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EDITORIAL

# Role of catheter ablation of ventricular tachycardia associated with structural heart disease

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## Abstract

In patients with structural heart disease, ventricular tachycardia (VT) worsens the clinical condition and may severely affect the short- and long-term prognosis. Several therapeutic options can be considered for the management of this arrhythmia. Among others, catheter ablation, a closed-chest therapy, can prevent arrhythmia recurrences by abolishing the arrhythmogenic substrate. Over the last two decades, different techniques have been developed for an effective approach to both tolerated and untolerated VTs. The clinical outcome of patients undergoing ablation has been evaluated in multiple studies. This editorial gives an overview of the role, methodology, clinical outcome and innovative approaches in catheter ablation of VT.



**Key words:** Catheter ablation; Electroanatomic mapping; Implantable cardioverter-defibrillator; Radiofrequency energy; Sudden cardiac death; Ventricular tachycardia

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## INTRODUCTION

Ventricular arrhythmias may occur in patients with or without structural heart disease. A dissertation on ablation of the different forms of ventricular arrhythmias in different patient settings may be too long. Therefore, this editorial will focus only on catheter ablation of ventricular tachycardia (VT) associated with structural heart disease. A list of diseases possibly associated with VT is shown in Table 1.

Catheter ablation is a therapeutic option aimed at the prevention of recurrences of ventricular arrhythmias. Other options used for the same purpose are antiarrhythmic drugs and antiarrhythmic surgery, while an implantable cardioverter-defibrillator (ICD) terminates ventricular arrhythmias and prevents sudden cardiac death. Class Ⅲ antiarrhythmic agents, such as amiodarone (especially in combination with β-blockers) or sotalol, can be used to reduce recurrences of ventricular arrhythmias and, therefore, appropriate ICD interventions[1,2]. However, their use is associated with a significant risk of detrimental adverse effects, including proarrhythmia. Moreover, in patients with structural heart disease and life-threatening ventricular arrhythmias, antiarrhythmic drug therapy, used alone, is inferior to ICD for secondary prevention of sudden death[3]. Mapping-guided antiarrhythmic surgery is still a valuable option in abolishing ventricular arrhythmias, which can be used especially in patients who require concomitant cardiac surgery. However, surgical treatment

achieves the best results only in a selected patient population, implies open-chest intervention and requires an experienced center/surgeon[4]. Finally, although undoubtedly valuable to decrease arrhythmic and all-cause mortality, ICDs represent a suppressive therapy and do not prevent recurrences of ventricular arrhythmias. Repeated shocks for the termination of multiple episodes of ventricular arrhythmias may affect both quality of life[5] and survival[6], while inappropriate shocks for supraventricular arrhythmias represent a clinical problem, especially in patients receiving an ICD for primary prevention[7].

In this scenario, a single therapy might not be the best option for every patient. Hybrid treatments including more than one therapy, far from representing an overtreatment, are the best strategy to improve both survival and quality of life in the cohort of patients with structural heart disease and ventricular arrhythmias. Catheter ablation is currently an effective treatment to abolish or minimize recurrences of ventricular arrhythmias and ICD interventions. New techniques and sophisticated technologies have contributed to improve acute and mid-term outcomes of catheter ablation. Peculiarly, it becomes of crucial importance in the case of storms of intractable VTs, when all the other options are ineffective or may not be applicable.

## CATHETER ABLATION OF VT ASSOCIATED WITH STRUCTURAL HEART DISEASE: METHODS AND RESULTS

### *Pathophysiological considerations*

In patients with structural heart disease, the VT can be macroreentrant or focal. If focal in origin, the arrhythmogenic mechanisms can be either enhanced automaticity or macroreentry. In the vast majority of cases, VT associated with large areas of fibrosis/necrosis surrounded by a border zone of slow conduction is sustained by a macroreentrant mechanism[8,9]. This is the case in ischemic cardiomyopathy with an old myocardial infarction, arrhythmogenic right ventricular dysplasia, and some forms of dilated cardiomyopathy. In other cardiomyopathies, an arrhythmogenic focus able to generate and sustain VT is located in a discrete area of the ventricular myocardium in strict anatomical relationship with an area of minimal, moderate, or extensive fibrosis[10]. The mechanism of the VT is clarified in the diagnostic phase of the procedure and determines the electrophysiologic criteria used to identify the ablation target. Some other variables, such as VT inducibility by ventricular stimulation and hemodynamic tolerance, determine the approach for catheter ablation of VT in a given patient.

