13 Ventricular Tachycardia with Structural Heart Disease

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Ventricular tachycardia (VT) is a major cause of sudden cardiac death. The majority of malignant VTs which carry an elevated risk for sudden cardiac death (SCD) occur in patients with structural heart disease (SHD), and implantable cardioverter defibrillators (ICDs) are the mainstay of therapy [1]. In these individuals, catheter ablation is being increasingly performed as adjunctive therapy to prevent or reduce ICD therapies when antiarrhythmic drugs are ineffective or not desired. In addition, in developing countries, some VT patients with structural heart disease cannot afford the expense of ICD; antiarrhythmic drugs and catheter ablation may become the ultimate therapy to prevent VTs and the onset of SCD.

Electrophysiology (EP) procedures in patients with SHD, including catheter ablation, are traditionally performed under fluoroscopic guidance and are often complex and prolonged enough to involve a non-negligible radiation exposure, which may increase the risk of cancer and genetic anomaly [2]. In recent years, non-fluoroscopic three-dimensional (3D) mapping systems have been developed to guide ablation during EP procedures [3, 4]. Their use has certainly allowed to better understand and ablate complex arrhythmias, but they have been proven to confer the additional benefit of significantly reducing radiation exposure. In this chapter, we will discuss the mechanisms and management of VT in the setting of structural heart disease and discuss the role of catheter ablation and patient populations who are most likely to benefit from this treatment modality. Furthermore, the role of three-dimensional mapping systems in EP procedure is also been discussed.

Mechanism

In patients with SHD, the main challenge for catheter ablation of VT is the complex arrhythmogenicity of the myocardial scar. Electroanatomical remodeling of the scar that occurs in either ischemic or nonischemic SHD may prompt arrhythmias through different mechanisms: enhanced normal automaticity, abnormal automaticity, triggered activity induced by early or late afterdepolarizations, and various forms of reentry [5, 6].

Ectopic automaticity and ***triggered activity*** are likely causes of focal origin VTs, although small reentry circuits can often not be excluded. Automatic VTs can occur in structural heart disease, and automatic premature beats may initiate reentrant VTs. Triggered activity by delayed after depolarizations is the underlying mechanism for

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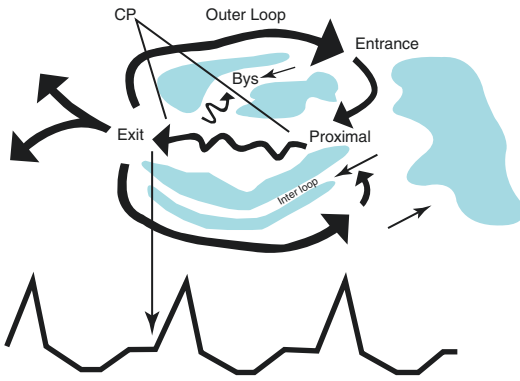


Fig. 13.1 Schematic reentry circuit of ventricular tachycardia (and its components). Conduction pathways can be seen surrounded by areas of scar

VT in the setting of digoxin toxicity, catecholaminergic polymorphic VT, idiopathic outflow tract ventricular arrhythmia (VA), and heart failure [7]. Unlike automaticity, triggered activity is not a self-generating rhythm. Instead, triggered activity occurs as a response to a preceding impulse (the trigger).

Reentry is the underlying mechanism for most sustained VA in the presence of structural heart disease (Fig. 13.1). The existence of structural reentrant substrates provides the rationale for VT ablation in scar-related VTs (Fig. 13.2) [8, 9]. Functional reentry around areas of functional block without anatomical obstacles can also occur. The mechanisms for functionally determined reentrant circuits include the leading circle type of reentry, anisotropic reentry, and spiral wave reentry [6, 10].

Assessment and Preparation Before RFCA

Before the procedure, patients should undergo a comprehensive clinical evaluation and a series of examinations to determine the underlying etiology and prognostic significance of VT. Analysis of the ***12-lead VT electrocardiogram (ECG)*** is essential for diagnosis. Clinical VT morphology helps to localize the exit site of the reentrant circuit from the protected isthmus, and helps with procedural planning [10].

Cardiac imaging plays a potentially important role for preprocedural assessment of cardiac anatomy and myocardial scar, intraprocedural integration of the structural and electrophysiological VT substrate, and post procedural assessment of the efficacy of ablation. Firstly, cardiac imaging could help to make preprocedural determination of the optimal access route. A priori information on cardiac anatomy and 3-dimensional scar architecture has an important influence on planning the access route for VT ablation. **Late gadolinium enhancement-CMR** accurately defines epicardial and intramural scar. Among patients with epicardial substrates, success rates are enhanced by epicardial ablation. Identification of the subset of patients who benefit from first-line epicardial ablation using LGE-CMR has been reported to significantly improve outcomes [11]. Secondly, cardiac imaging could be used as preprocedural exclusion of intracardiac thrombus. ***Transthoracic echocardiography (TTE)*** represents a readily available and inexpensive tool for the assessment of cardiac structure, valve function, the presence of mobile left ventricular thrombus (Fig. 13.3). In recent years, CMR and multidetector cardiac computed tomography (MDCT) have emerged as superior imaging modalities for detailed assessment of left ventricular thrombi. Thirdly, the most important role of cardiac imaging is used as guidance of VT ablation [12–14]. Integrating information on the structural VT substrate, as defined by noninvasive imaging, and the electrophysiological substrate, as defined by invasive electroanatomic mapping (EAM), has the potential to enhance safety and efficacy of ablation procedures (Fig. 13.4a, b). For example, coronary arteries could be localized by introducing MDCT/LGE-CMR data into the navigation system and their fusion with the EAM [15].

Cardiac Imaging Merging with 3-D Mapping Systems Process

EAM was performed during sinus rhythm using CartoV3 (Biosense-Webster, Diamond Bar, CA) or NavX (Ensite NavX, St Jude Medical, St. Paul,

