Chapter 7
Ventricular Tachyarrhythmias

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Ventricular tachyarrhythmias often are associated with different symptoms and variable prognoses, and therapy needs to be tailored accordingly. A clear understanding of the underlying substrate and how these ventricular tachyarrhythmias develop is critical in the proper treatment of the patients with these conditions.

How Do Arrhythmias Start?

Electrical impulse normally travels through the heart chambers in a very organized and uniform manner. However, disturbances in how electrical signals are initiated or how they propagate through the cardiac chamber can lead to the onset of arrhythmias. In general, there are three mechanisms of how arrhythmias start. A common arrhythmogenesis is impulse

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D.T. Huang, T. Prinzi (eds.), *Clinical Cardiac Electrophysiology in Clinical Practice*, In Clinical Practice,
DOI 10.1007/978-1-4471-5433-4_7,

reentry. Tissues may intrinsically exhibit multiple pathways (as in dual atrioventricular nodal physiology, see "Chap. 3 SVT") or develop multiple pathways in the healing process (as in scars post myocardial infarction) through which the electrical signal may travel. If these paths are associated with different conduction velocities and correspondingly different refractory periods, the electrical signals can circle around in an "endless loop" through these circuit paths. Conditions suitable for reentry require the slower conducting pathway to have a shorter refractory period and the faster conducting pathway to have a longer refractory period. Most VTs, though certainly not all, related to post myocardial infarction substrate or cardiomyopathy are due to impulse reentry. Monomorphic VT is often due to stable and fixed circuits of reentry whereas polymorphic VT can be due to unstable and meandering or even multiple circuits of reentry. Another mechanism for arrhythmia onset is due to triggered activity. Increased intracellular calcium concentration due to heightened adrenergic stimulation (such as exercise) initiates a cascade of reaction through activation of stimulatory G proteins, resulting in enhanced calcium entry through the cellular calcium channels and calcium induced calcium release in the sarcoplasmic reticulum. This increase in the intracellular calcium may then activate the sodium calcium exchanger leading to abnormal sodium entry into the cell which may trigger depolarization of the heart cell, resulting in premature beats or even tachycardia [1]. Forms of normal heart idiopathic ventricular tachycardia associated with exercise, such as one originating right ventricular outflow tract, are often resulting from triggered activity. A third mechanism for arrhythmogenesis is enhanced automaticity, where cardiac muscle tissue develops spontaneous electrical activity through abnormal depolarization during phase 4 of the action potential. These arrhythmias are usually referred as "automatic" tachycardia. Some examples of these include variants of tachycardia related to diseased tissue where the baseline membrane potential may be unstable. Typically, sources of tachycardia that are focal are due to either triggered activity or enhanced automaticity.

The management of ventricular arrhythmias is thus complex and quite variable, with this variance discussed in the following segments. Our initial focus will be scar mediated VT (VT with an abnormal left ventricular ejection fraction [LVEF]) followed by idiopathic PVC's and less frequent forms of idiopathic ventricular tachycardia (VT with a normal LVEF).

Electrocardiographic Evaluation of Ventricular Arrhythmias

The first and most important diagnostic tool remains the 12 lead electrocardiogram during a wide QRS complex tachycardia. Often the type of cardiomyopathy, location of scarring, and VT exit can be estimated from the appearance of the tachycardia. Multiple algorithms have been developed and studied for the electrocardiographic diagnosis of VT including the Brugada criteria [2] and various individual lead (Lead II, AVR) [3, 4] analysis techniques, all with good specificity and sensitivity for the identification of VT in distinction from supraventricular tachyarrhythmias (SVT) with aberrancy. All these algorithms take advantage of the initial forces of activation to distinguish VT from SVT. Tachycardia of ventricular origin depolarizes the myocardial muscles by cell-to-cell contact and thus will have slower forces of activation represented by delayed or fragmented portions early in the QRS signals. On the other hand, during SVT, even with aberrancy, the heart muscles are activated via engaging the specialized conduction tissues and are generally associated with a more smooth and rapid initial QRS signals. Of note, all of these algorithms are qualified and should be used with caution in patients with manifest preexcitation (i.e., Wolff-Parkinson-White syndrome) or on antiarrhythmic medical therapy. Updated morphology criteria developed in recent years have resulted in more elegant and simplified algorithm to decipher whether a wide complex tachycardia may be VT or SVT with aberrancy. Inspecting the morphology of the initial QRS signals in lead aVR can be used to suggest ventricular origin of a wide complex tachycardia. As illustrated in

Fig. 7.1, QRS with slow or notched initial forces as well as those with unusual axis all suggest a diagnosis of VT rather than SVT. Similarly, if the duration of the QRS signal from the beginning of the onset to the peak of the R wave in lead II measures to be greater or equal to 50 ms, then VT can be diagnosed with better than 95 % confidence (Figs. 7.1, 7.2, and 7.3).

Once diagnosis is made that the arrhythmia is VT, the next step is to determine the activation vector of the ventricle. As a simplistic starting point, the bundle branch block appearance of the QRS complex indicates the culprit chamber where the VT is originating from. A left bundle branch block appearance indicates an RV or septal LV VT while a right bundle branch appearance indicates an LV VT origin. Taking this approach a step further, using the right sided leads (V1, AVR), inferior leads (II/III/AVF), lateral leads (1/AVL) and apical leads (V5/V6) one can identify where the VT is coming from

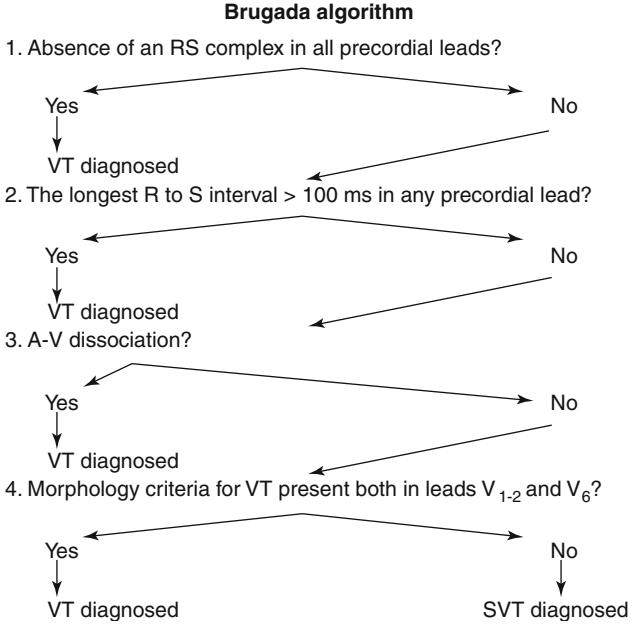

FIGURE 7.1 Algorithm to determine VT vs. SVT by Brugada et al. [2] (Used with permission)

