Review Article

Methods and Indications for Ablation of Ventricular Tachycardia

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Key words:
Ventricular tachycardia, electroanatomical mapping, ablation.

Manuscript received:
September 17, 2010;
Accepted:
April 18, 2011.

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Ventricular tachycardias (VTs) are broadly divided into two categories based on the presence or absence of structural heart disease. This division entails differences in the mechanism, the prognosis, and the treatment of ventricular arrythmias.[1-6]

Idiopathic VT is observed in patients who have no structural heart disease and rarely causes sudden cardiac death.[3,4] Ablation is indicated in cases of symptomatic, drug-refractory ventricular ectopy or tachycardia. Most idiopathic VTs have a focal origin, where the activation spreads out from the centre towards the periphery.[1-4]

In contrast to idiopathic VT, VT in the setting of structural heart disease is associated with increased mortality and a high incidence of sudden cardiac death.[1,2,5,6] An implantable cardioverter-defibrillator (ICD) is the first-line therapy in the majority of cases. Recurring episodes of VT are observed in 40-60% of patients with a history of spontaneous sustained VT who receive an ICD.[6] Multiple ICD discharges significantly affect quality of life, lead to repeat hospitalisations, and are associated with higher mortality.[7-9] Treatment with antiarrhythmic drugs can be attempted; however, it is often ineffective and can lead to adverse effects, including a proarrhythmic action. In these cases, ablation is useful for the reduction of the number of discharges.[10] Also, ablation is clearly indicated in cases of electrical storm.[11,12]

The most common mechanism of VT in patients with structural heart disease is scar-related re-entry.[1,2,5,6] It often appears late after myocardial infarction and in patients with cardiomyopathies. The re-entry circuit is usually maintained by a slow-conduction zone (isthmus), which is created by surviving myocardial cells between zones of interstitial fibrosis and which exhibits properties of very slow conduction. This zone is electrically "silent" and the QRS complex recorded on the surface ECG represents the activation of the remaining ventricular myocardium. The start of the QRS denotes the exit of the electrical front from the protected isthmus. The tachycardia is further stabilised by regions of dense, refractory scar tissue and/or neighbouring anatomical structures (such as the mitral annulus), which act as barriers to conduction. In most cases, this anatomical formation allows the existence of multiple re-entry circuits, which result in different kinds of VT and can be triggered via programmed stimulation in the electrophysiological laboratory.[1,2,4,6]

The surface ECG provides important information in these patients. The sinus

rhythm ECG may give information about the underlying heart disease (e.g. old myocardial infarction, arrhythmogenic right ventricular cardiomyopathy). The VT may be monomorphic, polymorphic, or pleomorphic. The morphology of the QRS complex on the 12-lead ECG in a monomorphic VT indicates the source of a focal VT or the exit zone of a scar-related VT (Figure 1). VTs with left bundle branch morphology have the focus or exit zone in the right ventricle or in the interventricular septum of the left ventricle. In contrast, a right bundle branch morphology indicates a focus or exit zone in the left ventricle. A superior axis shows an origin or exit from the inferior wall, whereas an inferior axis indicates the outflow tract or the anterior wall. The precordial leads help to locate the VT between the base and the apex. A dominant S-wave indicates a focus or exit zone in the apex, whereas a dominant R-wave locates the focus or the exit channel in the base.

Apart from the endocardial re-entry circuits there are endomyocardial and epicardial circuits. A high incidence of epicardial re-entry circuits was initially observed in Chagas disease.[2] In non-ischaemic cardiomyopathy larger epicardial regions with low potentials are observed, compared with the endocardial surfaces that are located close to the valve annuli.[10] In contrast to non-ischaemic cardiomyopathy, patients with ischaemic cardiomyopathy tend to have a larger endocardial than epicardial scar. A high incidence of epicardial circuits is observed in patients with old infarctions of the left ventricular inferior wall.[8] In patients with arrhythmogenic right ventricular cardiomyopathy there are epicardial regions with fractionated or late potentials. The epicardial scar is consistently bigger than the endocardial scar. There is suspicion of epicardial VT when the ascending branch of the QRS shows a pseudo-delta wave (>34 ms), wide QRS complex (>198 ms) and an increased intrinsicoid deflection time in lead V_2 (>85 ms).[13]

The approach for VT ablation is either retrograde through the aortic valve or through the intra-atrial septum via a transseptal puncture, while in the case of epicardial circuits a subxiphoid approach is used. In patients with recurring VT who are refractory to both drugs and ablation, surgical ablation or excision of the arrhythmogenic focus is a therapeutic option that is used in specialised centres. Surgical treatment requires preoperative and intraoperative mapping in order to determine the arrhythmogenic regions precisely.[1]