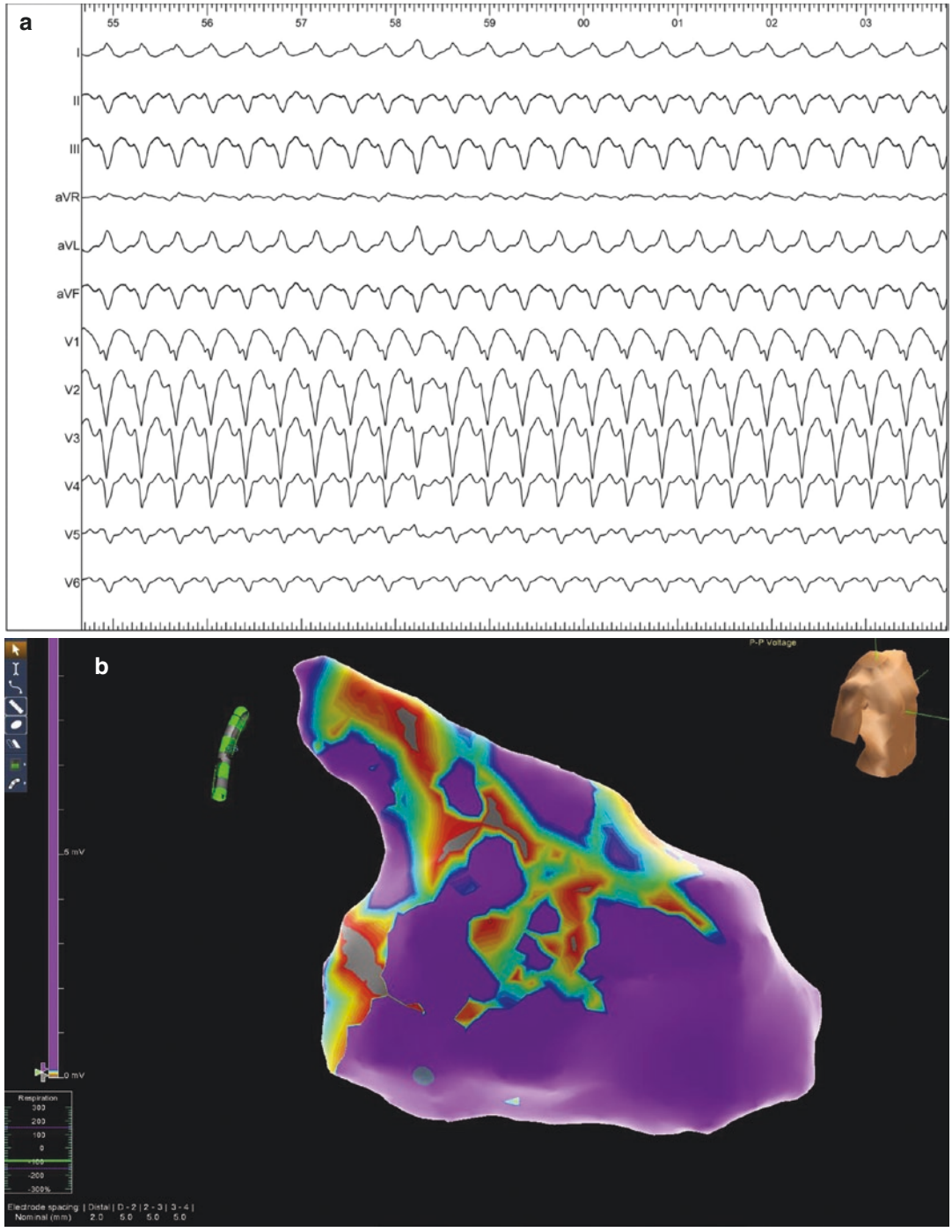
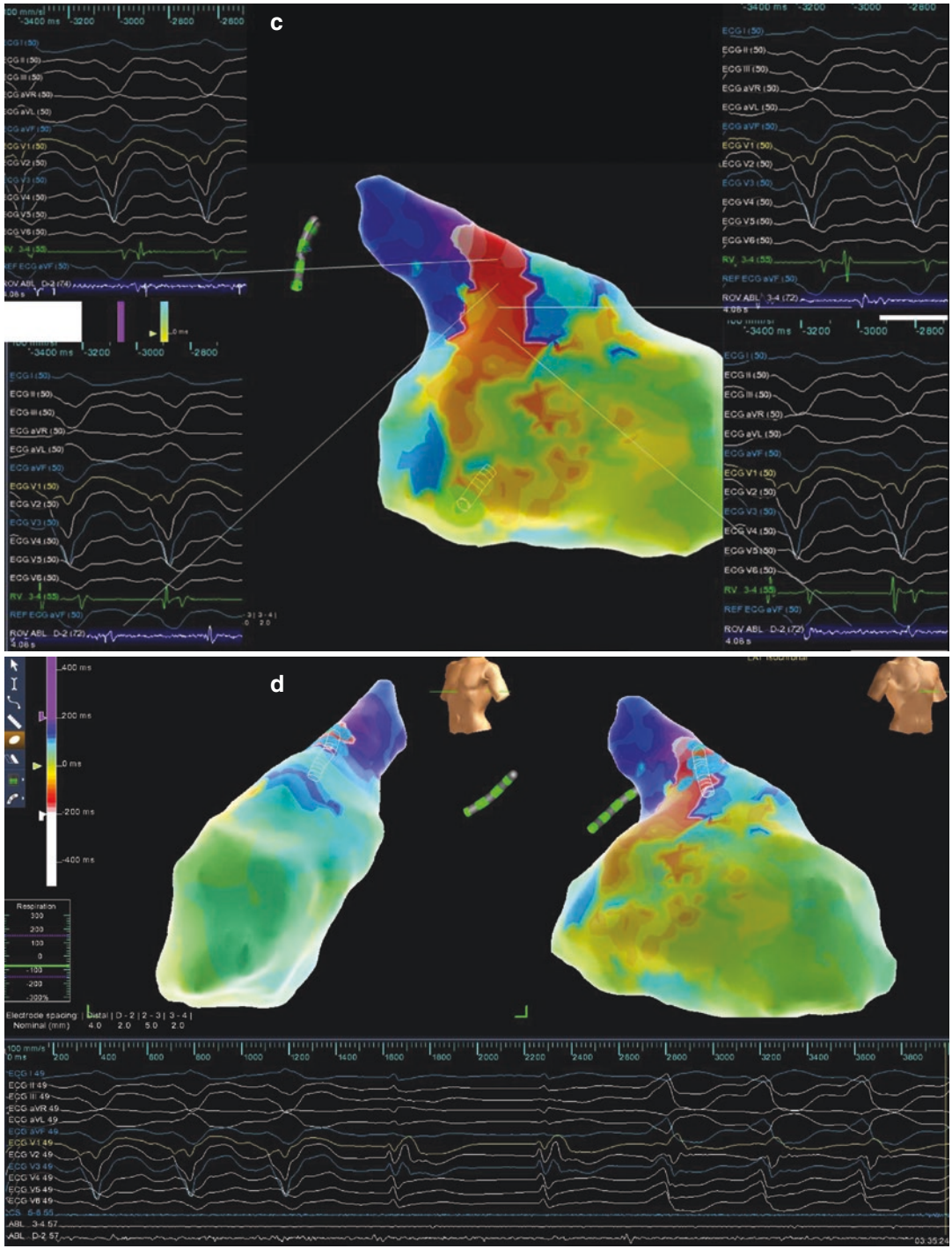


Fig. 13.2 (**a**) Ventricular tachycardia in patient with repaired tetralogy of Fallot. (**b**) Voltage mapping shows scar in the free wall of right ventricular outflow tract. (**c**) Activation mapping shows the central isthmus with mid-diastolic potentials in the free wall of right ventricular outflow tract. (**d**) Ablation terminated the VT at the free wall of right ventricular outflow tract. (**e**) Impulse propagation of VT in RVOT



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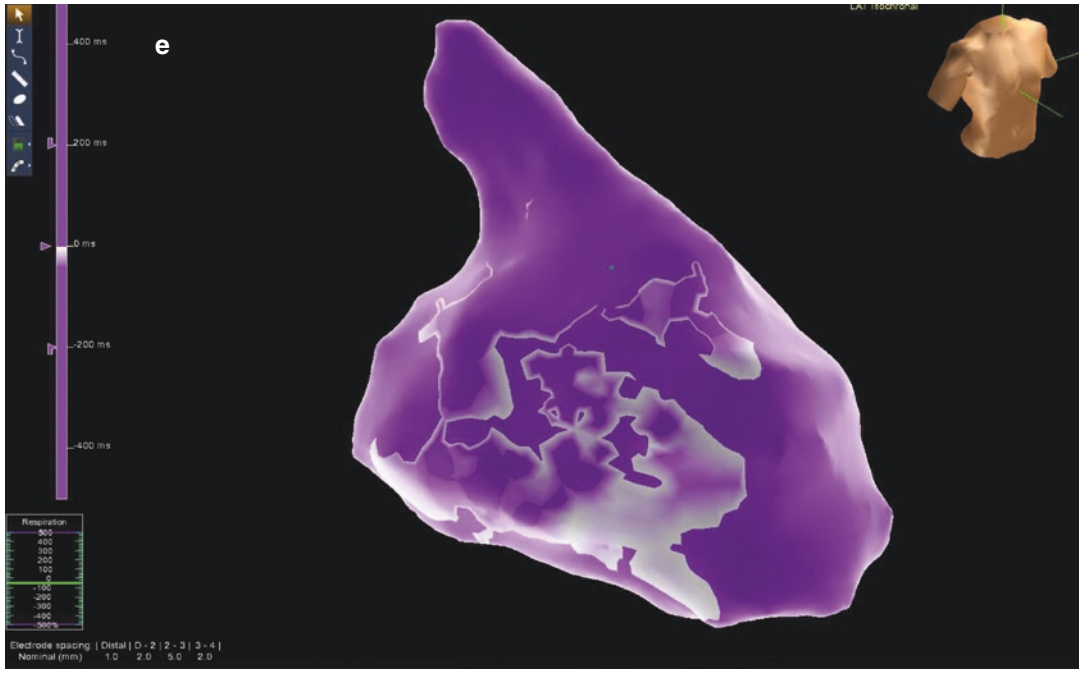