### *Methods*

Figure 1 shows the different approaches for VT ablation. The presence of left ventricular or atrial[11] thrombi and of inducible myocardial ischemia should be ruled out before the procedure. The left ventricle can be approached transaortically or transmitrally, after transseptal catheterization has been performed. This second option is preferable when critical peripheral vasculopathy or aortic disease is found and necessary when a prosthetic mechanical aortic valve is present. As mentioned before, the major determinants of the ablation strategy are arrhythmia inducibility by programmed electrical stimulation and hemodynamic tolerance. The latter very much depends on ventricular rate during tachycardia and the degree of left ventricular impairment.

**Table 1 Diseases frequently associated with sustained ventricular tachycardia**

| |
|---|
| Ischemic heart disease |
| Dilated cardiomyopathy |
| Hypertrophic cardiomyopathy |
| Arrhythmogenic right ventricular dysplasia |
| Hypertensive heart disease |
| Congenital heart disease (with or without prior surgical correction) |
| Noncompaction of ventricular myocardium |
| Sarcoidosis |
| Systemic sclerosis |
| Chagas disease |
| Myotonic dystrophy type I |

If a VT is easily inducible, hemodynamically well tolerated and shows a stable morphology (left hand side of Figure 1), then detailed activation mapping during tachycardia can be performed to clarify the arrhythmogenic mechanism. In the case of macroreentrant VTs[12], the attention is focused on identification of the critical isthmus of slow mid-diastolic conduction with the help of entrainment techniques[9]. In this area, low-amplitude, fragmented, long-lasting bipolar signals, which are a clear expression of slow conduction, are usually recorded. Ablation in this area is aimed at abatement of electrical signals and VT termination and, eventually, at conduction block over the critical isthmus. This procedure endpoint is a prerequisite to minimize recurrences of the same VT morphology during follow-up. Three-dimensional electroanatomic mapping precisely visualizes the reentrant circuit and, in particular, the critical isthmus of slow conduction[12-14]. Figure 2 shows an example of activation mapping using an electroanatomic system to reconstruct the circuit of a macroreentrant VT and identify the ablation target. A particular form of macroreentrant VT is represented by the bundle branch reentrant VT, which results from macroreentry within the bundle branches. This tachycardia occurs more frequently in patients with non-ischemic dilated cardiomyopathy in the presence of retrograde conduction delay over the left bundle branch. The easiest way to abolish this VT is radiofrequency ablation of the right bundle branch[15].

When the VT mechanism is focal, usually a centrifugally spreading activation pattern from the site of earliest activation is observed in the map generated by the electroanatomic system. Here, the earliest bipolar signal is recorded and, concomitantly, a fast negative intrinsicoid deflection is also recorded in the unipolar recording from