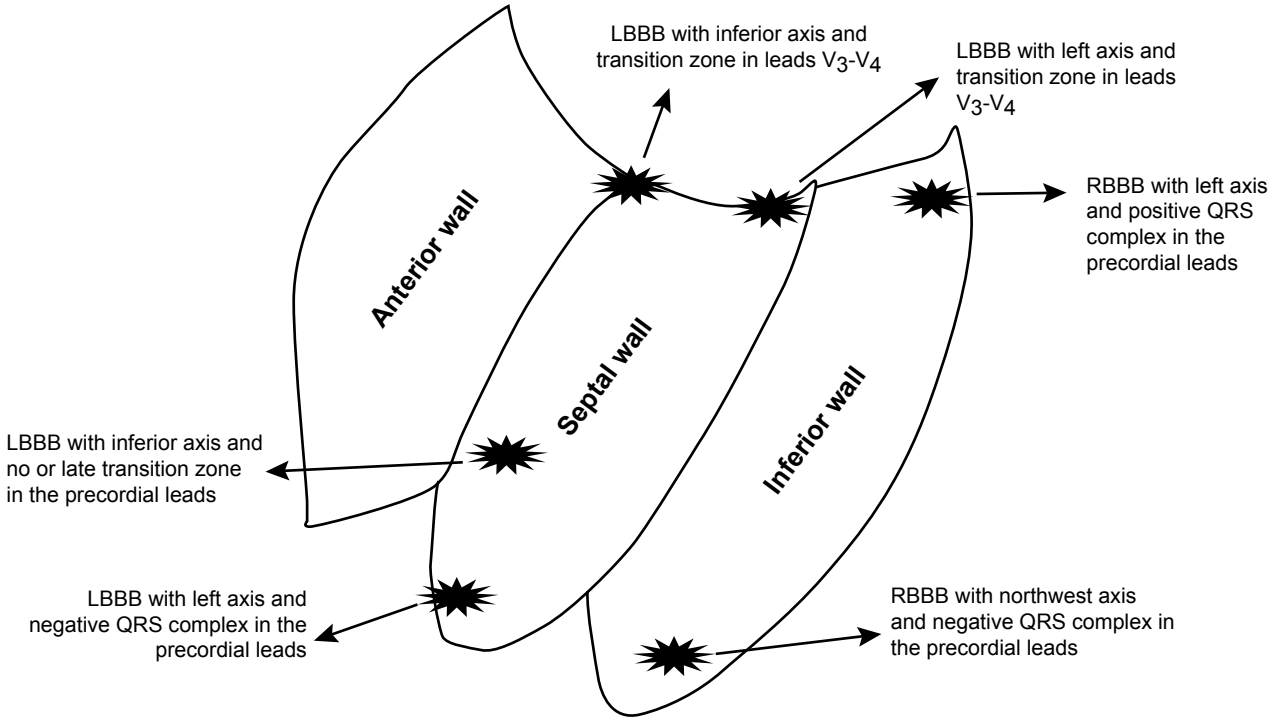

Figure 1. Morphology of the QRS complex on the 12-lead ECG in scar-related ventricular tachycardia, according to the focus or exit zone in the left ventricle. LBBB: left bundle branch block; RBBB: right bundle branch block.

VT mapping

The selection and use of various mapping techniques in patients with VT depends on the mechanism of the arrhythmia and on whether the mapping must be carried out in sinus rhythm or during the VT. Mapping during VT requires both VT inducibility and haemodynamically acceptable tolerance. The target of ablation in idiopathic VT is the discrete focus of the arrhythmia, which does not exhibit anatomical or electrophysiological abnormalities. Mapping to identify the location of the focus is carried out during the VT. In contrast, in scar-related VT the ablation target is the slow-conduction zone (isthmus). This zone may be located anatomically during sinus rhythm, although its participation in the VT circuit must be proven during the VT using pacing manoeuvres.

Activation mapping

Activation mapping is recommended for reproducing the course of the electrical front. This is achieved by comparing the local activation time at various points in relation to a pre-selected point of reference. The electroanatomical mapping systems CARTO® (Biosense Webster, Diamond Bar CA, USA) and NavX® (Endocardial Solutions, St. Jude Medical, Inc., St. Paul MN, USA) can create a three-dimensional reproduction of the course of the electrical stimulus in the ventricular myocardium by demarcating a specific activation time for each selected point and converting these arithmetic data into a coloured map. These maps can contribute to a better understanding of the tachycardia's mechanism and circuit.[14,15] The CARTO system uses three magnetic fields that are created below the patient's chest. A magnetic field sensor that is incorporated in the tip of the ablation catheter measures the magnetic field strength and converts it to an optical signal in the three-dimensional space.