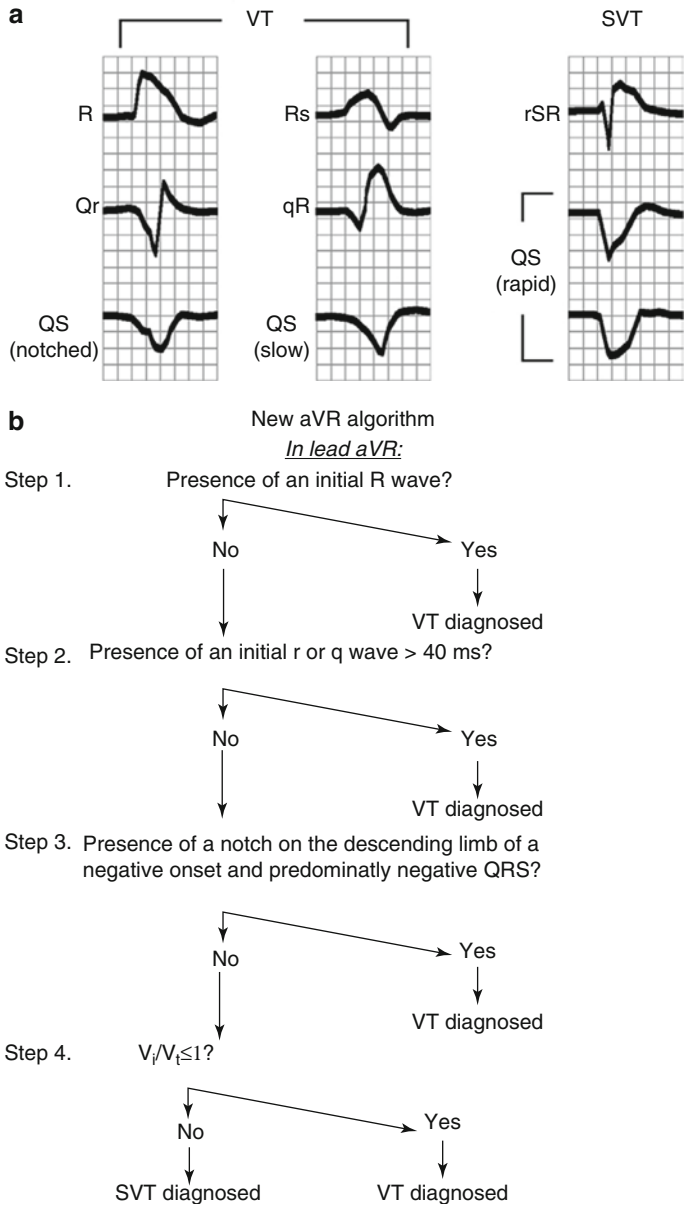

FIGURE 7.2 Algorithm to determine VT vs SVT with aberrancy by criteria developed in aVR. (**a**) By aVR QRS morphologies. (**b**) Algorithm decision ladder (Used with permission)

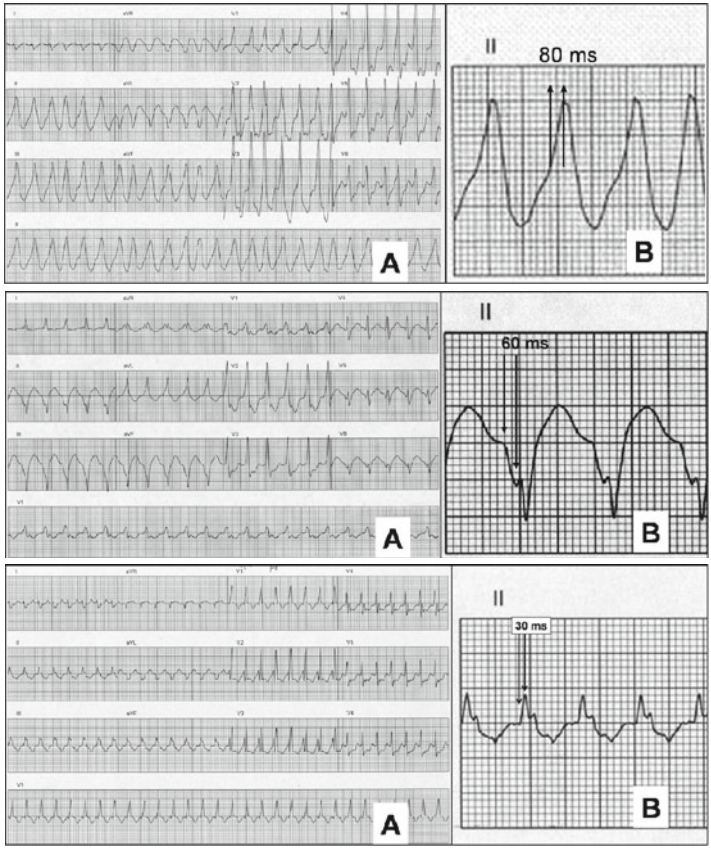

FIGURE 7.3 Algorithm to determine VT vs. SVT by measuring the duration of onset of QRS to the peak of R wave lead II. Signals measuring ≥50 ms (*top two panels*) suggest VT whereas <50 ms suggest SVT with aberrancy [4] (Used with permission)

(negative QS vector) and going towards (positive RS vector). Generally, biphasic QRS vectors mean that the origin is somewhere in the middle of that individual vector, i.e. a biphasic QRS vector in 1 and V1 likely indicate that the origin/exit site of the VT is in the septum and not on the right (RV, Q wave in V1) or left (posterior LV, Q wave in 1). This approach is primarily useful in reentrant VT's and idiopathic PVC's.

A close examination of the resting 12 lead ECG can also provide significant clues as to the underlying process and likely culprit areas of myocardial scarring. The presence of Q waves in the distribution of a coronary artery should indicate the presence of scarring that will often play a critical role in sustaining VT. Fragmentation (extra notching) of the surface QRS complex in a similar distribution to a major coronary vessel and Q waves often can provide a more sensitive indicator of myocardial scarring and may indicate the presence of epicardial scarring in that region.

For idiopathic VT's such as RVOT and LVOT VT, the appearance should be consistent with an outflow origin (inferior axis, i.e., positive in the inferior leads II/III/AVF) with an earlier precordial transition (V3 or less) indicating an LV origin and a later transition (V3 or later) indicating an RV origin. Transition at V3 could represent either LV or RV origin. Idiopathic VT utilizing the conduction system (bundle branch and fasicular reentry VT's) are quite uncommon but do have characteristic features that should set them aside from VT associated with structural heart disease. The VT is generally slower with a typical bundle branch or fasicular block appearance during tachycardia that is usually very similar to the appearance of the baseline QRS complex at rest Figure 7.12. Bundle branch and fasicular reentry are usually associated with baseline conduction disease, including bundle branch block of either right or left and prolonged AV or PR interval, with the absence of such on a resting 12 lead ECG making them very unlikely to be the mechanism for the VT.

Scar Mediated Ventricular Tachycardia

Within westernized cultures the most common underlying cause of recurrent VT is remote coronary disease. Secondary to a focus on shorter door to balloon times for the acute myocardial infarction patient, more patients are surviving their myocardial infarctions only to eventually experience downstream

congestive heart failure and ventricular tachycardia. The ischemic insult of a myocardial infarction results in the formation of myocardial scarring with variable transmural extent and resulting in altered electrical conduction within the infarcted myocardial region. Electrical conduction through scar is delayed and the resultant zone of slow conduction represents one of the critical elements of the reentrant circuit. This along with differential refractory periods in the surrounding tissue comprise the necessary substrate to sustain VT [5]. Ventricular ectopy is often the inciting event that initiates the ventricular tachyarrhythmia, either VT or VF. Premature ventricular contractions are sent through the slow conduction zone present within the ventricular scar with sufficient delay to result in their exit on the opposite side of the scarred region, finding the myocardium ready for depolarization. The electrical wave propagates around the more dense areas of scarring or anatomical barrier, such as heart valve annulus, and back into the entrance of the zone of slow conduction through the scar, creating a figure of eight pattern with the critical isthmus representing the central portion of the figure of eight (Fig. 7.4).