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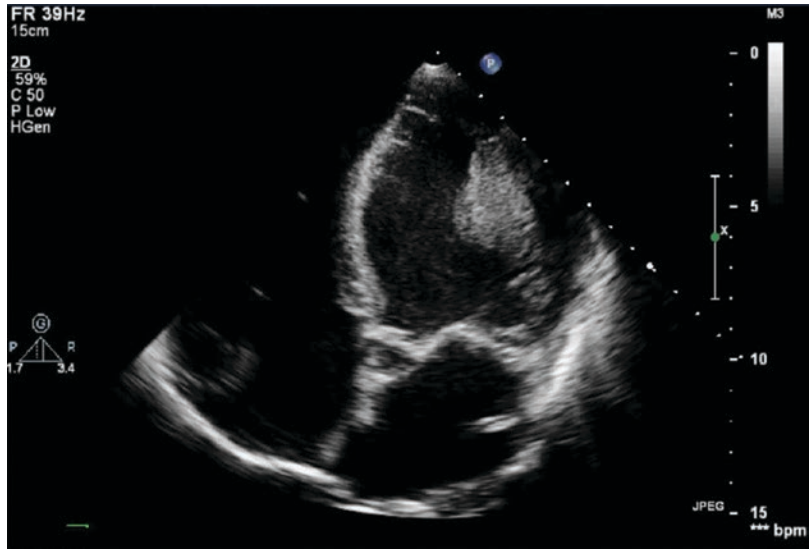

Fig. 13.3 Thrombus formation in left ventricular lateral wall in patient with dilated cardiomyopathy (Thanks Dr. Zhenhui Zhu of Fuwai Hospital who provided the echo image of this patient)

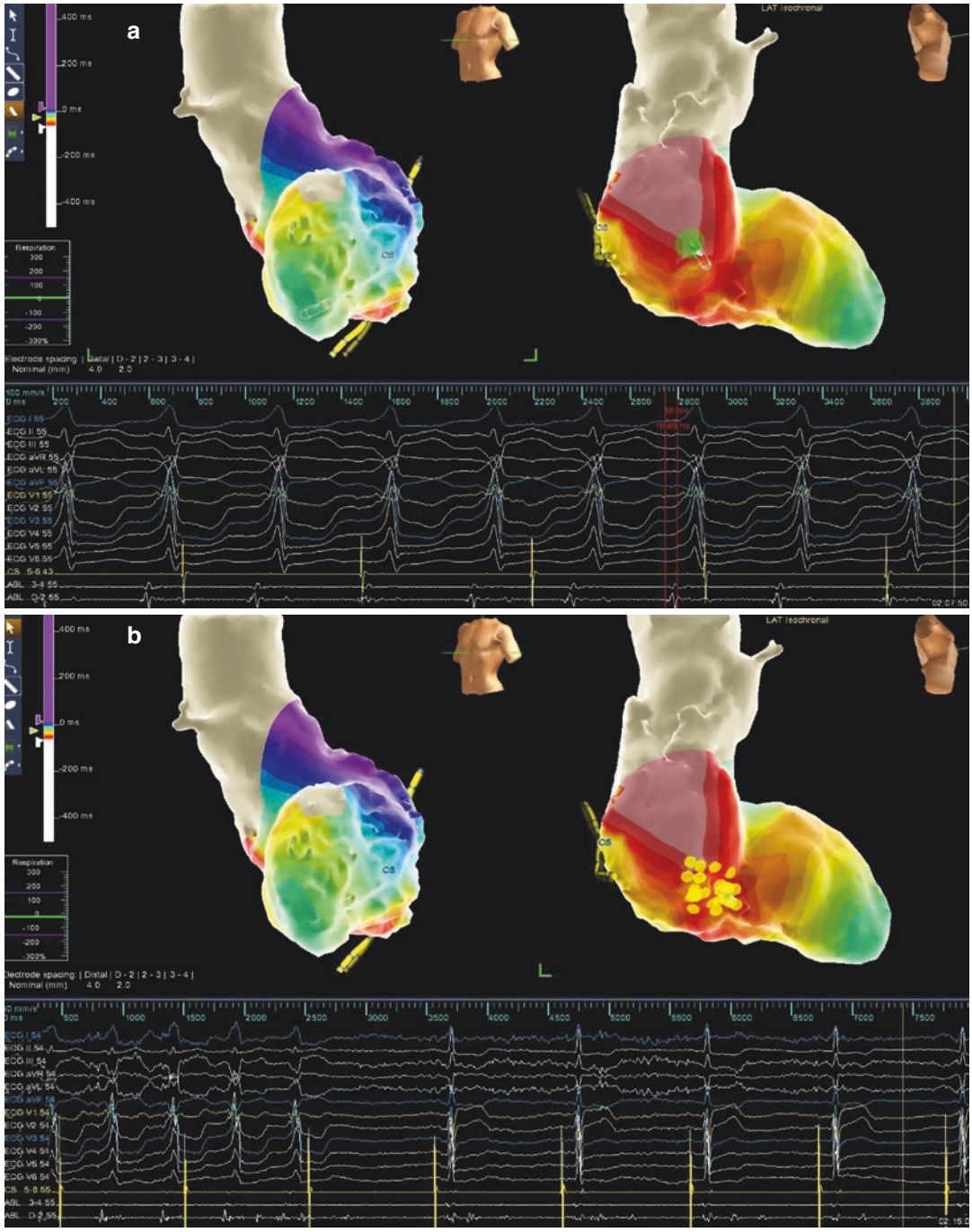

Fig. 13.4 (**a**) Ablation under the guidance of NavX mapping immerging with CT imaging in a 54-year-old male with inferior wall myocardial infarction. (**b**) Ablation at the inferior-septal wall of LV terminated the VT. Note the late potential at the ablation catheter during sinus rhythm. Extensive ablation at the area after termination of VT

MN) system. Mapping was performed with a multipolar high-density mapping catheter (such as PentaRayNav, Biosense Webster; or duodeca polar mapping catheter, St. Paul, MN). A peak-to-peak bipolar amplitude of <1.5 mV is defined as the bipolar low-voltage zone and unipolar amplitude <5.5 mV as the low unipolar voltage. After the acquisition of endocardial and/or epicardial geometries, 3D-EAM geometry is registered with the imported MDCT model. Identifiable anatomic reference points (such as coronary sinus, RV apex, and tricuspid annulus [3, 6, 9, and 12 o'clock] or mitral annulus [3, 6, 9, and 12 o'clock]) is used as landmarks for alignment and orientation of the 3D-EAM and imaging models. For NavX system, to keep the coronary sinus catheter stable is very important, as the body of the coronary sinus catheter is taped to provide a stable spatial reference, and a discreet anatomic boundary for guidance of the 3D-EAM/imaging model registration that could be monitored fluoroscopically throughout the procedure. When using Carto, after initial alignment using these fixed reference points as landmarks for registration, automatic surface registration using CartoMerge (Biosense Webster) was performed. The NavX Fusion registration algorithm allowed dynamic molding of the 3D-EAM geometry to the static MDCT surface (Fig. 13.4a, b). After primary registration, the registered model was refined using a second set of fiducial points, judiciously placed in a stepwise fashion to further align both surfaces at sites of local mismatch [16].