Non-contact mapping allows the simultaneous recording of the stimulus from multiple points and can be especially useful in the reproduction of activation maps during haemodynamically unstable or non-sustained VT.[16] Briefly, the Ensite non-contact mapping system (Endocardial Solutions, St. Jude Medical, Inc., St. Paul MN, USA) uses a multi-electrode array to record electrical signals endocardially from various sites. This catheter has the form of a balloon with 64 electrodes on its surface and can be used to record a single extrasystole reliably. The three-dimensional visualisation of the electrodes on the surface of the balloon and the geometry of the cavity is achieved through the application of a low-level electrical field between each balloon electrode and a catheter.

Idiopathic VT is usually well tolerated clinically and is therefore suitable for the above mapping techniques. The target of ablation in these focal tachycardias is the point with the earliest activation. Apart from the timing of the electrograms, careful analysis of the unipolar electrograms, which usually have a QS morphology, can be useful in locating the focus of the VT.

Monomorphic VTs due to scar are usually re-entrant tachycardias.[5,6] Mapping should be aimed at the isthmus or the exit of the stimulus from the ventricular myocardium. The isthmus is reproduced during the VT with mid-diastolic potentials located between two successive QRS complexes (Figure 2). Presystolic potentials that precede the QRS complex reproduce the exit of the VT circuit. However, the real contribution of these potentials to the VT circuit must be confirmed by entrainment mapping.

Entrainment mapping

Entrainment mapping is used as a supplement to activation mapping in re-entry VT (Figure 3). The basic limitations of entrainment mapping are when "clinical" VT cannot be induced or is poorly tolerated haemody-

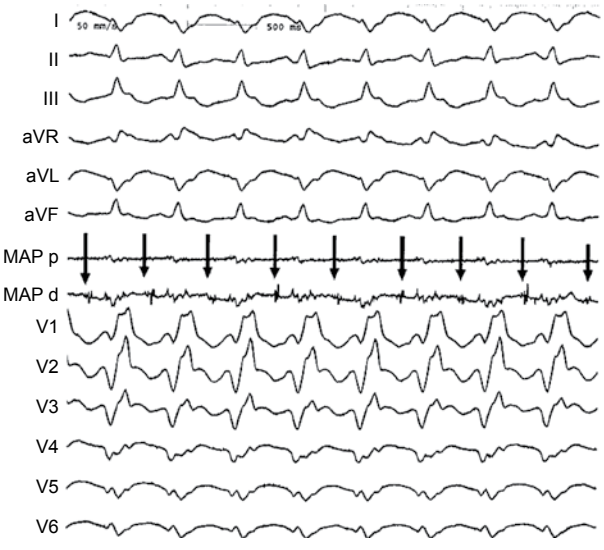

Figure 2. Mid-diastolic potentials during ventricular tachycardia. The tracings show the 12-lead ECG (50 mm/s) and bipolar recordings from the distal and proximal poles of the ablation catheter (Map p and Map d). The tachycardia has a cycle length of 450 ms and a right bundle branch block morphology. Bipolar electrograms of the distal poles of the ablation catheter (Map d) show mid-diastolic potentials (arrows).

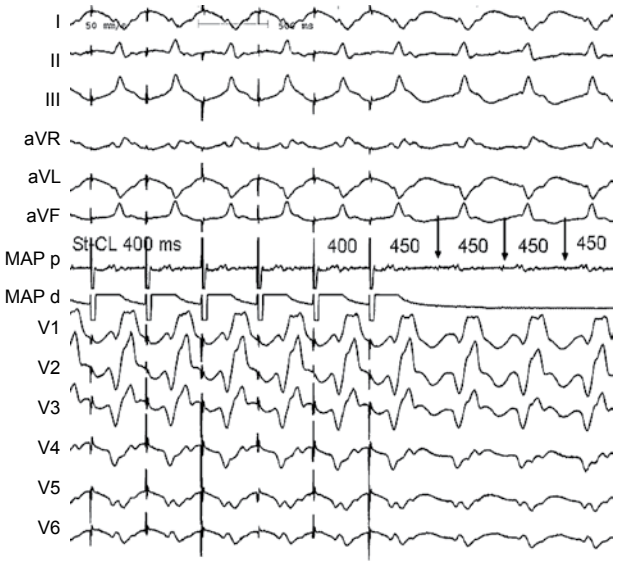

Figure 3. Entrainment mapping showing concealed entrainment. The electrograms are as in Figure 2. Entrainment mapping is performed over the tip of the catheter with a cycle length of 400 ms. The QRS complex arising from the stimulus has the same morphology as the ventricular tachycardia in all 12 leads (concealed entrainment). The stimulus-QRS interval during pacing corresponds to the local potential-QRS interval during the tachycardia and the post-pacing interval matches the tachycardia cycle length. This proves that the ablation catheter has been placed in the isthmus zone of the re-entry circuit.