The typical ischemic ventricular tachycardia patient will present to medical care with either recurrent defibrillator therapies (shocks) or recurrent presyncope. Presyncope VT patients are often being treated with antitachycardia pacing and thus being prevented from progressing to either shocks or actual syncope. Of concern is the patient that presents with incessant VT or frequent - defined as >3 episodes of VT within 24 hours. This is termed VT storm and often requires multiple shocks and acute inpatient admission for management, accompanied by a significant rise in both inpatient and short term mortality [6]. The management of all of these patients is similar with a few exceptions made for the VT storm patient. In general, recurrent antitachycardia pacing or lone outpatient ICD shocks should result in the initiation or acceleration of antiarrhythmic drug therapy, starting with sotalol or amiodarone, followed by the addition of mexiletine if necessary. It is also important to make sure that these patients are adequately beta blocked since the majority of

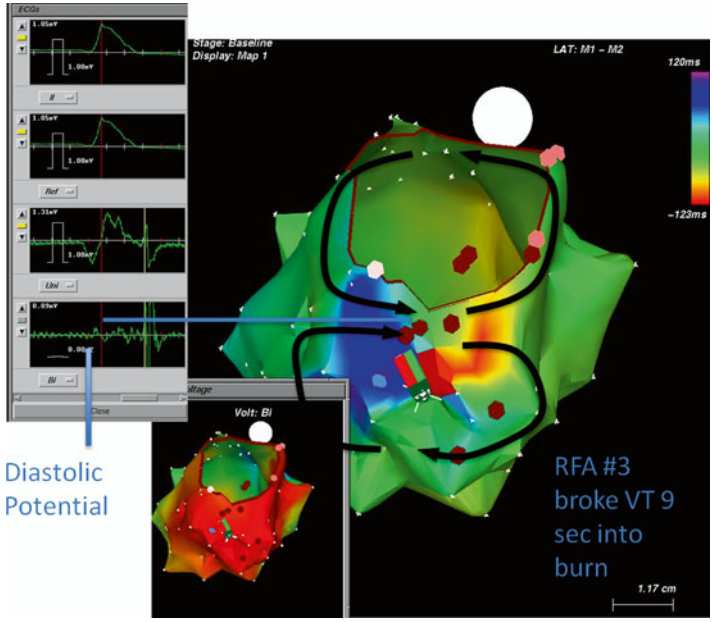

FIGURE 7.4 Figure of eight reentrant ventricular tachycardia circuit encircling around a dense inferoposterior left ventricular scar and the mitral valve annulus. The central portion of the figure of eight comprises the critical isthmus with a zone of slow conduction and the area where diastolic potentials may be recorded. This site, following confirmation with pacing maneuvers, often is the target of successful ablation

ischemic VT's are going to possess an adrenergic component and be beneficially treated with beta blockade. Once patients experience recurrent ICD therapies for VT while on an appropriate antiarrhythmic drug regimen, ventricular tachycardia ablation should be considered.

The management of VT storm patients is more difficult and varied than the ambulatory ischemic cardiomyopathy patient with recurrent ATP or lone ICD shocks for ventricular tachycardia. VT storm patients who present with recurrent ICD shocks often are accompanied by hemodynamic instability. The first step in their management is to establish control of their

ventricular rhythm. Intravenous antiarrhythmic agents are utilized acutely to achieve this with both amiodarone and lidocaine used commonly. Although limited by toxicity, lidocaine can be a useful agent especially if there are concerns for ongoing ischemia. However, a combination of amiodarone (or sotalol) and lidocaine is often required to achieve control of the malignant ventricular arrhythmias in this situation. In addition to antiarrhythmic drug therapy and aggressive beta blockade, sedation and sometimes intubation are utilized to minimize adrenergic contribution to the VT. As an illustrative point to the contribution of sympathetic tone to the generation of recurrent VT in patients with an abnormal LVEF, bilateral sympathectomy has been recently shown to be effective for the suppression of acute ICD therapies for VT storm patients and has displayed durability over medium term follow up [7].

Following the initiation of intravenous antiarrhythmic drug therapy, ongoing myocardial ischemia should be considered and evaluated, likely with coronary angiography. If myocardial ischemia is present and is the main substrate of the VT, successful treatment with revascularization often subsides the VT. However, it is usually required to accelerate the patient's oral antiarrhythmic drug therapy prior to discharge and to establish short term follow up to make sure that the treated lesion was indeed the ischemic driver of the VT.

For VT storm patients or ambulatory patients with recurrent VT despite antiarrhythmic drug therapy, electrophysiologic testing and ablation of their VT should be considered. VT ablation has evolved since its inception to a well defined repeatable process designed to identify electrical scar, the culprit ventricular tachycardia and its dependent isthmus.

Electrophysiologic Evaluation of VT with Structural Heart Disease

For patients being evaluated for EP testing and ablation of a ventricular arrhythmia it is important to provide the appropriate setting. The use of general anesthesia for these

procedures has become standard secondary to the length of the procedure and the risk for multiple defibrillations. Three dimensional mapping systems have also become standard and represent one of the most powerful tools to allow an in depth study of the ventricular chamber in question and increase the efficacy of VT ablation.

Prior to the procedure, the patient should be thoroughly assessed regarding the appropriate approach to the chamber in question. Generally the right ventricle does not pose much of a challenge outside of patients with complex congenital heart disease or tricuspid valve surgery. However, the left ventricle may prove inaccessible from a retroaortic or transseptal approach due to the presence of mechanical aortic or mitral valves. The location of culprit scar is also an important consideration as posterior, inferior and lateral scars are more easily accessible from a transseptal approach while LVOT, and anterior and septal scars are via a retroaortic approach. Peripheral arterial disease can make a retro aortic approach difficult if not impossible, which should prompt the performance of a peripheral arterial exam (bruits and pulses) prior to procedure onset. In addition, the ability to access the epicardial space (the absence of a prior sternotomy) should also be considered at procedure onset to determine if it is an option if required.

During procedure onset and throughout the duration, control of the patient's bradyarrhythmia should be available through the implanted device (in the likely event that it is present). It is best to leave patients that are not ventricular pacing dependent paced in the atrium only to allow intrinsic conduction for more accurate endocardial scar mapping. For ventricular pacing dependent patients it is often necessary to leave biventricular pacing in place to maintain appropriate hemodynamic status for the purpose of anesthesia. It may also be necessary to review the induced ventricular tachycardia on the far field electrogram through the device, which is only possible if the programmer is on and communicating with the device. The implanted device can also provide a means of intracardiac defibrillation if such a rescue is needed.

Once access to the LV endocardium or epicardium has been obtained, electrophysiologic study and ablation of ventricular tachycardia proceeds in five steps: (1) anatomic definition; (2) endocardial scar mapping; (3) induction and study of ventricular tachycardia, including entrainment mapping; (4) ablation of ventricular tachycardia, late potentials within scar; (5) attempt reinduction of ventricular tachycardia. With abolition of the clinical ventricular tachycardia and inability to induce anything other than ventricular fibrillation (rate >200 bpm) the study is complete.

To define the left ventricular anatomy a three dimensional map is generated with the aid of either fluoroscopy or ultrasound, and often both. Other than the geometry of the left ventricle itself, this often includes structures such as the mitral annulus, left ventricular outflow tract, left sided His bundle, Purkinje potentials and papillary muscles. It is important to make sure that the anatomy collected is complete. Correspondingly, the right ventricular structures including the AV node and the His conduction system, tricuspid valve, pulmonic valve, right ventricular outflow tract should be noted. Generally the risk of right ventricular perforation is higher and thus more care during mapping is needed. As the definition of endocardial scarring is dependent upon catheter contact with the wall, it is essential to know if contact is indeed present; otherwise areas will be labeled as scar inappropriately. This has been aided with real time two dimensional intracardiac echocardiography along with the evolution of contact force catheters, both of which can be used to provide definitive evidence of endocardial contact. Multi electrode mapping catheters are commonly utilized to collect a significantly larger amount of data in a shorter period of time both regarding anatomy and endocardial voltage.

