar catheter to deliver 300J DC shocks at the earliest activation of VT with successful elimination of VT for an acute 2-3 month follow-up period. In both patients with structural heart disease, VT recurred but was more easily medically controlled. Catheter ablation with DC shock ablation clearly represented a major step forward from surgical ablation techniques, but the collateral damage with DC shock was unpredictable, and the exact target tissue to eliminate VT based on limited catheter mapping data was unclear.

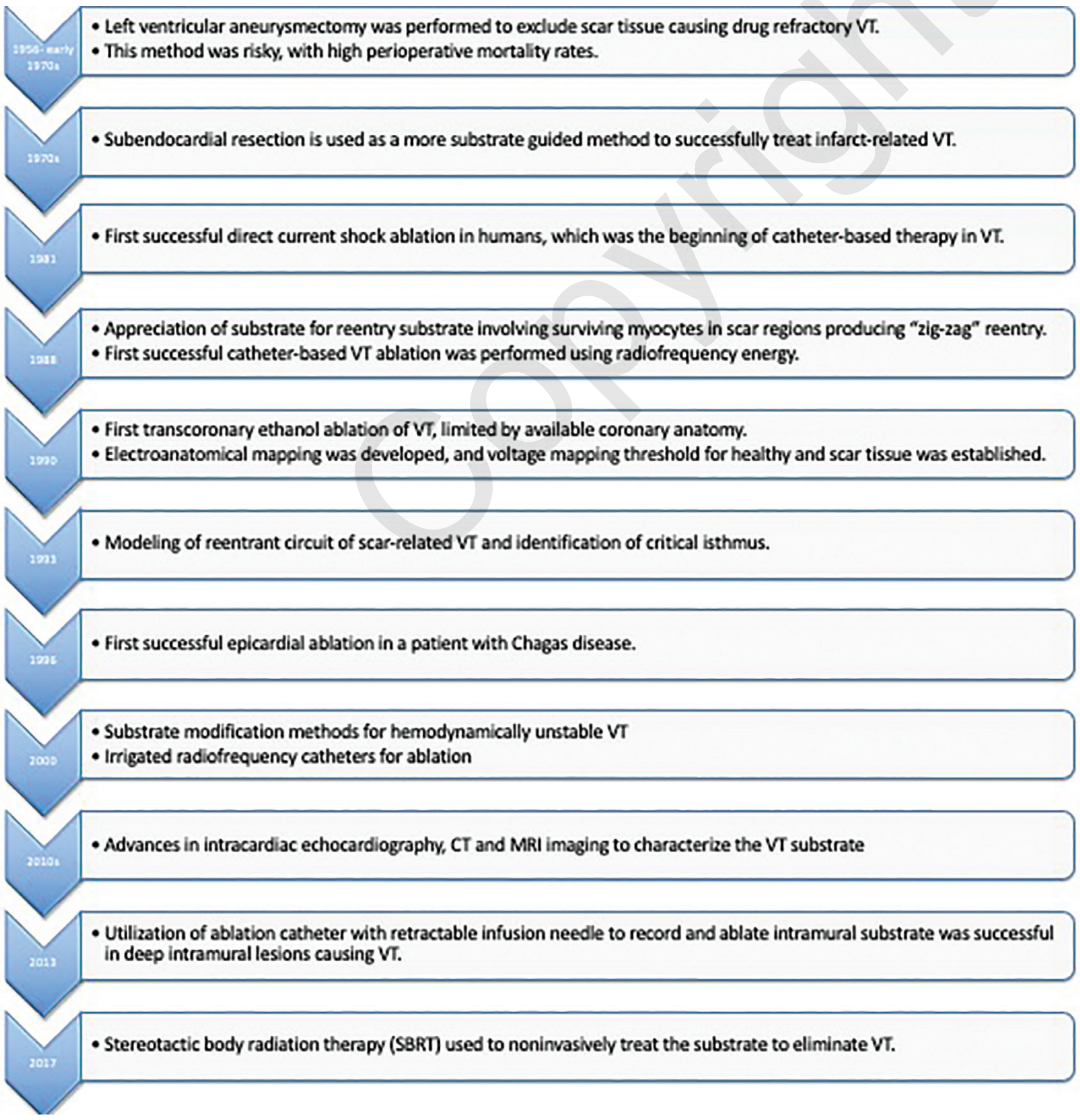

Figure 1. Timeline of historical advancement in VT ablation.