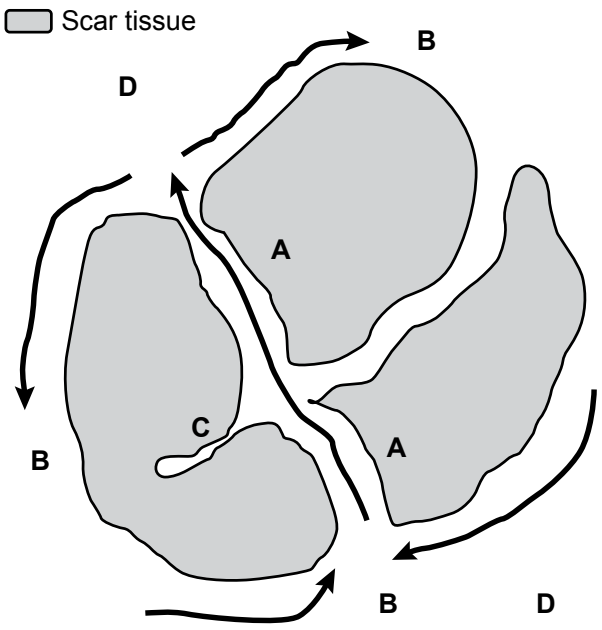

Figure 4. Basic principles of entrainment mapping (see text).

namically. It consists of continuous resetting of the VT circuit by performing overdrive pacing approximately 20 to 30 ms faster than the tachycardia cycle length. For the evaluation of entrainment mapping, the tachycardia must be accelerated transiently to the paced cycle length and then allowed to return to its own cycle length after the cessation of pacing. The presence or absence of "fusion" (changes in the morphology of the QRS complex) during pacing, as well as the duration of the post-pacing interval (PPI) at the stimulation site should be analysed thoroughly. As shown in Figure 4, the sites/regions of the ventricular myocardium that may or may not participate in the VT cycle based on entrainment mapping are classified as follows:[17]

A. Pacing from the slow-conduction zone (isthmus) leads to concealed entrainment (same QRS morphology during stimulation and spontaneous VT) with a similar PPI (±30 ms), while the stimulus-QRS interval during pacing is comparable with the local potential-QRS interval during tachycardia (±20 ms). The protected isthmus with slow conduction is the ideal target for ablation.
B. Entrainment from zones within the scar that do not participate actively in the tachycardia circuit may lead to concealed entrainment, but it will typically display long PPI (adjacent bystanders).
C. Points corresponding to the outer loop of the circuit exhibit entrainment with fusion, while the PPI matches the VT cycle length.
D. Points far away from the VT circuit (remote bystanders) show entrainment with fusion and a prolonged PPI (>30 ms more than the tachycardia cycle length).

Substrate mapping

Substrate mapping is a useful way to go beyond the limitations of activation and entrainment mapping as regards scar-related VT. It is recommended for the identification of an anatomical substrate that could lead to VT (scars and zones of slow conduction) through the creation of a coloured, three-dimensional map of the left ventricle that records the amplitude of the local electrogram potential during sinus rhythm. Regions with low potential (<1.5 mV) are considered pathological.[5,6,14,18] Apart from regions designated as scar (<0.5 mV), points should be recorded on the map to depict isolated end-systolic (split potentials) or fragmented potentials (>133 ms) and may represent zones of anisotropy or slow conduction that are responsible for maintaining the tachycardia (Figure 5). Isolated

end-systolic potentials that are separated from the local ventricular electrogram by an isoelectric interval ≥20 ms have a sensitivity and specificity of 80% and 84%, respectively, in the detection of a slow-conduction zone. For an isoelectric interval ≥50 ms the sensitivity and specificity are 54% and 90%, respectively.[19]

When the VT is inducible and stable, short entrainment should be performed at the same time as substrate mapping in order to limit the number of ablation lesions. Many ablation strategies target the anatomical substrate of the tachycardia, mainly via the creation of linear lesions that cross the isthmuses.[11,2,5,6,20]

Pace mapping

Pace mapping consists in stimulating different sites during sinus rhythm to compare the morphology of the stimulated QRS complexes to the clinical VT morphology. Pace mapping is very useful for locating the origin of focal VT (Figure 6), especially when only a few ventricular extrasystoles are recorded or when clinical VT cannot be induced. However, it is less precise than activation mapping.

In scar-related VT, stimulation from any point within the slow-conduction zone produces a 12-lead ECG having identical morphology to that of the VT, proving that the activation exits the isthmus at the same point as in clinical VT. The gap between the stimulation point and the VT exit point indicates the conduction delay in the slow-conduction zone.[21] Pace mapping can be used to supplement substrate mapping in the determination of electrically refractory zones that may demarcate a possible isthmus zone,[22] and exit zones at the scar boundary that show a QRS morphology similar to clinical VT, with a short stimulus-QRS interval.[19,23] Regions with excellent pace mapping and a stimulus-QRS interval >40 ms correspond to the slow conduction region (isthmus).