While there are clear data that demonstrate 3-D mapping systems can decrease fluoroscopy use and minimize radiation exposure to patients undergoing ablation. Cardiac imaging merging with 3-D mapping systems process could provide valuable information to recognize the extent and specific distribution of VT substrate (such as local abnormal ventricular activities, LAVA), and guide the manipulation of ablation catheter without fluoroscopy. Integration of MDCT-imaged myocardial fat into 3D-EAM is feasible in patients with ARVC. Its segmentation superimposed on 3D-EAM provides valuable information to recognize the extent and specific distribution of VT substrate and demonstrates ablation targets clustering in its border zone.

Induction and Mapping of Ventricular Tachycardia with Structural Heart Disease

Induction of VT with Structural Heart Disease

In patients with SMVT, reproducible initiation by programmed stimulation is the rule. Reproducible initiation excludes both normal and abnormal automaticity. The common protocol of programmed stimulation includes up to four extra stimuli from two ventricular locations with coupling intervals down to 200 ms or refractoriness, whichever occurred first. The same induction protocol is used to reinduce the VT after catheter ablation.

For patients with ARVC, one study demonstrated that fast rate (≥250 beats/min) right ventricular burst stimulation might provide a useful supplemental method for VT induction in ARVC patients. The author concluded that localized mechanism including micro-reentry and triggered activity might be the main mechanism of VT in ARVC [17].

Mapping of VT with Structural Heart Disease

The mapping technique used to identify potential sites for ablation depends on the mechanism of the VT. Activation mapping and pace mapping are most useful for focal mechanisms, whereas substrate mapping (with or without associated pace mapping) and entrainment mapping are used for reentrant mechanisms. For patients with

SHD, the majority of VTs are not hemodynamically tolerated and therefore do not permit either activation or entrainment mapping. Substrate mapping has evolved over the past decade and a half as an alternative to activation and/or entrainment mapping to deal with hemodynamically untolerated VTs. This methodology reduces or eliminates that need for mapping during prolonged periods of tachycardia.

Substrate mapping is the characterization of areas likely to support reentry based on anatomy and electrophysiological characteristics that can be determined during stable sinus or paced rhythm. Current criteria to define the abnormal substrate rely on a combination of abnormal electrogram characteristics (such as wide, split, and late electrograms) with lower amplitude. Substrate mapping generally begins with identification of the region of ventricular scar, based on electrogram characteristics (usually voltage) in an electroanatomic map of the ventricle of interest. **Scar tissue** can be identified based on bipolar electrogram amplitude. Bipolar electrograms recorded from regions of infarction have a peak-to-peak amplitude <1.5 mV (1 mm interelectrode spacing with a 4-mm distal electrode and filtered at 10–400 Hz) [18]. Areas of extremely low voltage (<0.5 mV or even less) have been designated as ***dense scar***, but it is important to recognize that these regions can still contain viable myocytes and reentry circuit isthmuses. Other study suggested that a bipolar voltage of <0.1 mV could be used to define electrical inexcitability [19]. Changing the bipolar viability voltage range to 0.1–0.5 mV increased the heterogeneity within the area of previously defined "dense scar." Of note, use of different mapping catheters with different electrode sizes and interelectrode distances may have an impact on voltage and abnormal electrogram recording. For instance, catheters with large-tip electrodes (3.5–4.0 mm) often record low-amplitude signals in areas of heterogeneous scar, while catheters with smaller electrodes (0.4–1.0 mm) record high-voltage signals at similar scar sites, thus identifying surviving myocardial bundles. Similarly, decreasing the interelectrode distance may decrease the voltage amplitude and duration, but may better identify near-field obscure activity, increasing the resolution of mapping.

In patients with VT, these infarct regions are typically large, with a circumference exceeding 20 cm [20]. In order to limit the area of ablation within the scar, a region of the scar is targeted for ablation based on pace mapping or additional electrogram characteristics. ***Exits*** can be identified based on pace mapping along the border of the scar. Radiofrequency lesions can then be placed in the infarct border zone (where bipolar electrogram amplitude is typically between 0.5 and 1.0 mV) roughly parallel to the infarct border. ***Late potentials*** during sinus rhythm and **isolated potentials** during VT are usually present within these low-amplitude regions and are often produced by conduction through potential conduction channels in the infarct areas [21, 22]. A number of ablation approaches have been described with the aim of targeting the abnormal substrate defined with mapping in sinus or paced rhythm. Some of these strategies (such as late potential and local abnormal ventricular activity ablation or scar homogenization) target the entire abnormal substrate harboring abnormal electrograms, defined with a variety of different criteria [23]. Scar dechanneling, linear ablation through sites matching VT with pacing, and the core isolation approach focus on more discrete regions with in the abnormal substrate that have been proven relevant to the clinical and/or inducible arrhythmias by means of physiologic maneuvers.

Some randomized trials examined the effect of substrate-based ablation on the long-term success rate of VT ablation. The VISTA trial demonstrated that an extensive substrate-based approach was superior to a more focused activation mapping strategy [24]. The randomized, primary-prevention trial, SMASH VT, showed a reduction in VT events using a substrate-based approach [25]. However, the positive results of this trial, which achieved a 70% reduction in arrhythmic events at 2 years, have not been replicated.

Ablation of Common Ventricular Tachycardia with Structural Heart Disease

Ventricular Tachycardia in Patients with Ischemic Heart Disease

Ventricular tachycardia occurs in 1–2% of patients late after myocardial infarction (MI), often after an interval of several years. The mechanism of VT is usually macroreentry [26]; focal non-reentrant mechanisms are responsible for fewer than 5–10% of VTs [27]. Patients with scar-related VTs often have multiple potential reentry circuits, giving rise to more than one morphology of inducible monomorphic VT. VTs may be hemodynamically well tolerated and inducible, but the majority of patients have one or more VTs that are unstable and not amenable to extensive mapping, due to poor hemodynamic tolerance, or inconsistent inducibility. Substrate mapping approaches that identify target regions for ablation during stable sinus rhythm often allow ablation of these tachycardias.

Several large multicenter series have reported outcomes following combined entrainment and substrate mapping approaches (Table 13.1). The **SMASH-VT** (Substrate Mapping and Ablation in Sinus Rhythm to Halt Ventricular Tachycardia) trial compared ICD implantation along with VT substrate ablation to ICD implantation alone in patients with recent VT [25]. In this trial, ablation of the VT substrate reduced ICD shocks from 31 to 9% over a mean follow-up of 22.5 ± 5 months (p = 0.003) and reduced VT from 33 to 12% (p = 0.007) in comparison to a control group that did not use antiarrhythmic drug therapy. The **VTACH** (Ventricular Tachycardia Ablation in Coronary Heart Disease) **trial** [28] studied the effect of catheter ablation in patients with ischemic cardiomyopathy, who experienced hemodynamically tolerated SMVT without specific antiarrhythmic drug therapy in the control arm (approximately one-third of patients in both arms were treated with amiodarone). Over a mean follow-up of 22.5 months, ablation prolonged the time to recurrent VT significantly (HR = 0.61), with a relative risk reduction of 25% and an absolute risk reduction of 18% at 2 years. In the **SMS** (Substrate Modification Study) [29], catheter ablation failed to decrease the primary endpoint of time to first VT/VF recurrence in post-MI patients with LVEF ≤40% and hemodynamically unstable VT. However, ablation did result in a >50% reduction in total ICD interventions. The **VANISH** (Ventricular Tachycardia Ablation versus Escalated Antiarrhythmic Drug Therapy in Ischemic Heart Disease) trial randomly allocated patients with ICD, prior infarction, and SMVT despite first-line antiarrhythmic drug therapy to catheter ablation or more aggressive antiarrhythmic drug therapy [30]. During 28 months of follow-up, catheter ablation resulted in a 28% relative risk reduction in the composite endpoint of death, VT storm, and appropriate ICD shock (p = 0.04).