Generally at the same time that anatomic information is collected, voltage is collected to provide visual information of the distribution of endocardial scarring relative to the anatomy collected. The definition of endocardial scar varies somewhat but is generally accepted to be any myocardial tissue with less than 0.5 mv. In similar fashion to the necessity of complete anatomic collection, it is just as important to

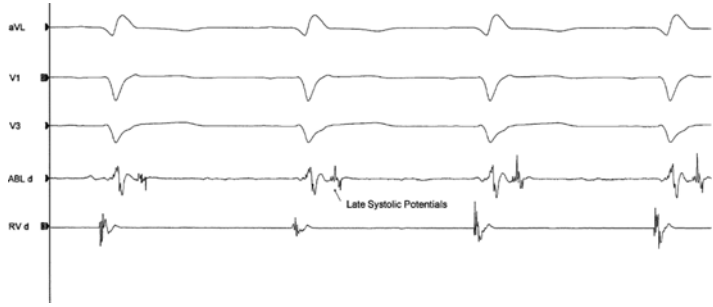

FIGURE 7.5 Late systolic potentials noted during substrate ventricular mapping recorded in sinus rhythm

generate a complete detailed voltage map. With identification of the scar, additional focus should be placed upon the scar itself for either fragmented potentials or late potentials most recently coined to be late areas of ventricular activation or LAVA. LAVA are characterized by high frequency or fragmented signals that can be either late relative to the QRS complex and local signal or buried completely within the local signal (Fig. 7.5).

In addition, areas that LV capture may be generated from within the endocardial scar are suspicious for possible VT isthmus and should be tagged for eventual ablation.

With a complete endocardial scar map, the next step within our lab is to induce ventricular tachycardia via programmed stimulation via a quadripolar pacing catheter in the RV. A predominantly substrate based approach does not involve induction of VT and ablation is carried out with the intention to abolish all LAVA within and around the endocardial scar. At this point it is crucial to reference the tachycardia 12-lead ECG for the rate and appearance of the VT, if available. Within the defibrillator population one may not ever have a surface electrocardiogram as the majority of their events are treated prior to presentation to medical care via their device. As a result only the cycle length and far field electrogram through the device are available to correlate the VT induced during the procedure with the clinical VT.

Once VT is induced matching the clinical VT the approach to study of the VT can vary from patient to patient. If the VT is hemodynamically tolerable (most often seen with slower VT's [<150 bpm] in patients with only moderate LV dysfunction) then activation and entrainment mapping may be pursued. If not hemodynamically tolerable, then the VT is pace terminated or terminated via defibrillation and pace mapping to define the VT isthmus based upon the VT morphology followed by a LAVA ablation strategy is appropriate. Both situations will be discussed in the following sections.

The Study of Hemodynamically Tolerable VT

Hemodynamically tolerable VT has become increasingly uncommon within the electrophysiology lab. Most of the VT's that are being mapped currently are fast VT's in patients with more advanced LV dysfunction. It may often be necessary to utilize vasopressors or in some cases device based hemodynamic support (Intra-aortic balloon pump, percutaneous left ventricular assist device such as Impella or ECMO) to provide adequate perfusion pressure in an effort to allow mapping. Goal blood pressures should be a mean arterial pressure above 50 mmHg.

When VT is tolerable it is beneficial to perform activation and entrainment mapping to confidently abolish the clinical VT. Points are taken and recorded via the mapping system to identify early and late EGM's with the area in the scar connecting early to late representing the slow component of the VT circuit and the critical isthmus. Within the early meets late area entrainment via the mapping catheter is performed by pacing 10–20 ms faster than the VT. Capture is maintained for sufficient time to overtake the VT circuit (5–10 beats) followed by cessation. If the morphology of the tachycardia changes during pacing, termed "manifest entrainment", and is associated with a post pacing interval (PPI) longer than the tachycardia cycle length then the pacing site is from an area outside of the tachycardia circuit. Ablation at these sites will not result in termination of VT. Suitable targets for successful

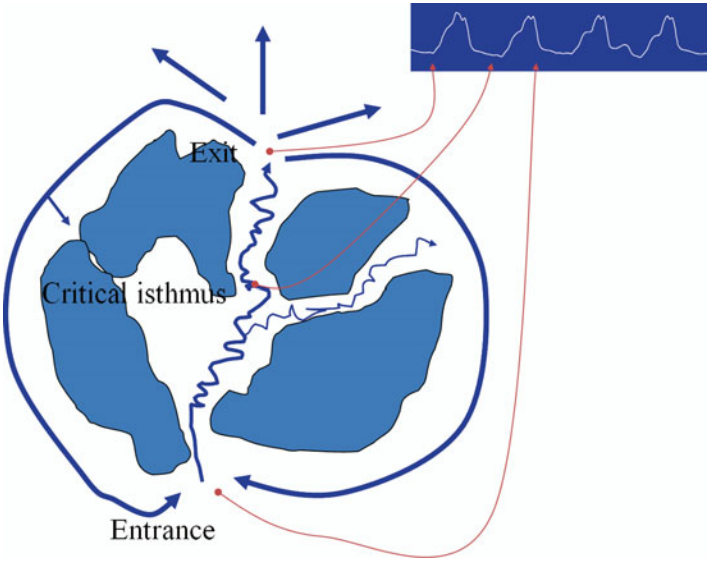

FIGURE 7.6 Diagram illustrating VT circuit with entrance, exit and critical isthmus sites denoted

ablation should results in "concealed entrainment". Pacing from these sites results in acceleration and exact morphology match as the tachycardia. Sites associated with the highest short and long term success in eliminating the VT circuit with ablation are in the critical isthmus. In addition to concealed entrainment, pacing from within the critical isthmus, the difference between the PPI and the tachycardia cycle length (TCL) will be small (likely less than 20 ms), and recording of presystolic potentials with an activation time to surface QRS similar to stimulus to surface QRS (also less than 20 ms) indicate that the mapping catheter is located within the VT critical isthmus (Fig. 7.6).

Ablation at this site typically at 30–45 W with an irrigated ablation catheter should result in the termination of VT promptly (Figs. 7.7, 7.8, and 7.9).

In some cases the isthmus is wide and some catheter manipulation is required to completely transect the isthmus.

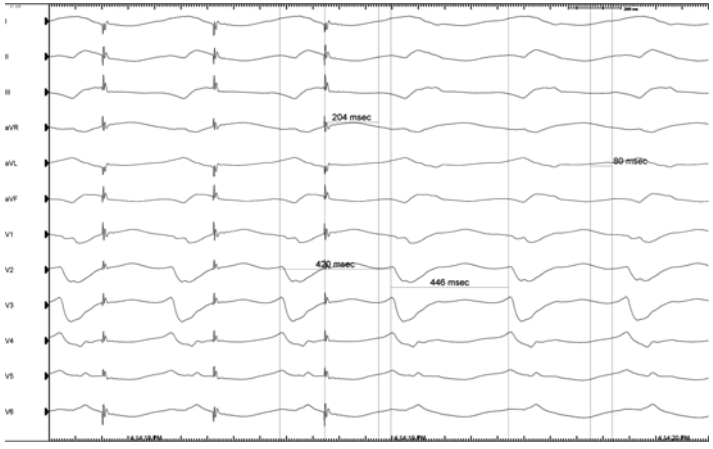

FIGURE 7.7 Pacing from the critical isthmus of the VT circuit resulting in "concealed entrainment" with 12 out of 12 lead surface ECG match in pacing morphology as compared with VT morphology