Understanding the Target and Underlying Substrate

Josephson and colleagues observed areas of slow conduction through diseased myocardium as fractionated signals on cardiac electrogram recordings and proposed that these signals represented the substrate for scar-related VT.[8] They further described localization of VT by utilizing 12-lead EKG and use of pace mapping in the 1980s.[9] de Bakker and colleagues described signal propagation in infarcted myocardium using an elegant *ex vivo* model in the 1990s.[10] They observed surviving myocytes interwoven with fibrosis forming a complex network of connection tracts, resulting in a "zigzag" course of activation. These important observations provided the physiology behind the observation of signal fractionation in areas of ventricular slow conduction.

Also in the 1990s, Stevenson and colleagues pioneered the understanding of the reentrant circuit of scar-related VT using intracardiac recordings, computer modeling, and entertainment mapping. These important observations helped to determine the critical portions of the scar-related VT circuit: diseased myocardium with slow conduction zones, inner and outer loops, bystander areas, and identification of the critical isthmus.[11] Additionally, in the 1990s, electroanatomical mapping was developed,[12] and voltage thresholds for scar tissues and healthy myocardium were established.[13] (Figure 2)

Radiofrequency Ablation

DC shock ablation was used in the treatment of VT until the late 1980s. However, DC shock ablation resulted in high-energy discharge, needed general anesthesia, was associated with collateral damage and barotrauma including the effect on LV function. It also had potential for arrhythmogenicity due to heterogeneous tissue injury. Scheinman and colleagues successfully performed radiofrequency (RF) ablation in the late 1980s in patients with drug-refractory SVT,[14] and this was followed by successful VT ablation using RF energy in 1988.[15]

RF energy is superior to DC shock ablation in several ways. It delivers energy in a relatively small and controlled area. There are more predictable and homogeneous areas of coagulation necrosis of the tissue where energy is applied. Further, the procedure can be performed on conscious patients without extracardiac stimulation. In the early 2000s, open irrigated tip and larger tip catheters were explored to create deeper lesions.[16] Initial limitations of open irrigated catheters included fluid overload and heart failure exacerbation that needed intraprocedural attention. However, benefits of open irrigation included reduction in coagulum formation and reduced risk of thromboembolism compared to large-tip catheters. Additionally, mapping could be performed with relatively improved fidelity compared to large-tip catheters, owing to the relatively smaller electrode size with open irrigated ablation catheters.