Ventricular Tachycardia in Patients with Dilated Cardiomyopathy

The ventricular myocardium of individuals with dilated cardiomyopathy (DCM) is histologically characterized by multiple regions of patchy interstitial and replacement fibrosis, myofiber disarray, and variable degrees of myocyte hypertrophy and atrophy. Sustained monomorphic ventricular tachycardia is not common in nonischemic dilated cardiomyopathies, but 80% of those that occur are due to scar-related reentry, with the remainder due to bundle branch reentry or a focal origin. Unlike post-MI scar that has a predilection for the subendocardium, scar in DCM may be mid-myocardial or epicardial and most often occurs in the basal anteroseptal and inferolateral LV regions [11, 31, 32]. The results of catheter ablation in dilated cardiomyopathy are generally worse than in ischemic cardiomyopathy, due to a higher preponderance of intramural substrate and infrequent substrate ablation targets (i.e., fractionated and late potentials) [33–35].

Table 13.1 Clinical trials of catheter ablation in patients with ischemic heart disease

Trial	Control therapy	No. of patients	Population	Mean follow-up (Mo)	Endpoint	Outcomes (%)		
						Ablation	ICD	*P*
SMASH-VT	ICD	128	OMI underwent ICD for VT or VF	22.5 ± 5.5	Appropriate ICD therapy	12	33	0.007
VTACH	ICD	107	OMI, LVEF ≤50%, stable VT	22.5	Survival free from VT or VF	47	29	0.045
SMS	ICD	111	Post-MI patients with LVEF ≤40%	27.6 ± 13.2	Event- free survival	49	52.4	0.84
VANISH	Escalated antiarrhythmic drug therapy	259	Prior infarction, ICD and SMVT	27.9 ± 17.1	Composite endpoint of death, VT storm, and appropriate ICD shock	59.1	68.5	0.04

SMASH-VT substrate mapping and ablation in sinus rhythm to halt ventricular tachycardia, *VTACH* ventricular tachycardia ablation in coronary heart disease, *SMS* substrate modification study, *VANISH* ventricular tachycardia ablation versus escalated antiarrhythmic drug therapy in ischemic heart disease, *Mo* month, *ICD* implantable cardioverter defibrillator, *MI* myocardial infarction, *LVEF* left ventricular ejection fraction, *SMVT* sustained monomorphic ventricular tachycardia, *VT* ventricular tachycardia, *VF* ventricular fibrillation

Ventricular Tachycardia in Patients with Arrhythmogenic Right Ventricular Cardiomyopathy/ Dysplasia

Arrhythmogenic right ventricular dysplasia/ cardiomyopathy (ARVC/D) is characterized by fibrofatty replacement of myocardium within the so-called triangle of dysplasia which encompasses the RV inflow, outflow, and apex. Scar-related reentry is the most common cause of VTs, focal VTs in ARVD/C have also been described, although some of these cases may represent epicardial reentry with a focal endocardial breakthrough [36].

Conventional VT induction protocols including extra-stimuli and incremental stimulation had been found to increase the yield of inducible VT; some study also found that fast rate (≥250 beats/min) right ventricular burst stimulation provides a useful supplemental method for VT induction in ARVC patients, which might indicate that localized mechanism including micro-reentry and triggered activity might be one of the mechanisms of VT in ARVC [17]. The methods for mapping and ablation are as for scar-related VTs; activation mapping, entrainment mapping, substrate mapping, and combined approaches have been used. Our previous study demonstrated the efficacy of endocardial regional RF catheter ablation under the guidance of noncontact mapping (Fig. 13.5) [37]. Endocardial VT ablation in this setting can produce acute success,

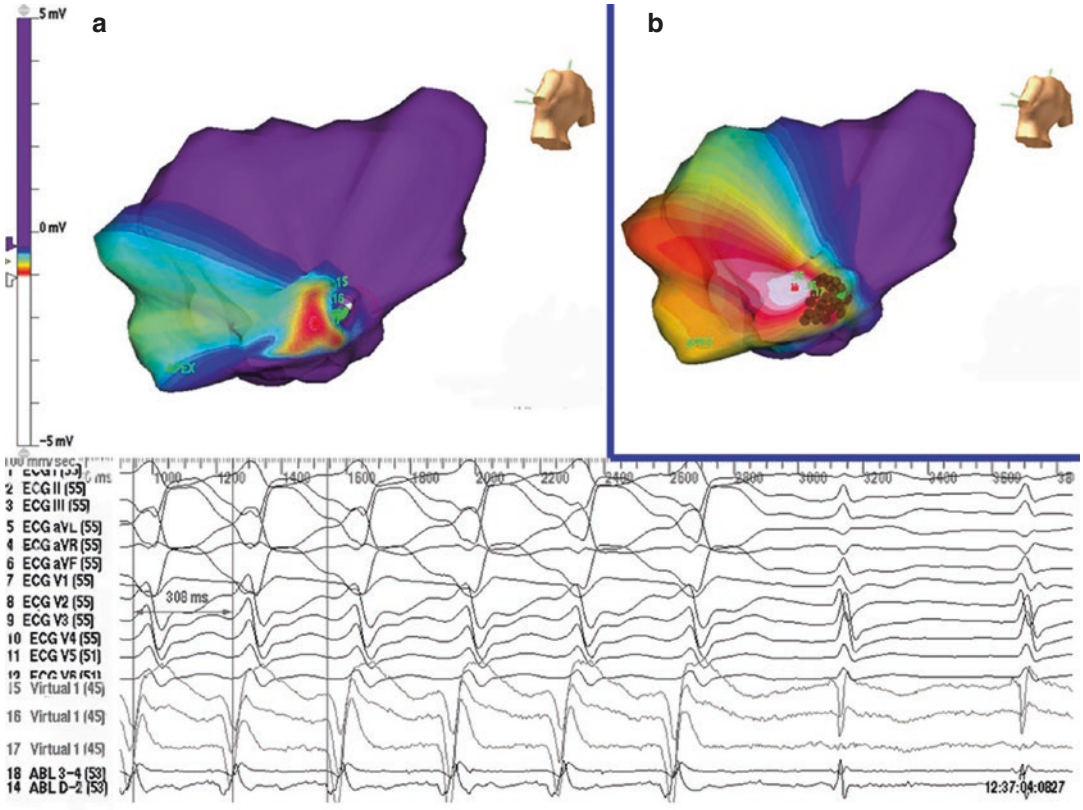

Fig. 13.5 Regional ablation under the guidance of isopotential mapping in a 36-year-old female with a 4-year history of palpitation and pre-syncope. The VT origin was at the basal septum. Panel **a** indicates that the VT with cycle length of 308 ms was terminated by RF ablation but was induced again. The ablation region was expanded stepwise according to the mapping results. Panel **b** indicates that VT was still inducible after 23 RF applications. The morphology of the VT is similar and was finally terminated after further attempts at adjacent sites. *HIS* His bundle, *Apex* apex of the right ventricle

though recurrence rate is quite high, which may be explained by the more epicardial and patchy nature of the disease. Combined endocardial-epicardial ablation has since been shown to be feasible, safe, and with significantly better acute and long-term success, particularly when combined with scar dechanneling or homogenization of the scar (Fig. 13.6a–c) [38].