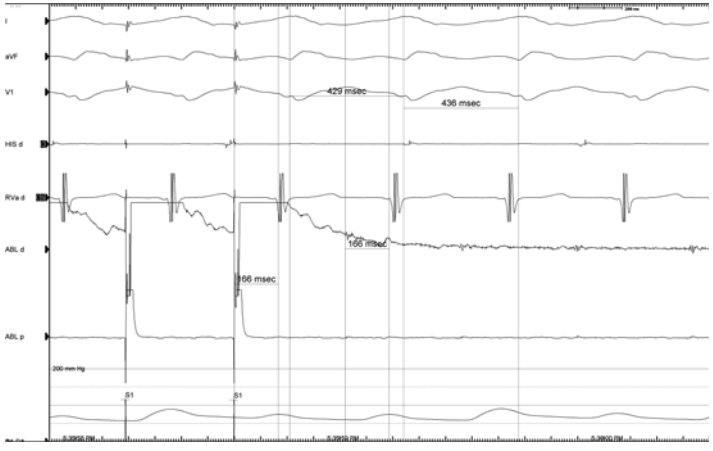

FIGURE 7.8 Other criteria for pacing within the critical isthmus of the VT circuit with pacing stimulus to QRS = EGM to QRS and post pacing interval (PPI) roughly equal to VT cycle length

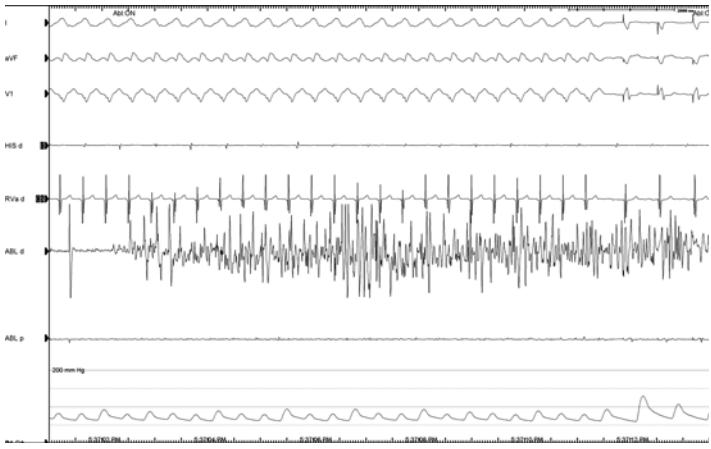

FIGURE 7.9 Ablation at the critical isthmus site results in termination of VT

Following thorough ablation of the isthmus (noncapture of the targeted region with high output pacing via the mapping catheter), the culprit VT should be rendered non-inducible. Once found to be non-inducible, scar modification via targeting of LAVA is usually undertaken to improve the long term success of the VT ablation and hopefully prevent subsequent VT's. This approach will be discussed in the third and final study/ablation approach.

The Study of Hemodynamically Unstable VT

Induction of hemodynamically unstable VT should be followed by termination of the VT either via pacing or defibrillation. The VT is then reviewed with the vector of the VT QRS utilized to identify the exit site from the identified endocardial scar. At this stage, pace mapping to the induced VT with a software based approach typically provided through the 3D mapping system is used to identify

the exit (best match, generally >90 % in 12 leads, ideally >95 % match) and entrance (worst match) with the isthmus connecting the two. Entrance and exit are usually spatially related as the length of the typical VT isthmus is usually 20–30 mm and with detailed pace mapping can be readily identified. This isthmus is transsected with thorough ablation rendering the targeted area not able to be captured with pacing and the VT should be rendered non-inducible. Following abolition of the clinical VT additional scar modification is generally performed similar to that following successful entrainment and activation guided VT ablation.

Scar Modification for VT

Scar modification or homogenization has been utilized as a primary approach for patients with unstable VT's or multiple VT's and is often performed after successful abolition of the culprit clinical VT. With detailed scar mapping, LAVA are targeted with the goal of elimination of the late potential. A more diffuse approach can also be taken where the entire scar is ablated, or linear transsecting lesions are placed through the scar, or the scar is circumscribed with contiguous ablation. Areas with the ability to capture the LV within the scar are also common targets for scar modification. The major downside of this as a primary approach is the uncertainty regarding successful treatment of the culprit clinical VT. Of the strategies available, LAVA, linear lesions, abolition of pacing capture or scar isolation, the selected strategy is often dependent upon the location and size of the scar accompanied by the comfort level of the performing physician. Recent data supports favorable results following LAVA guided scar homogenization; however, it is not always possible to completely eliminate every LAVA within the scar which may make the use of a more diffuse strategy an appropriate backup measure.

Epicardial Mapping

Often when one is unable to successfully eradicate the culprit VT via endocardial mapping and ablation, the consideration of epicardial mapping follows. It is important to consider this option when discussing the procedure with the patient to make sure that the risks of this approach are understood. Prior cardiac surgery makes this significantly more difficult and should serve as a relative contraindication to this approach.

Our approach is to obtain epicardial access via a dry pericardial tap; a subxyphoid approach is generally utilized with the area prepped and draped as per routine to start. A Tuohy needle is utilized and advanced slowly under the sternum staying as anterior as possible under fluoroscopy. The glide wire present within the needle can be advanced intermittently to see if the needle has found the pericardial space. Once the wire enters the pericardial space it should be advanced aggressively to wrap the entire outline of the pericardium in multiple loops. Once the wire is in place a short steerable epicardial sheath is advanced over the wire into the pericardial space. Following this, anatomic and voltage/scar mapping using a multi electrode or ablation catheter can proceed in the same fashion as endocardial mapping. Entrainment and pace mapping can be performed similar to the endocardium as well with the major limiting factor being the ability to capture the LV due to the presence of epicardial fat in areas. It is always important to define coronary anatomy via coronary angiography with the ablation catheter in the area of desired ablation prior to ablation in an effort to not directly ablate a major epicardial vessel. Once the scar has been mapped and the isthmus defined to be within a safe region, ablation is generally performed at 25–35 W via an irrigated ablation catheter.

With a complete ablation within the pericardium a pigtail drain is exchanged for the steerable sheath over wire with the drain left in place overnight. Prior to removal of the drain,

our practice is to infuse corticosteroids (either Solumedrol or hydrocortisone) within the pericardial space to minimize the risk of subsequent pericarditis.

Electrophysiologic Evaluation of Idiopathic VT

There is a well-defined category of VT seen in patients who are typically younger without structural or ischemic cardiovascular disease. Outflow tract VT's and high volume PVC's are the predominant arrhythmias encountered within this category. These normal heart VT's may originate from the outflow tract and some originate from the inferoposterior septal LV, termed as fascicular or "Belhassen" variant VT. It is important to have a clinical suspicion for these less common VT's as the procedural approach to their electrophysiologic study and treatment is quite different from VT with structural cardiovascular disease. VT's originating from the outflow tracts typically have an inferior axis with large R wave amplitudes in the inferior leads (Fig. 7.10).

The morphology of the idiopathic inferoposterior septal LV VT is consistent with typical right bundle branch block pattern with an LAFB axis (Fig. 7.11).

Outflow tract VT's, especially those originating from the right ventricular regions, are well understood to be due to triggered activity and are generally responsive to beta blockade and calcium channel blockade medical therapy. On the other hand, fasicular or "Belhassen VT" has also been coined "Verapamil Sensitive VT" because its mechanism is thought be due to a small circuit reentry in the inferoposterior septum that is calcium dependent. Often invasive electrophysiologic study and ablation are reserved for medical treatment failures with these agents.