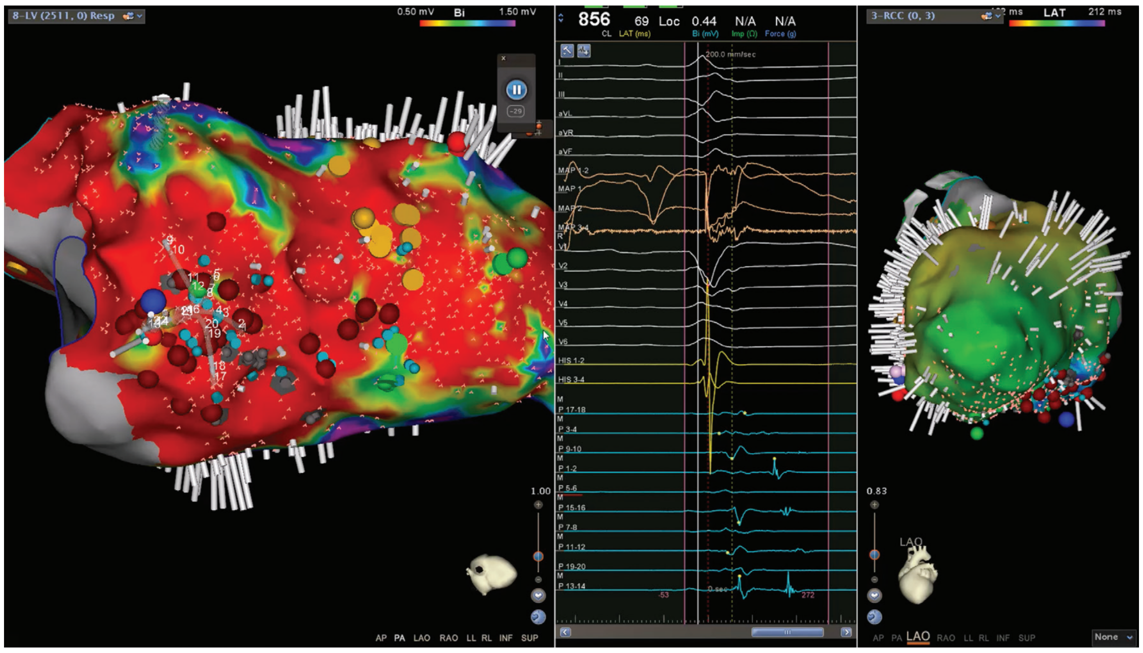

Figure 2. Depiction of a large inferior scar on electroanatomic mapping. Note late potentials from multielectrode catheter during diastole which is the critical substrate for VT. *(Video available at eplabdigest.com)*

	Substrate	Description
Josephson et al, 1978[8]	Fractionated signals	Areas of slow conduction through diseased myocardium depict as fractionated signals on intracardiac electrogram recordings and proposed that these signals represented the substrate for scar-related VT
Josephson et al, 1980s[9]	VT localization on 12-lead EKG	Using QRS morphology on surface 12-lead EKG to localize origin of VT
de Bakker et al, 1993[10]	Zigzag conduction	Infarcted myocardium consist of diseased and surviving myocytes and surround fibrosis, causing a "zigzag" course of activation
Stevenson et al, 1993[11]	Modeling of circuit	Diseased myocardium has slow conduction zones, inner and outer loops, bystander areas. Identification of the critical isthmus is possible by entrainment mapping
Gepstein et al, Marchlinski et al, 1990s[12,13]	Electroanatomical mapping	Electroanatomical mapping and voltage threshold for identification of scar tissue
Marchlinski et al, 2000[13]	Substrate modification	Modifying substrate by creating a linear lesion mimicking surgical ablation in unstable and unmappable VT
Soejima et al, 2001[20]	Mapping unstable VT in sinus rhythm	Delivering fewer ablation lesions by targeting isthmus or likely exit of unstable VT guided by EP study using combination of electroanatomical mapping and pace mapping in sinus rhythm
Tzou et al, 2015[21]	Core isolation	Isolating areas of myocardium critical to VT circuit
Sacher et al, 2015[22]	LAVA	Local abnormal ventricular activities (LAVA), sharp local electrograms manifest as fractionated, double or multiple component signals before or after the QRS. LAVA in sinus rhythm was closely correlated with mid-diastolic potentials during VT
Jackson et al, 2015[23]	DEEP mapping	Decrement evoked potential (DEEP) mapping: substrate expanded to include areas with late potentials revealed by single extrastimuli
de Chillou et al, 2017[24]	Pace mapping in sinus rhythm	Pace mapping in sinus rhythm can be used to identify VT isthmuses or exit zones as compared to border zone or entrance zone depending on the percentage of QRS morphologic match
Tung et al, 2011[19] Anter et al, 2015[17] Tschabrunn et al, 2016[18]	Small electrode mapping	Multielectrode catheters allow identification of channels within an area of heterogeneous scar. Delays seen in sinus rhythm and areas of functional block are critical to VT circuits.

Figure 3. Important studies that enabled better understanding of substrate mapping in VT.