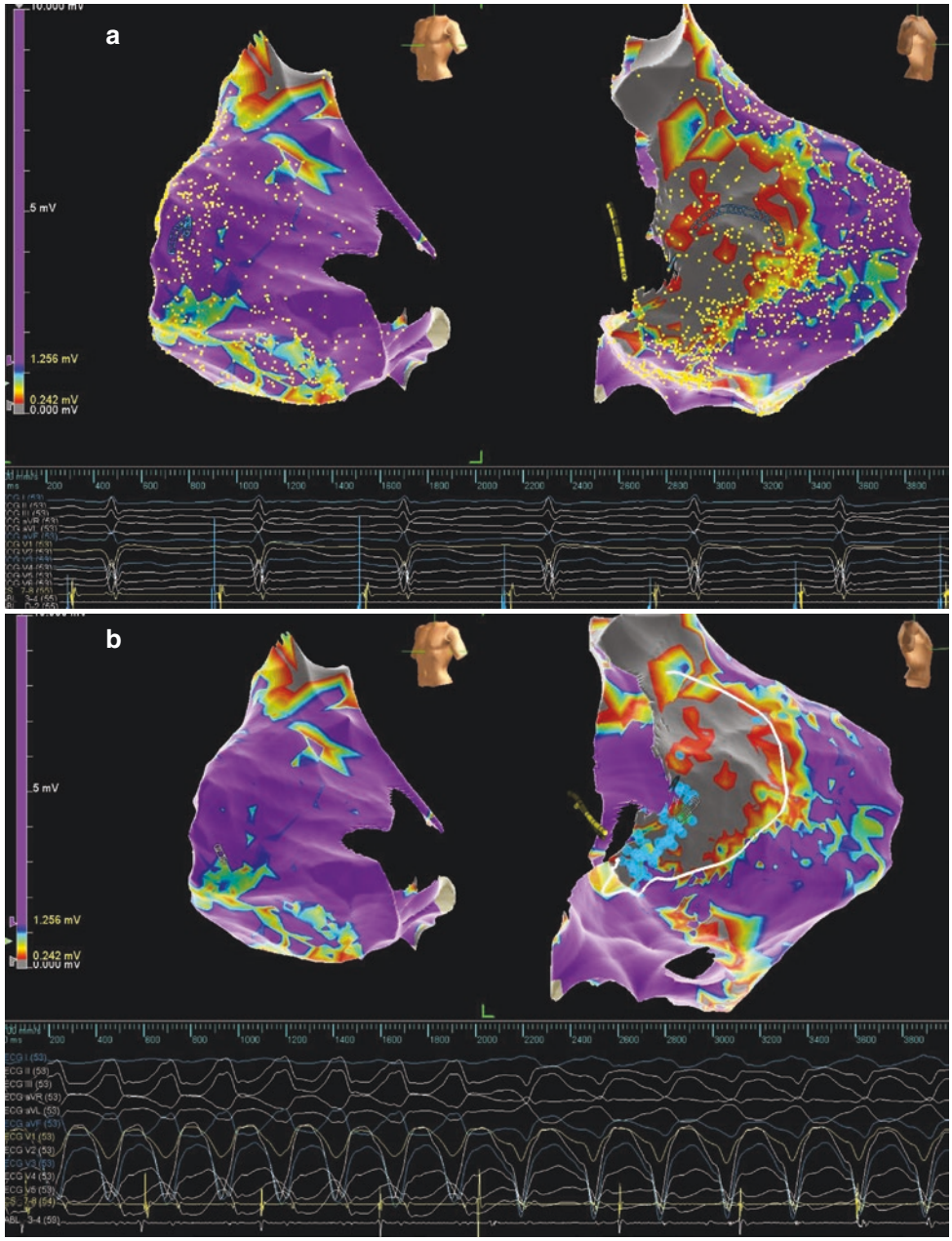

Fig. 13.6 (**a**) Epicardial mapping and ablation in patients with ARVC. Substrate mapping shows huge low-voltage area and scar at RVOT and basal-lateral wall of RV. (**b**) The morphology of VT changed after ablation at the basal-lateral wall of RV. (**c**) Extensive and regional ablation at the Basal-lateral wall of RV terminated the VT finally

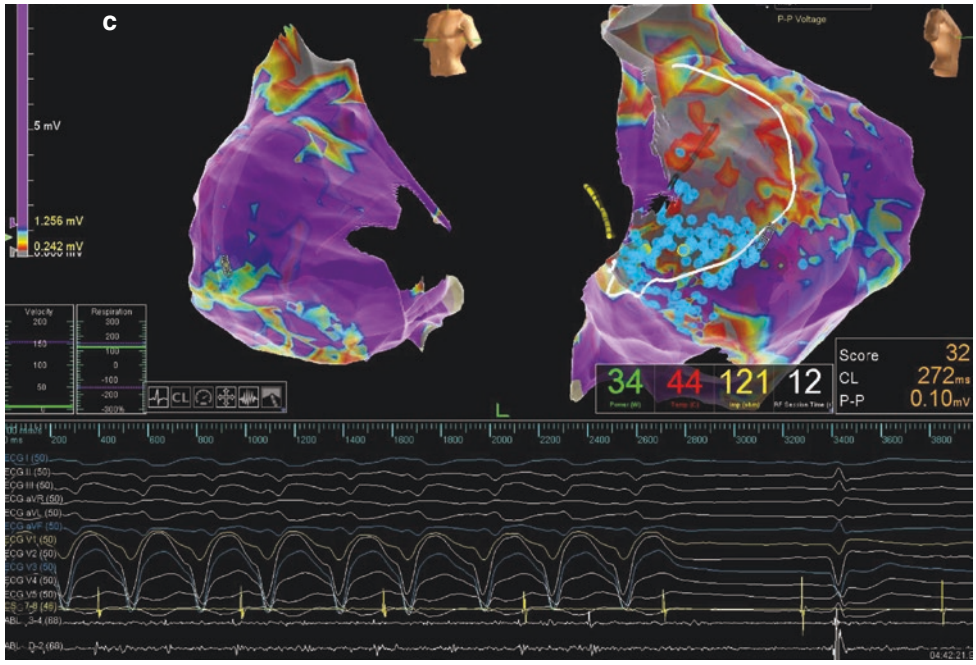

Fig. 13.6 (continued)

Ventricular Tachycardia in Patients with Hypertrophic Cardiomyopathy

In high-risk individuals with hypertrophic cardiomyopathy (HCM), monomorphic VT accounts for 38% of episodes [39]. HCM patients with LV aneurysms are at higher risk for the occurrence of monomorphic VT. Ventricular arrhythmias occur more frequently (4.7% vs. 0.9%/year; $p < 0.001$) in HCM patients with and without LV aneurysms [40]. The mechanism of monomorphic VT in these patients is also scar-related reentry and is amenable to catheter ablation [41]. Standard endocardial mapping and ablation alone are likely insufficient to target the relevant VT circuits. In this highly selected population, a combined epicardial and endocardial approach to catheter ablation is feasible and of reasonable efficacy to consider if aggressive trials of antiarrhythmic medications and trials of antitachycardia pacing fail to control VT [42].

Ventricular tachycardia could also occur in other diseases including valvular heart disease, sarcoidosis, Chagas disease, and in repaired congenital heart disease (Fig. 13.1). In patients with sarcoidosis, due to progressive inflammation and a complex underlying substrate, ventricular arrhythmias may have several mechanisms, including abnormal automaticity, triggered activity, or scar-related reentry [43]. When inflammation is present, the combination of immunosuppressive therapy and antiarrhythmic drugs may decrease VT recurrence. However, when these treatments fail, catheter ablation is feasible [44]. Chagas disease is caused by the flagellate protozoan parasite Trypanosoma cruzi and is transmitted by hematophagous triatomine insect vectors. It is endemic to Latin American countries, but is also prevalent in non-endemic areas due to population migration. Monomorphic VT due to scar-related reentry can arise from any scar location, but the inferolateral segment is the most common source, accounting for ~80% of VTs [45]. Combination

of endocardial and epicardial mapping and ablation in Chagas patients markedly improved the long-term success rate [46].

Minimization of Fluoroscopy During Mapping and Ablation of VAs

Traditionally, mapping and catheter ablation of VAs in patients with structural heart disease is complex and guided by three-dimensional mapping system and fluoroscopy. Actually, it is quite difficult to perform the process with zero fluoroscopy, but minimization of the fluoroscopy is reasonable with the accumulation of more experiences.