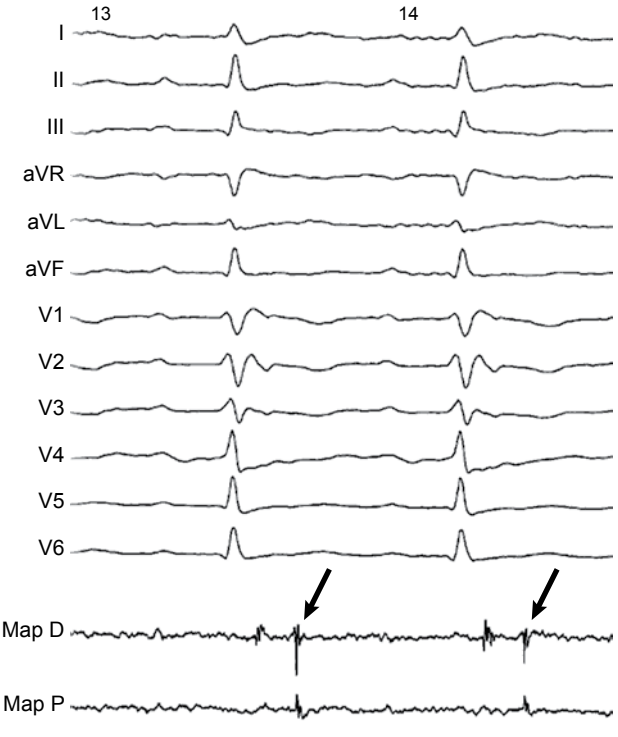

Figure 5. Isolated end-systolic potentials (arrows) that represent zones of anisotropy or slow conduction in a patient with arrhythmogenic right ventricular cardiomyopathy.

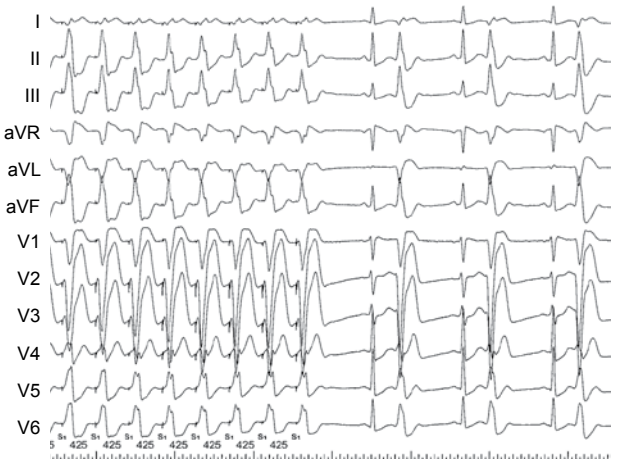

Figure 6. Pace mapping in a patient with extrasystolic ventricular arrhythmia from the right ventricular outflow tract, showing that the extrasystole and the paced beat are absolutely identical (12/12).

Failure of endocardial ablation raises the suspicion of an epicardial substrate, especially in the presence of specific electrocardiographic features.[13] The pericardial area may be reached either via a subxiphoid approach[24] or surgically.[25] Epicardial ablation should be performed using an irrigated tip catheter. High amplitude pacing and coronary angiography, respectively, are essential to determine the route of the phrenic nerve and rule out the presence of coronary arteries close to the ablation region.[26]

Ablation of idiopathic VT

Idiopathic VT from the right or left ventricular outflow tract (adenosine-sensitive)

Tachycardias originating from the outflow tract are the most common forms of idiopathic VT. They usually originate from the right ventricular outflow tract (RVOT), although a number of other foci have been

described and will be discussed below.[3,4] The R/S ratio in lead V_3 is useful for orientation of the mapping between right and left ventricular outflow tract (LVOT). If R<S in V_3, the tachycardia probably originates from the RVOT; whereas if R>S with a dominant R-wave in V_1 or V_2, an LVOT origin should be suspected. When the R-wave is equal to the S, the origin may from either the RVOT or the LVOT, or from the epicardium.[27]

VT from the RVOT

Various ECG algorithms have been created for the orientation of electrical mapping in the RVOT.[28,29] An example of a posteroseptal RVOT is presented in Figure 7A. The precise origin of the VT is located using both activation mapping (seeking the earliest activation, about 30 ms before the onset of the QRS complex) and pace mapping. The rate of successful ablation of VTs from the RVOT varies from 65% to 95% according to the literature.[30-32] The complication rate is low, although some cases of cardiac tamponade have been reported after perforation of the right ventricular free wall. Rarely, the VT may originate from muscle sleeves that extend above the pulmonary artery.[33]