Further Advances in Substrate Mapping

In many cases, ventricular arrhythmias are hemodynamically unstable and require immediate termination. In these situations, mapping in VT is usually not feasible. Additionally, all possible VT circuits for a given patient may not be reproduced with programmed stimulation. Marchlinski and colleagues described substrate mapping and modification, and linear lesion mimicking surgical ablation in unstable VT.[13] Additionally, the infarcts can be extensive and the precise areas to deliver ablation lesions can be unclear. In the 1990s, multielectrode catheters were developed to help create dense, high-resolution maps to better identify local activation, often obscured by the larger electrode ablation catheter.[17,18] Detailed scar channels and late potentials could be identified with multielectrode catheters.[19] This ushered in a series of important studies on substrate mapping (Figure 3).[20-24]

Alternative Approaches to the Myocardium

Ablation of ventricular arrhythmias by using transcoronary ethanol injection to target coronary arteries supplying the origin of tachycardia was performed in early 1990s. However, it was not widely used given that the ventricular arrhythmias in ischemic cardiomyopathy can come from areas of myocardium that have already been infarcted. Also, the technique is highly limited by the coronary supply to the territory of interest.[25]

Endocardial mapping and ablation of VT can be limited by intramural or epicardial substrates, especially for patients with non-ischemic cardiomyopathy. Sosa and colleagues first explored percutaneous access to the pericardium and successfully ablated VT with epicardial origin in patients with Chagas' disease.[26] It was soon appreciated that other nonischemic and ischemic cardiomyopathies had VTs with critical tissue that could be targeted from the epicardium.[27]

Irrigated catheters traditionally use normal saline as an irrigant. Sauer and colleagues described that using half normal saline may increase lesion size and can be more effective in ablating deeper intramural substrates.[28,29] In recent years, successful elimination of VT by sequential unipolar ablation at two different sites directly opposite from the target area, or using a simultaneous bipolar ablation at the opposite surface of the myocardium, have also been reported.

Utilization of an ablation catheter with a retractable infusion needle to record and ablate intramural substrate has been explored to be a feasible and effective method in creating deeper lesions, and is gaining clinical evidence as an effective tool for patients who have failed conventional VT ablation.[30-32]

In 2012, stereotactic body radiotherapy (SBRT) was used for the first time by Zei and colleagues to treat drug-refractory VT in a patient with significant comorbidities.[33] Cuculich and colleagues have further applied and expanded the use of this therapy, first in a 5-patient series and then further in the Phase I/II ENCORE VT trial demonstrating SBRT is an effective treatment for ventricular arrhythmias.[34] SBRT delivers focused beams of radiation that provide ablative energy at the precise location of the determined target volume (Figure 4).[35] The patient does not require anesthesia or invasive catheter mapping. Long-term safety is currently being investigated.

Adjunctive Imaging

Substrate for VT can be endocardial, or extend to mid myocardium or epicardium. In patients with intramural scars, endocardial or epicardial ablation alone can be challenging to identify and ablate. Intracardiac echocardiography has been an important adjunct for safety and also for establishing registration for effective electroanatomical mapping. Emerging imaging technologies can help preprocedural planning, safety, and efficacy. Cardiac MRI can be used with gadolinium contrast to identify and localize scar tissue, coupled with electroanatomical mapping to identify areas of interest. Additionally, other imaging modalities such as cardiac computed tomography can be used to identify wall thinning, calcification, and myocardial fatty metaplasia. Advances in image processing help to bring these data into the mapping space and may improve ablation outcomes.[36,37] (Figure 5)

Large-Scale Clinical Trials

Evidence for VT ablation is evolving, and multiple randomized clinical trials have shown its efficacy in decreasing recurrence and burden of VT as well as preventing appropriate ICD shocks. In 2007, the SMASH-VT trial showed prophylactic catheter ablation is associated with reduced ICD therapy and VT storm as compared