The Placement of Diagnostic and Ablation Catheter

We can position the diagnostic (such as the His bundle, right ventricular, and coronary sinus catheter), mapping, and ablation catheter into the exact sites guided by the 3D mapping system with zero fluoroscopy (Fig. 13.7a, b). The trajectory of the catheter could be clearly monitored and observed under the guidance of the 3D mapping system. In addition, operators could continuously observe two projections at the same time through 3D mapping system—such as the position of CS catheter, firstly, the CS catheter is bent at 90° and direct to the lateral wall of right atrium in the left anterior oblique (LAO) 30° view, then rotate it clockwise and direct to the inferior septal wall of right atrium; it is generally easy to enter the ostium of coronary sinus. For the manipulation of retroaortic approach, we can bent the catheter as inverted U shape at ascend aorta, the tip of catheter direct to the anterior aorta, then push it and pass through the aortic valve in the right anterior oblique (RAO) 30° view. All the manipulation of catheter could be performed under the guidance of 3D mapping system without fluoroscopy.

Of note, retract the catheter and advance it again when we feel resistance during the procedure of placing catheter. However, it is necessary to replace the catheter under the X-ray guidance for the safety consideration, if persistent resistance encountered in the process of pushing the catheter, which may indicate the stenosis, distortion, or occlusion of vascular access. In this circumstance, angiography could be used to evaluate the vascular condition. Schwartz sheath can be applied by pushing long wire across narrow or twisted vessels if encounting the vascular stenosis or distortion. Otherwise, we need to choose other vascular accesses, such as the internal jugular vein, subclavian vein, contralateral femoral vein, or artery.

The Substrate Mapping and Ablation During Sinus Rhythm

The main challenges for catheter ablation of ventricular tachycardia are the complex arrhythmogenicity of the myocardial scar and hemodynamically unstable state during onset of VT. Substrate mapping in sinus rhythm could allow hemodynamically unstable VAs to be successfully mapping and ablation, and consequently minimizing the fluoroscopic dose [47]. This approach uses a non-fluoroscopic three-dimensional mapping system that produces an electroanatomical reconstruction of the heart.

The substrate mapping of RV/LV endocardium and epicardium could be performed using ablation or high-density mapping catheter (typically Pentaray® catheter, Biosense Webster, Diamond Bar, CA, USA). High-density EAM of RV/LV was obtained during stable sinus rhythm using the CARTO system (Biosense Webster, Diamond Bar, CA) or Ensite Navx (St Jude Medical, St Paul, MN). Endocardial access to the LV was obtained by single transseptal puncture or retroaortic approach. Transseptal LV access was achieved, and manipulation of the ablation catheter was assisted by a steerable sheath (Agilis St Jude Medical, St Paul, MN). Epicardial mapping and ablation are performed when preprocedural ce-CMR showed epicardial scar, endocardial mapping do not identify subendocardial scars, ECG of clinical or induced VT suggested epicardial origin, or endocardial ablation is unsuccessful.

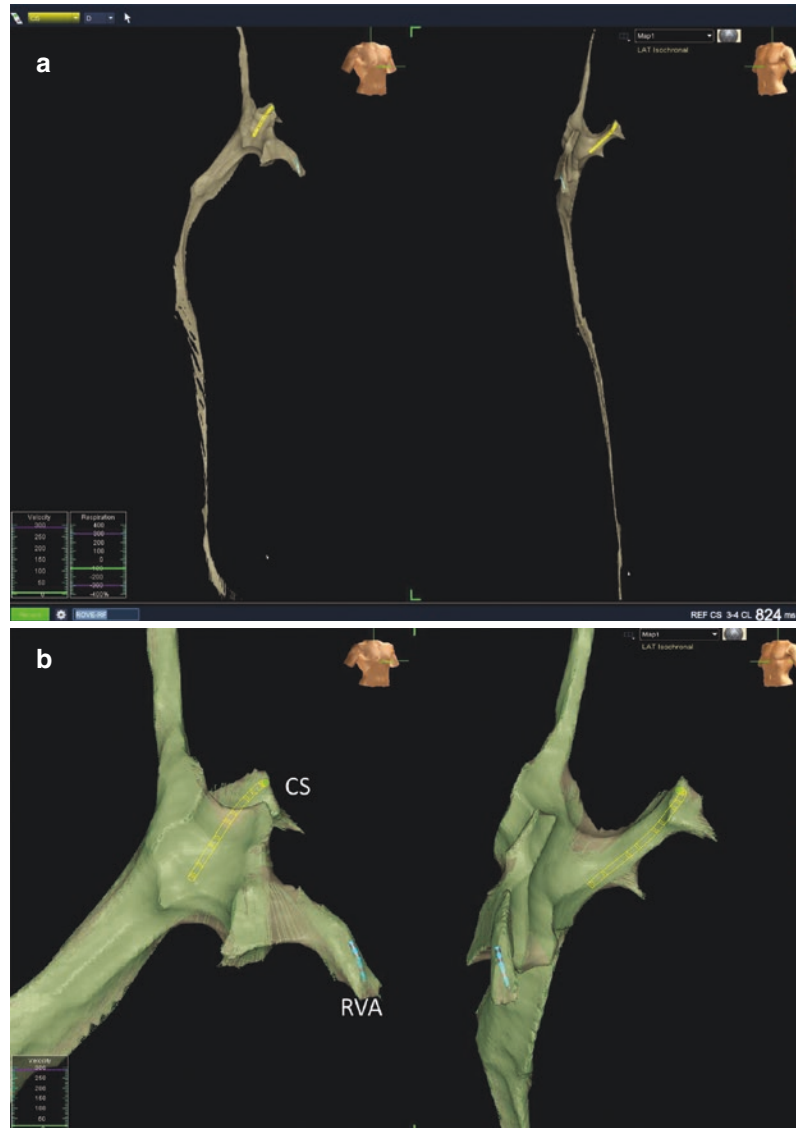

Fig. 13.7 (**a**) The trajectory of the catheter could be clearly monitored and observed under the guidance of the 3D mapping system. (**b**) The position of CS and RVA catheter under the guidance of Ensite system

All the mapping procedure could be performed under the guidance of EAM system during sinus rhythm, which minimizes the radiation dose. With regard to our previous procedural data with guidance of fluroscopy, we found that our total procedural times are comparable to previous data despite longer geometry times. We believe this highlights the importance of accurate geometry reconstruction, as it suggests that spending more time obtaining an accurate geometry reconstruction with voltage and abnormal electrograms may speed up subsequent phases of the procedure. Indeed, basing ablation on an accurate 3D model of cardiac anatomy provides certain remarkable technical advantages, which both save time and make the procedure easier. Electroanatomical mapping confers a deeper insight into the patient's individual cardiac anatomy, as geometry reconstruction will already have compelled the operator to exercise the movements necessary to reach the various intracardiac sites relevant to the subsequent ablation procedure. The latter is also speed up by the fact that the 3D model enables the operator to continuously observe two projections at the same time.