For patients with symptomatic idiopathic PVC's, the majority of the clinical decision making centers around the

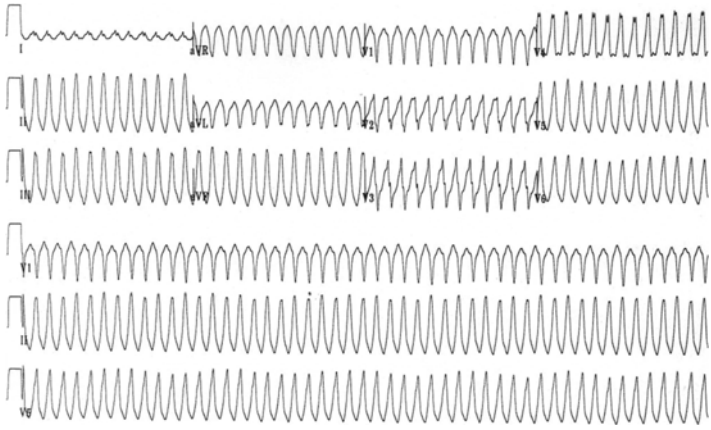

FIGURE 7.10 Twelve lead ECG of a patient with ventricular tachycardia originating from the right ventricular outflow tract. Note the typical left bundle branch block morphology with inferior QRS axis as well as the large R wave amplitudes in the inferior leads

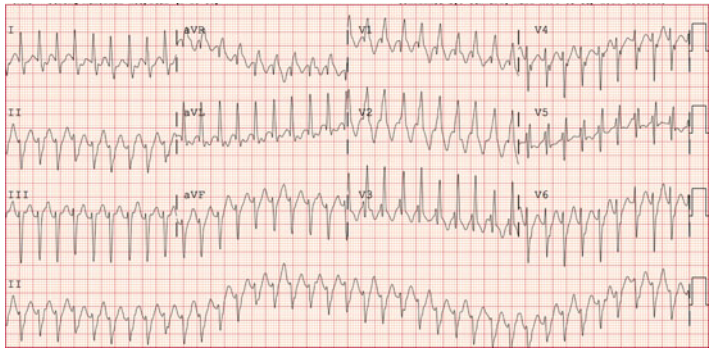

FIGURE 7.11 Twelve lead ECG of a patient with an idiopathic normal heart ventricular tachycardia originating from the left ventricular inferoposterior septum. Note the right bundle branch block morphology with superior QRS axis

burden of PVC's and their location. A PVC burden of at least 10 % or greater on ambulatory monitoring is considered high volume and at risk for developing subsequent PVC induced LV dysfunction. PVC origins within the RVOT are lowest risk for PVC cardiomyopathy with any LV location being higher risk for downstream LV functional decline. In addition, PVC's with a QRS duration of greater than 150 ms are also high risk for LV dyssynchrony and if frequent enough volume can cause LV dysfunction.

Medical therapy via beta blockade alone often may result in some reduction in the PVC burden, but some patients may still be left with high volume PVC's and at risk for cardiomyopathy even on medical therapy. Antiarrhythmic drug therapy via Class 1 (flecainide, propafenone, mexiletine) or Class III (sotalol, amiodarone) may result in a further reduction in PVC burden which may be utilized as adjunctive therapy for patients with high volume PVC's. Once on medical therapy, PVC burden should be reassessed with repeat ambulatory monitoring to make sure that the PVC burden on therapy is low enough to preclude the development of PVC induced cardiomyopathy. Persistence of high volume PVC's or symptoms despite antiarrhythmic drug therapy should prompt consideration for electrophysiologic study and ablation of the PVC focus. The presence of multiform PVC's or non-outflow tract locations are risk factors for requiring multiple procedures to effectively reduce the PVC burden and should be discussed with the patient prior to proceeding.

Electrophysiologic Testing and Ablation of Idiopathic PVC's and Outflow Tract VT's

After medical treatment failure or in patients unwilling to attempt medical therapy, electrophysiologic testing with an eye to ablation therapy for those with documented outflow tract VT or high volume idiopathic PVC's is appropriate. Of note, these procedures generally do not require general anesthesia and are best approached with as little sedation as possible secondary to a reduction in PVC burden associated

with aggressive sedation. Without spontaneous PVC's it is difficult to confidently map and eliminate the PVC. Isuprel, atropine, aminophylline and programmed stimulation from either the RV apex or outflow tract are often utilized. Generally outflow tract patients have single outflow tract PVC's that represent the source of their more sustained episodes of VT. Mapping and ablation of these PVC's usually treats their sustained VT. For PVC patients it is important to determine their triggers. If their PVC's come out when they are resting, sedation may be appropriate; in contrast, if they are associated with caffeine, aminophylline may be appropriate, etc.

The PVC morphology is captured once the patient is on the table and logged within the 3D mapping system for comparison should pace mapping be utilized. Usually QRS morphology can be used to identify location of the PVC (LVOT or RVOT) with the precordial transition being the most useful but imperfect tool. R/S transition prior to V3 can confidently be labeled as LVOT with a transition after V3 labeled as RVOT. The use of multielectrode mapping catheters for anatomic, pacing and activation mapping can make identification of the PVC focus easier. Once in the appropriate location anatomy is collected, first with particular attention to details such as the His bundle on the RV side and the aortic cusp/coronary anatomy on the LV side. Ultrasound can be of great use for aortic and LVOT PVC's/VT to minimize the risk of damage to sensitive structures such as the ostium of the left or right coronary arteries.

With anatomy collected, the preferential modality is activation mapping of spontaneous PVC's focusing on only including PVC's that are completely consistent with the clinical PVC and not catheter induced. Once the area of activation is narrowed down sufficiently the exact area should be able to be identified usually with a small pre potential prior to the larger local EGM on the mapping or ablation catheter. Favorable ablation sites usually have a pre potential or EGM onset of 35 ms or greater ahead of the surface QRS complex. When mapping in the RVOT provides no significantly early sites of activation, consideration of LV mapping should be undertaken prior to ablating. Pace mapping with software

guided matching can be used to successfully locate infrequent PVC's within the EP lab (albeit with a somewhat lower success rate) and/or confirm the best site identified via activation mapping.

Electrophysiologic Study and Ablation of Idiopathic LVVT's

Idiopathic reentrant LVVT's make up less than 5 % of the LVVT's encountered in general electrophysiologic practice (with the majority being ischemic, outflow tract or high volume PVC's). However, their behavior and treatment differs such that they are deserving of special interest. With the electrocardiographic characteristics discussed previously, it is important to have a high index of suspicion dependent upon the resting and tachycardia ECG going into the invasive electrophysiologic study. During EP study it is important to induce the VT with a morphology that is consistent with the clinical VT. Entrainment from the inferoposterior septum of the left ventricle generally provides a favorable PPI-TCL, with entrainment from the atrium remaining possible but usually showing a less favorable PPI-TCL.

Ablation of this rhythm is usually accomplished through targeting of Purkinjie potentials along the distal LV apex via a retroaortic (or transseptal) approach. Purkinjie potentials are sharp high voltage fragmented signals distal to the LBBB. Radiofrequency energy applied at this point with an ablation catheter often would terminate the tachycardia and render the VT non-inducible. With a loss of tachycardia inducibility the procedure is terminated and deemed successful.

VT of Special Consideration, Bundle Branch Reentry

Bundle branch reentry VT is a rare but important subtype of VT. These are mostly observed in patients with dilated

cardiomyopathy with underlying conduction disease. When suspicious of BBRVT, it is important to remember that BBRVT typically uses the diseased bundle branch (present on the resting 12 lead ECG and typically the left bundle branch) as the antegrade conducting limb of the circuit with the healthy limb as the retrograde limb. An important clue to bundle branch reentry VT is presentation of tachycardia that displays the same QRS morphology at baseline but with findings of atrio-ventricular dissociation (Figs. 7.12 and 7.13).