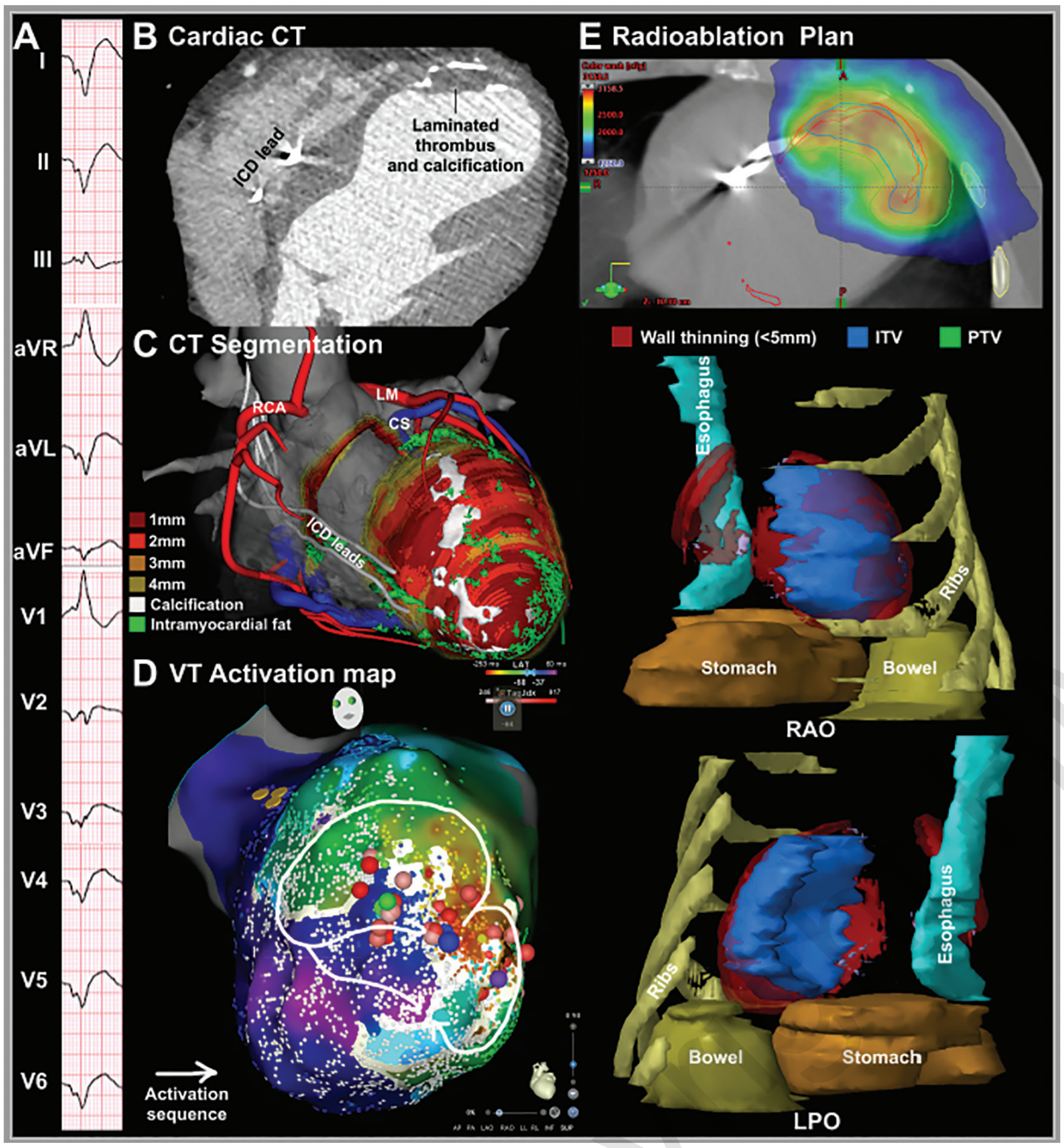

Figure 4. **Procedural planning for stereotactic body radiotherapy in a patient with ischemic cardiomyopathy and scar-related VT. Identifying the area of interest by combining information from surface 12-lead EKG, cardiac CT scan, and activation mapping to precisely localize the critical VT circuit for radiotherapy delivery.**

(Reprinted from Qian PC, et al. Substrate modification using stereotactic radioablation to treat refractory ventricular tachycardia in patients with ischemic cardiomyopathy. JACC Clin Electrophysiol. 2021 Jul 27;S2405-500X(21)00599-5, with permission from Elsevier.)