The Scar Homogenization and/or Dechanneling During Sinus Rhythm

The scar homogenization approach targets all the abnormal electrograms within the scar defined with conventional bipolar voltage criteria when mapping in sinus or paced rhythm (Fig. 13.8a–c). The acute ablation endpoint is either elimination of abnormal electrograms or loss of local capture at high-output pacing (20-mA output at 10 ms pulse width). One large multicenter randomized study comparing scar homogenization with stan-

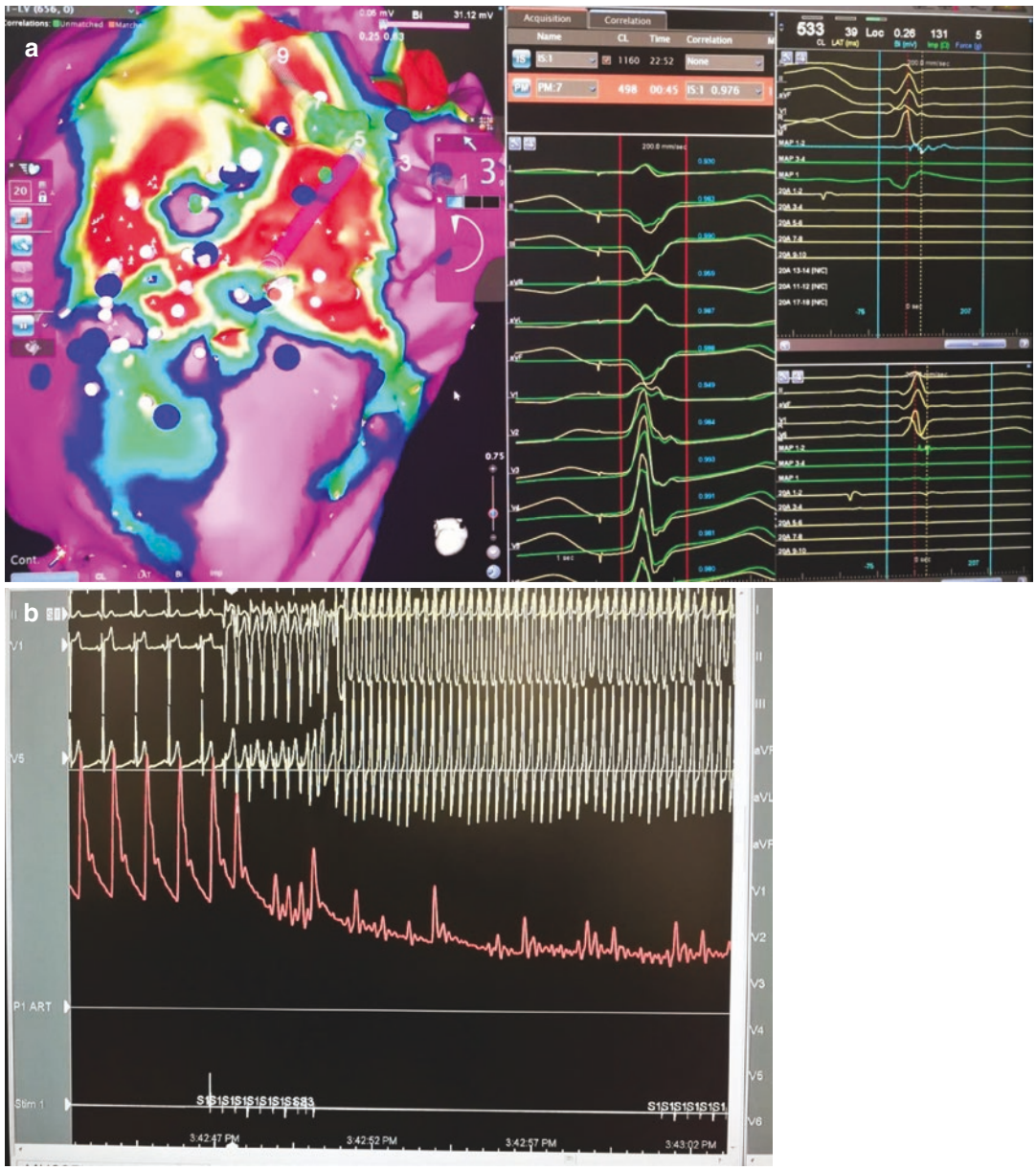

Fig. 13.8 (**a**) One VT patient with OMI (inferior wall). Late potential was recorded in the border zone of inferior wall during sinus rhythm, pace mapping at this site produce morphology was similar to the VT morphology. (**b**) Non-tolerable VT was induced using programmed stimulation. (**c**) The scar homogenization approach targets all the abnormal electrograms within the scar was performed (Thanks Dr. Ivan Ho of Cedars-Sinai Medical Center who performed the radiofrequency ablation for this patient)

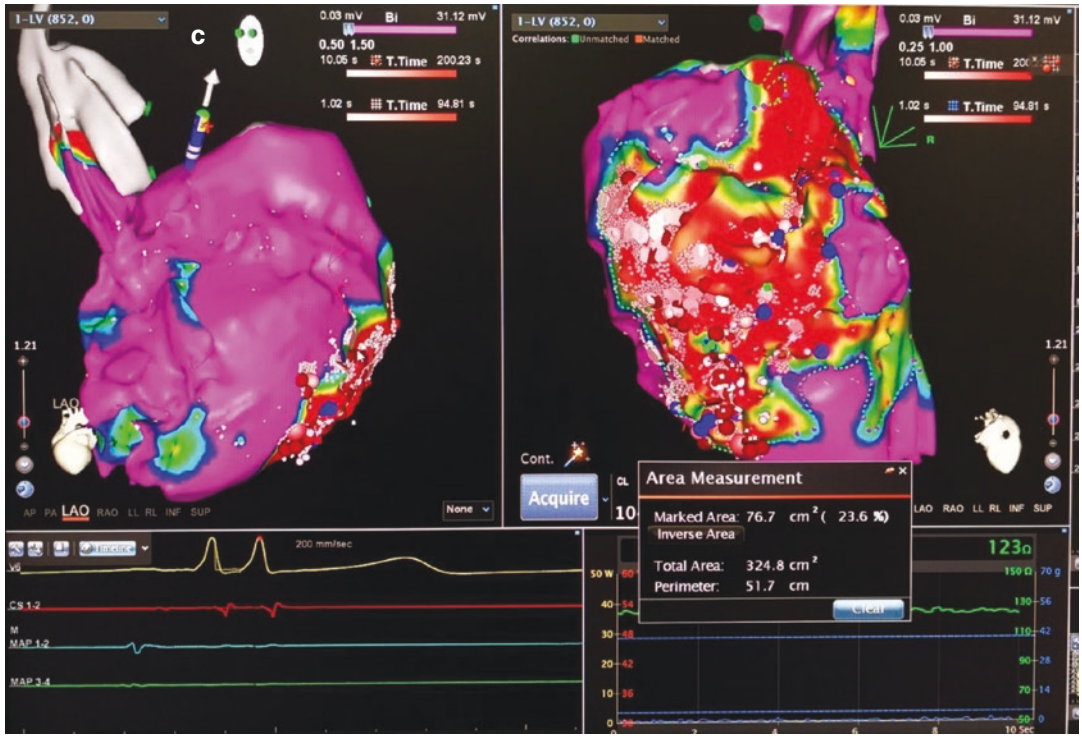

Fig. 13.8 (continued)

dard limited substrate ablation in 118 patients with ischemic cardiomyopathy presenting with stable VT demonstrated the efficacy of scar homogenization strategy. At 1 year post ablation, freedom from VT recurrence was achieved in 52% of patients who underwent clinical VT ablation only vs. 85% of patients who underwent scar homogenization.

The substrate ablation approach that targets channels within the abnormal substrate was described as scar dechanneling. After identified the conduction channel (CC), radiofrequency (RF) elimination of all identified CCs by RF ablation is performed at the CC entrance during sinus rhythm. In our center, substrate mapping routinely be performed as first step before VT induction and ablation in patients with hemodynamically stable or unstable VT. The acute and long-term outcomes of ablation are not inferior to the strategy of VT induction and mapping before substrate mapping. Our results are consistent with the findings reported by other centers [9, 47, 48]. As the accumulation of experience of VT ablation, the manipulation of mapping and/or ablation catheters could be done guided not by fluoroscopy but under three-dimensional mapping system. Therefore, minimization of radiation dose could be achieved during the whole procedure of mapping and ablation.

Conclusions

In general, catheter ablation of ventricular tachycardia in patients with structural heart disease can significantly reduce the incidence of recurrent ventricular arrhythmias. It is best used as an adjunct to ICDs, and as an alternative or adjunct to antiarrhythmic drugs. Nowadays, 3D mapping system provides an accurate geometry reconstruction with voltage information and abnormal electrograms, which is used to guide the subsequent mapping and ablation procedure without fluoroscopy. Technological advancements in substrate imaging, mapping, and ablation to improve outcomes and minimization of radiation dose are in the process of development.

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