Entrainment from the right ventricular apex usually results in a favorable PPI when compared to the tachycardia cycle length. It is usually possible to entrain the arrhythmia from the atrium given the necessary participation of the His-Purkinjie system in the tachycardia, but with a long PPI relative to the tachycardia cycle length. As atrial activation is usually retrograde and thus dependent upon the reentrant circuit distal to the His bundle, changes in atrial cycle length are preceded by changes in the ventricular tachycardia cycle length. BBRVT typically has a morphology consistent with a full bundle branch block either left or more commonly right bundle branch block.

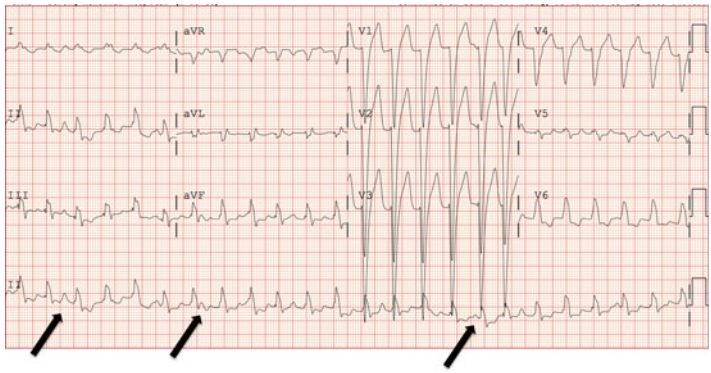

FIGURE 7.12 Twelve lead ECG of a patient with Bundle Branch Reentry VT. The *arrows* denote p waves with dissociation of atrial (p waves) and ventricular (QRS signals) signals

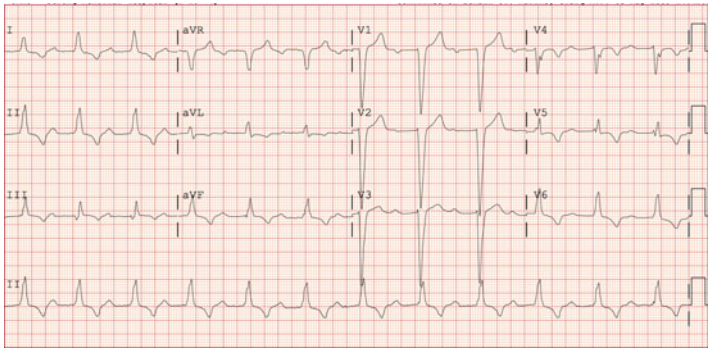

FIGURE 7.13 Baseline 12 lead ECG of the same patient with bundle branch reentry VT during sinus rhythm. Note with exact same QRS morphology in sinus rhythm as during VT

The ablation of this arrhythmia generally surrounds targeting the involved bundle branch with an ablation catheter, often during the induced ventricular tachycardia. Termination of the VT to sinus rhythm and loss of inducibility are appropriate endpoints for deeming the procedure successful and result in its termination. The right bundle potential is usually found apically to the His bundle on the septum. If the ablation catheter is positioned appropriately the right bundle potential can be separated from the His signal with a slight additional apical extension required to safely ablate the bundle branch and avoid the His bundle.

Mapping Considerations

When mapping idiopathic VT in 3D, most commonly the window of interest will be set to capture the PVCs that spontaneously occur throughout the procedure. In some rare instances in which the patient can hemodynamically tolerate the VT, a faster map can be acquired. Most of the time, however, a map will be constructed that acquires electroanatomic (EA) data only on the spontaneous PVC locations.

Anatomy is always a concern when mapping PVCs, especially in the outflow tracts, due to the way the ventricle contracts on a PVC. Continuous acquisition of anatomy during RS can confuse the picture while mapping. While it is a slower process, it may be better to take a traditional "point by point" map on the 3D mapping system (Biosene Webster's Carto system can take point by point, rather than continuous, anatomy). This way, the entire map is built as a PVC map, rather than as a mix of NSR and PVCs. Another way to acquire a consistent anatomy is to use the CartoSound mapping technology. Each ultrasound contour can be taken only on PVCs, so that a quick PVC-only anatomy can be built; EA data can then be plotted on the CartoSound map.

The key to setting a window of interest and mapping is to make sure only ventricular activation is being annotated. Points acquired by the tricuspid or mitral valve will have both atrial and ventricular signals. It is also important to be certain the points acquired are all in the intrinsic PVC, and not on a catheter-induced or clinically irrelevant PVC. This can become complicated when a patient presents with multiple morphologies of their PVC. It is usually best to try to map one PVC at a time, rather than constantly toggling between maps and risking missing beats.

Another difficulty with mapping and treating PVCs is frequency on the day of procedure. A wide variety of factors, including strength of sedation, can play a role in how many PVCs a patient has during a procedure. Electrophysiologists are often frustrated to see a large PVC burden on a holter monitor, only to have the PVCs go silent or occur only rarely during a procedure. In these instances, pace-mapping is often performed, with varying results. Traditionally this has been difficult to map and very time-consuming. Because the matching of a paced morphology to the intrinsic PVC is subjective, the best the operator and EA mapper can usually do is determine the best matches (usually placed on a scale with the 12 leads, e.g., "This is an 11/12 match") and mark these spots anatomically on the 3D map.

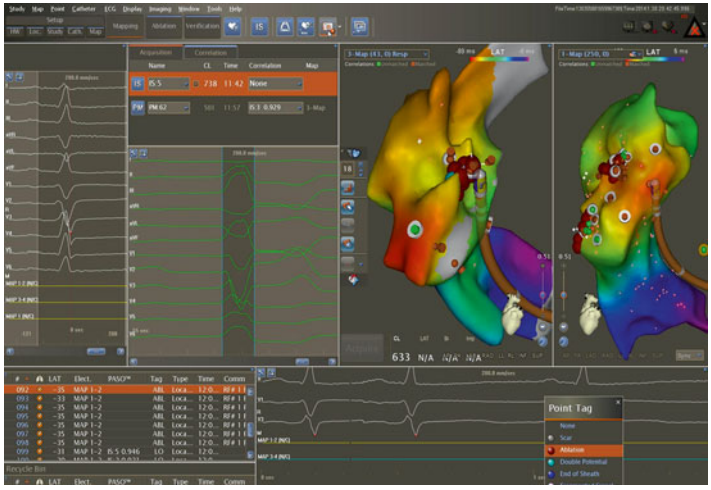

FIGURE 7.14 Paso Technology (Biosense Webster, Diamond Bar, CA). Pacemap points are tagged as *green* or *red* and given a percentage match to intrinsic PVC. Ablation strategy is targeted toward highest percentage pace map matches when intrinsic PVCs occur rarely

More recently, however, 3D mapping technology has advanced to make this process faster and more accurate. PASO software from Biosense Webster now allows the user to store the intrinsic PVC, or store multiple intrinsic PVCs, and then automatically tags and colors the map based on a percentage match to the various intrinsic PVCs (e.g., This is a 97 % match to the intrinsic PVC). This allows the electrophysiologist to pace-map multiple morphologies at a time (Fig. 7.14).

Mapping ischemic VT has its own unique challenges. Like with idiopathic VT, it is rare to map activation during VT, due to hemodynamic instability. Because VT that comes from ischemia is usually reentrant, in the rare case that activation mapping is performed, the window of interest should be set at 90 % of the cycle length, with a distinct, sharp peak of a body surface ECG lead as the "time zero" reference point.