VT from the LVOT

Tachycardias from the LVOT originate from the upper section of the left interventricular septum, immediately below the aortic valve or its cusps.[34-36] The septal sites (around the His bundle) show a QS or Qr morphology in lead V_1. In contrast, all the other sites show a qR, R or Rs morphology in lead V_1 (Figure 7B). More specifically, the presence of a qR morphology in lead V_1 is considered pathognomonic for the aorto-mitral continuity (Figure 7C, Figure 8).[36] VT may also originate from the left ("M" or "W" morphology in lead V_1) or the right aortic cusp (rS or QS morphology in lead V_1).[35,36] These tachycardias originate from extensions of ventricular myocardium that cross the aortic annulus. To avoid acute obstruction of the left or right coronary artery, it is necessary to use coronary angiography or intracardiac ultrasound to monitor the position of the ablation catheter during radiofrequency current delivery.

Idiopathic left fascicular VT (verapamil-sensitive)

The clinical features of idiopathic left fascicular VT, as described by Zipes et al[37] and Belhassen et al,[38] include the absence of structural heart disease, easy induction with atrial pacing and sensitivity to verapamil. There are three types of bundle VT: 1) left posterior fascicular VT, with right bundle branch block (RBBB) morphology and left axis deviation (most common type, 90%; Figure 7D); 2) left anterior fascicular VT, with RBBB morphology and right axis deviation; and 3) upper septal fascicular VT, with a narrow QRS and a normal or right-deviated axis. The VT circuit is not fully understood. It includes the left Purkinje system (posterior and anterior left bundle), while the ventricular myocardium probably participates as a bridge. Ablation should target diastolic potentials during VT.[39,40] If it is not possible to record diastolic potentials we target pre-systolic Purkinje potentials fused with the ventricular electrogram during VT. Pace mapping is not particularly useful in this kind of VT. Linear lesions in the distal third of the posterior fascicle (inferior interventricular septum), avoiding the proximal Purkinje system, are usually successful.[41] The success rate recorded in the literature is about 80% and complications are rare.

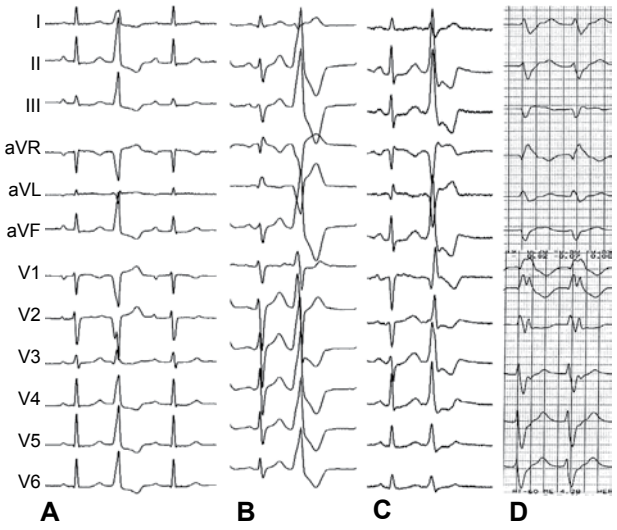

Figure 7. Idiopathic ventricular extrasystoles from the right ventricular outflow tract (A), left ventricular outflow tract (B), aorto-mitral continuity (C), and left posterior fascicular ventricular tachycardia (D).

VT ablation in patients with structural heart disease

Bundle branch re-entry VT

This tachycardia usually occurs in patients with structural heart disease and a diseased Purkinje system, which translates electrophysiologically into a prolonged HV interval.[42] Most patients have left bundle