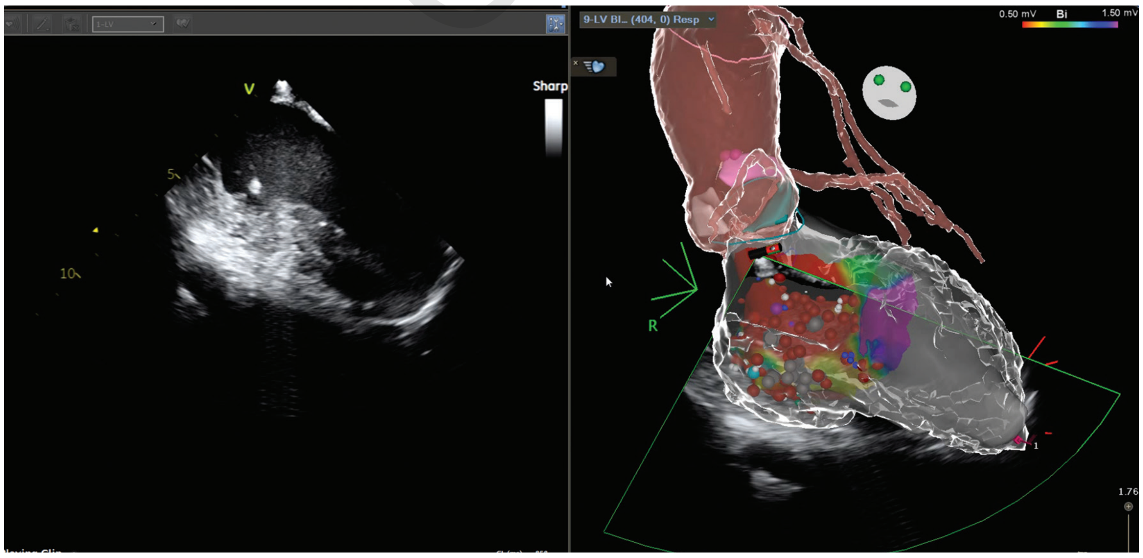

Figure 5. **Illustration of intracardiac ultrasound (ICE) and cardiac CT scan. ICE allows registration of pre-acquired CT image with ventricular geometry and aortic root. In this case, coronary arteries and bypass grafts are also seen on CT. *(Video available at eplabdigest.com)***

to patients with device therapy alone.[38] In 2016, the VANISH trial demonstrated ablation was superior to escalated medical therapy in patients with ischemic cardiomyopathy, ICD, and VT despite use of an antiarrhythmic drug.[39] IVTCC, a large-scale international collaborative registry of 2061 patients, demonstrated that VT ablation resulted in 70% freedom from VT recurrence with catheter ablation in patients with structural heart disease, as well as that freedom from VT recurrence was associated with improved transplant-free survival independent of heart failure severity.[40] However, the SMS trial in 2017 did not show benefit in prophylactic VT ablation in patients with sustained ventricular arrhythmia.[41] Most recently, BERLIN VT trial also compared preventive VT ablation prior to ICD implantation vs performing VT ablation in patients with multiple ICD therapy, and although there was no difference in all-cause mortality, there was a significant reduction in sustained VT and appropriate ICD therapy in the preventive ablation group.[42]

With currently available data, the indication for VT ablation is to decrease recurrence of VT, reduce VT burden, and reduce appropriate ICD shocks in patients with structural heart disease in whom antiarrhythmic medications are not effective, not tolerated, or contraindicated. Other more specialized indications also exist, such as epicardial ablation in selected patients with Brugada syndrome.[43] Looking at the published clinical trials, VT recurrence remains an important problem, and future work refining mechanistic understanding as well as improving ablation technology will be very important going forward.

Conclusion

Treatment of VT has evolved substantially since the first LV aneurysmectomy in 1956, and the first catheter-based RF ablation in 1988. We now have a much better understanding of the mechanisms of scar-related ventricular tachycardia, underlying substrate, and mapping techniques to identify the critical circuit of VT. Advancement and availability of mapping tools and technologies as well as different and advanced imaging modalities have enabled us to deliver more precise and successful treatment for ventricular arrhythmias. ■

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