Usually, however, substrate mapping will be performed. Setting the window of interest for mapping ischemic substrate is not complicated, but it is critical to get it right. Rather than collecting timing, the map will collect and represent bipolar voltage. Usually a scale of 0.5–1.5 mv is set – this makes everything below 0.5 mv to be considered dense scar, and everything above 1.5 mv to be considered healthy tissue. Everything between 0.5 and 1.5 mv is then displayed on a color scale representing various levels of tissue health. This color scale between 0.5 and 1.5 mv will usually show up on the "border zone" of dense scar. With a high density map, electrophysiologists will also be able to find channels of viable tissue through the dense scar, which helps locate the zones of slow conduction through the scar responsible for the reentrant tachycardias. Close attention should be paid to fragmented electrograms and late potentials, as these also represent zones of slow conduction through scar areas. Pace-map matching software can also be used to identify the critical isthmus for ischemic VT. If the VT is induced or observed during the procedure, the morphology can be saved in Paso, and pacing can be performed through scar areas to identify VT match (Figs. 7.15 and 7.16).

Substrate mapping can be problematic when careful attention is not applied to the acquired points. The points should be consistent. Mapping in SR is preferable, but not always possible. When mapping in SR, the 3D mapper must be careful only to take sinus points; paced beats or catheter-induced PVCs should be discarded. When mapping a paced ventricle, the window of interest must be set to exclude the pacing spike which will be present on the mapping catheter. If the window does not exclude this pacing spike, the map will confuse it with an actual electrogram. The most important, basic rule while mapping a substrate: anything inside the window is "seen" and measured as voltage by the mapping system! Be sure to exclude anything that is not intrinsic EGM.

A final difficulty to overcome when substrate mapping is the anatomical challenge of the left ventricle. Large papillary muscles, chordae tendonae, and trabeculation are all

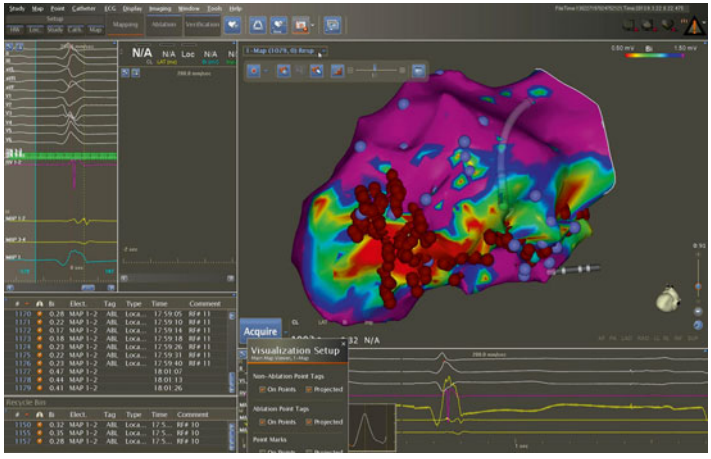

FIGURE 7.15 3D substrate map of ischemic VT. *Red areas* are dense scar (<0.5 mv). *Purple areas* are healthy tissue (>0.5 mv). *Yellow*, *green* and *blue* represent damaged "border zone" tissue. Ablation strategy here targeted a critical isthmus through the middle of the scar

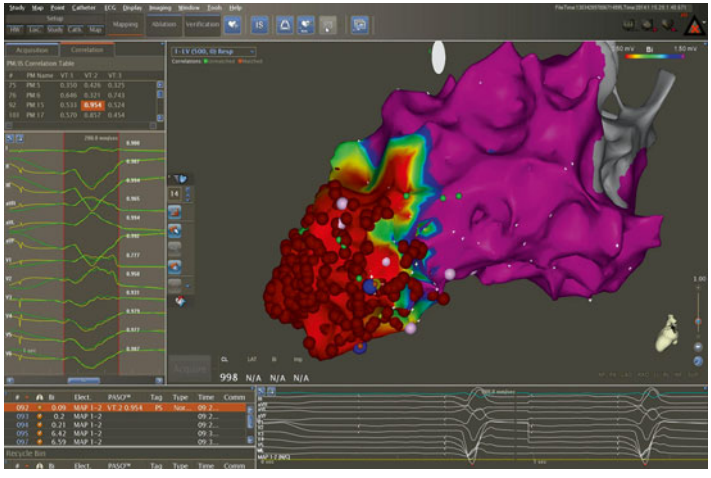

FIGURE 7.16 Paso technology in ischemic VT. Apical scar was targeted for extensive ablation. Paso revealed two channels (*small red dots* inside of dark blue tags), representing 95 % matches to two different VT morphologies. These VTs were non-inducible post-ablation

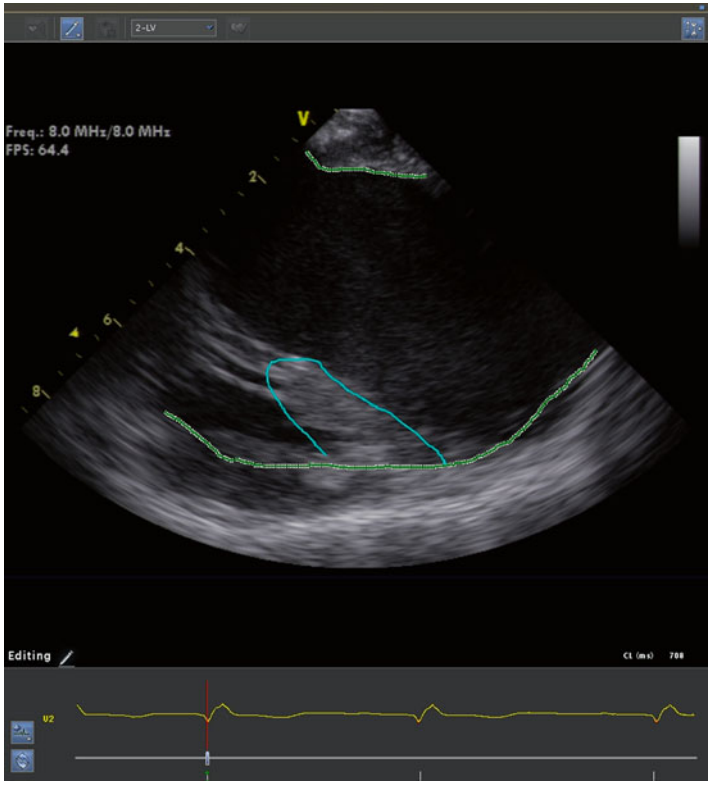

FIGURE 7.17 Papillary muscle drawn on the CartoSound technology. Papillary muscles are then represented in 3D on the map, so the catheter navigation and contact assessment can take these into account

anatomical challenges that make catheter maneuvering and mapping difficult. For this reason, phased array ICE is often used. When paired with CartoSound technology, the 3D map can actually display internal structures like papillary muscles, giving important knowledge for how to navigate and where to map (Fig. 7.17).

Additionally, with these internal structures, it can be hard to know whether or not the catheter is in good contact with the tissue. If the catheter does not have good contact, and a

point is acquired, the map will read the lack of electrogram as dense scar. This can be misleading, plotting scar on the 3D map while the catheter is actually floating off the tissue. Contact force sensing technology can solve this problem. By measuring the actual contact and force of contact against the tissue, operators can know whether they are in contact with the tissue and apply consistent force of contact throughout the map. The confusion surrounding "internal" versus "real" points on a map is virtually eliminated with a contact force sensing catheter.

Summary

Ventricular tachyarrhythmias are an increasingly common occurrence with the growing ICD population. Knowledge of the electrophysiologic characteristics and treatment of these arrhythmias continues to grow. For the majority of these patients recurrences downstream are a constant risk and may require ongoing antiarrhythmic drug therapy for complete suppression. In experienced hands it is possible to study and treat these arrhythmias successfully in a safe fashion. However, as our knowledge of the benefit of VT ablation grows it is becoming increasingly clear that this procedure, when performed properly, can further reduce the risk of subsequent ICD shocks and mortality [8, 9].

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