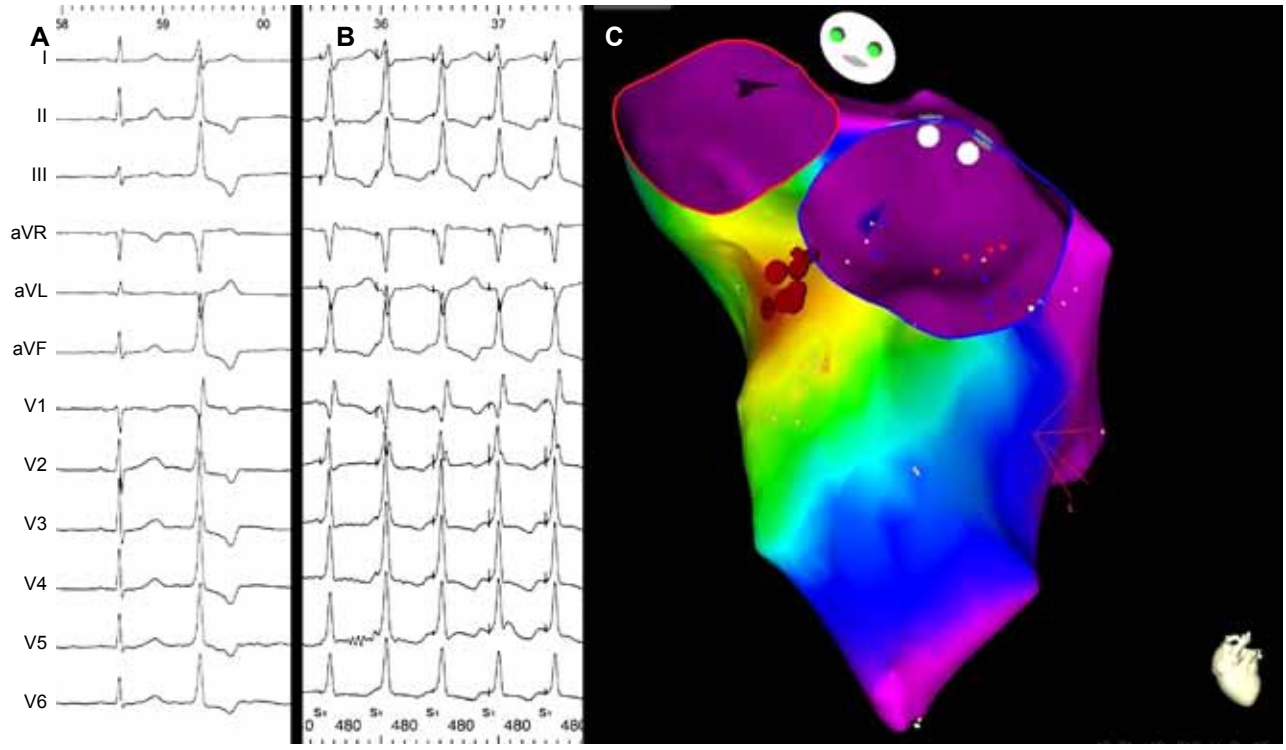

Figure 8. Successful ablation of extrasystolic ventricular arrhythmia from the aorto-mitral continuity. A: surface ECG with qR morphology in lead V_1 on the ventricular extrasystole; B: pace mapping with absolutely identical complexes (12/12); C: activation mapping with the CARTO 3 three-dimensional imaging system. The red dots correspond to the sites of successful ablation.

branch block (LBBB) on the surface ECG. In the usual type of bundle branch re-entrant VT, the wavefront is conducted antegradely via the right bundle branch and retrogradely via the left bundle branch. Thus, the VT exhibits an LBBB morphology. During tachycardia, the HV interval is greater than or equal to the HV during sinus rhythm. Also, as in all re-entrant tachycardias, it is susceptible to entrainment mapping. Treatment consists in ablation of the right branch of the His bundle. However, in about 30-40% of patients implantation of a pacemaker and/or defibrillator is required because other kinds of VT often coexist.[42]

Re-entrant VT due to scar

After myocardial infarction

As already discussed, VTs following a myocardial infarction involve scar-related re-entrant tachycardias. Ablation should target the slow-conduction zone (isthmus), which is usually located within the scar and is responsible for maintaining the re-entry circuit. The best way to locate the isthmus is by using activation or entrainment mapping. As mentioned above, this is not always feasible. An alternative solution is substrate mapping (Figure 9). In any case, ablation aims to eliminate the patient's clinical tachycardia and not the various "non-clinical" VTs that may be observed during the course of the procedure. The use of a cooled tip catheter that causes larger and deeper lesions is to be preferred in ablation of this VT.[43] Finally, the presence of an epicardial circuit should always be taken into consideration, as it is present in 10-30% of these VTs. VT ablation following myocardial infarction is initially successful in 70-95% of patients when substrate mapping is used.[6] However, comparisons are difficult since the endpoint of ablation differs among various studies. VT reoccurs in 20-50% of patients, although in the majority of cases the frequency of episodes is reduced. A recent study showed that preventive VT ablation based on the substrate succeeded in significantly reducing the number of defibrillator discharges during follow up.[10] Furthermore, the VTACH study found that the time to first discharge was significantly longer in patients who underwent ablation.[44]

Dilated cardiomyopathy

Although VTs in patients with dilated cardiomyopathy are also scar-related re-entrant tachycardias,

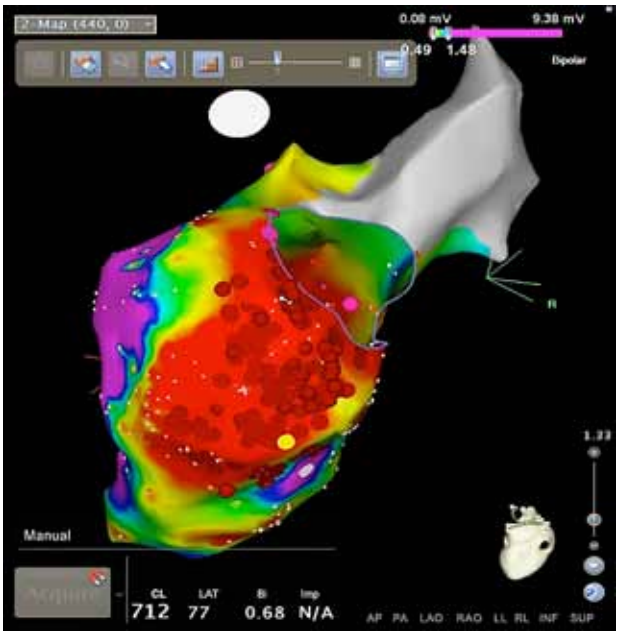

Figure 9. Substrate mapping with the CARTO 3 three-dimensional imaging system and ablation of the regions with low potentials (red dots) in a patient with ischaemic heart disease.

radiofrequency ablation is more challenging than in post-infarction patients because of the greater complexity of the substrate. These patients exhibit multiple scar regions that result in the development of different re-entry circuits. Epicardial circuits occur in more than 30% of these patients. The success rate described in the literature varies between 35% and 60%.[6]

Arrhythmogenic right ventricular cardiomyopathy

In patients with arrhythmogenic right ventricular cardiomyopathy, the re-entry circuits include scar regions around the tricuspid or the pulmonary valve annulus and lead to VT with an LBBB morphology. Epicardial circuits are also likely in this disease.[45,46] The precise success rate after ablation is unknown and depends on the progress of the disease.

Ablation of polymorphic VT and ventricular fibrillation

Haissaguerre et al were the first to describe a new approach to the ablation of polymorphic VT or ventricular fibrillation.[47] Because of the unstable nature of these arrhythmias, the method does not aim at mapping during the tachycardia, but at mapping and ablation of the original trigger (ventricular extrasystole) that is most commonly located in the Purkinje system (pre-systolic Purkinje potentials). Since then, this approach has been used in patients with long QT syndrome, Brugada syndrome,[48] or after a recent myocardial infarction,[49,50] as these patients exhibit frequent polymorphic VT or ventricular fibrillation. In these small studies success rates of up to 90% have been reported.

Complications of VT ablation

Catheter ablation of VT is a complex invasive procedure that entails severe risks, especially in the case of patients with advanced structural heart disease. In contrast, complications are rarely observed in the ablation of idiopathic VTs. Major complications are considered to be those that lead to a prolongation of hospitalisation, or require an additional procedure for treatment, or result in severe damage or death. Severe complications are observed in 8% of patients with advanced disease and mortality has been reported as 3%.[1,2] Significant vascular lesions at the perforation site (haematoma, arteriovenous shunt, pseudoaneurysm) are seen in 2% of patients.[1,2] Thromboembolic phenomena have been reported in 1.3% of patients who undergo ablation for VT.[1,2] The use of irrigated tip catheters and meticulous anticoagulation during the procedure reduces the risk of occurrence of thromboembolic episodes. Cardiac tamponade has been reported in 1% of cases.[1,2]

Indications for VT ablation

According to current guidelines (EHRA/HRS Expert Consensus on Catheter Ablation of Ventricular Arrhythmias),[1] catheter ablation of VT in the setting of structural heart disease is recommended:

- for symptomatic sustained monomorphic VT (SMVT), including VT terminated by an ICD, that recurs despite antiarrhythmic drug therapy or when antiarrhythmic drugs are not tolerated or not desired;
- for control of incessant SMVT or VT storm that is not due to a transient reversible cause;
- for patients with frequent premature ventricular complexes, non-sustained VT, or VT that is presumed to cause ventricular dysfunction;
- for bundle branch re-entrant or interfascicular VT;
- for recurrent sustained polymorphic VT and ventricular fibrillation that is refractory to antiarrhythmic therapy when there is a suspected trigger that can be targeted for ablation.

Catheter ablation of VT is recommended for patients with idiopathic VT:

- for monomorphic VT that is causing severe symptoms;
- for monomorphic VT when antiarrhythmic drugs are not effective, not tolerated, or not desired;
- for recurrent sustained polymorphic VT and ventricular fibrillation (electrical storm) that is refractory to antiarrhythmic therapy when there is a suspected trigger that can be targeted for ablation.

Conclusions

Recent years have seen significant progress in both our understanding of the pathophysiological mechanisms of VT and the development of mapping and ablation techniques. This has allowed specialised centres to carry out ablation of VT in patients with or without structural heart disease, with a high success rate and few complications.

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