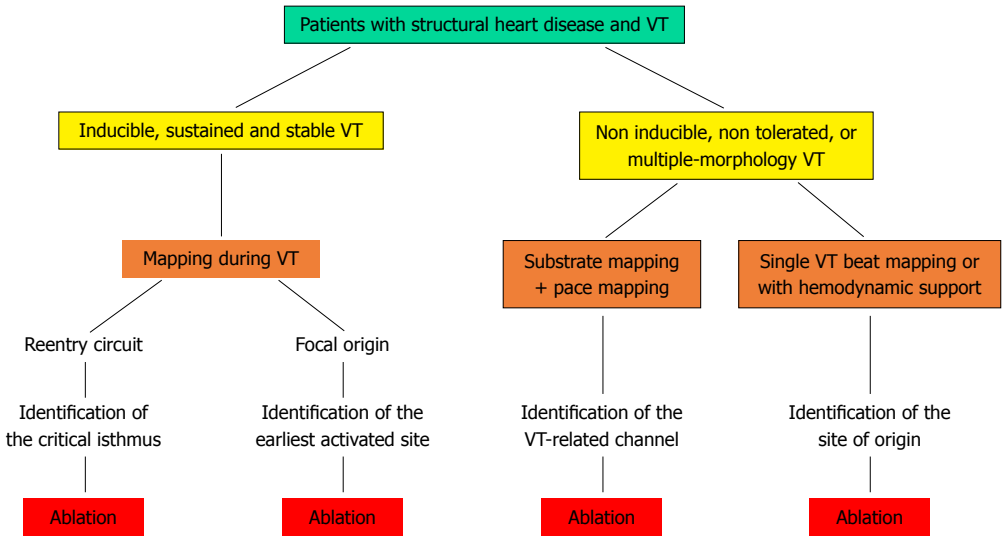


**Figure 1 Strategies for catheter ablation of ventricular tachycardia.** Flow-chart of the possible strategies for catheter ablation of ventricular tachycardia (see text for further explanation). VT: Ventricular tachycardia.

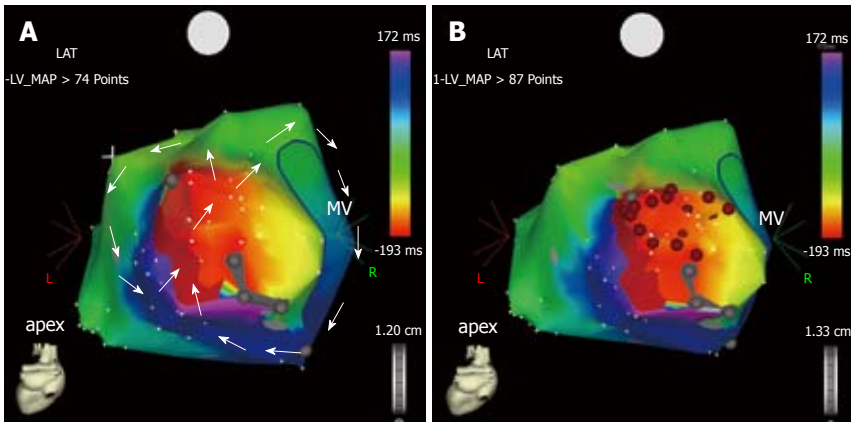


**Figure 2 Three-dimensional electroanatomic map during macroreentrant ventricular tachycardia (A, B).** Postero-anterior view of the electroanatomic activation map of the left ventricular endocardium, reconstructed during ventricular tachycardia with a cycle length of 380 ms in a patient with postinfarction ischemic cardiomyopathy. A: The ventricular tachycardia is sustained by a reentry circuit, which has been fully reconstructed during point-by-point mapping. The reentry course is shown by the sequence of colors from red to purple (arrows). There are two reentrant loops: one rotates clockwise around the mitral annulus, whereas the other has a counter-clockwise course in the lateral wall of the left ventricle. Both loops share the mid-diastolic isthmus, the dark red area between two electrically silent areas (gray dots) where the two arrowed circles meet each other; B: Sequential radiofrequency energy applications (red dots) were delivered linearly to transect the critical isthmus and produce a line of block between the two electrically silent areas and the mitral annulus. No arrhythmia was inducible at the end of the procedure and the patient had no recurrence at the mid-term follow-up. MV: Mitral valve.

the distal electrode of the mapping catheter. Ablation of this area usually results in early tachycardia termination. If the VT is subsequently no longer inducible then the procedure endpoint is met[16,17]. A rare but interesting form of focal VT has been described in patients with postinfarction ischemic cardiomyopathy[18,19]. In this form, the arrhythmogenic focus originates from surviving Purkinje fibers at the border zone of myocardial necrosis. Usually, the VT shows a relatively narrow QRS complex with right bundle branch block morphology and superior axis deviation and a heart rate of approximately 150 beats per minute. Ablation of the earliest activated site, where a high frequency Purkinje potential is recorded, results in permanent abolition of the VT, without the appearance of new conduction disturbances.

The approach based on activation mapping during VT is undoubtedly effective and time-tested. It has been used since the early phase of the experience in catheter ablation of VT[20,21]. Nevertheless, this approach has clear limitations. It can be applied only to a minority of patients with inducible and stable VTs, which has been estimated to account for no more than 30% of patients referred for recurrent VTs[20,22]. In some patients, even if recurrent VTs are clinically observed before the procedure, no arrhythmia is inducible during the procedure. Moreover, several patients have untolerated VTs, which trigger frequent ICD interventions. Finally, in particular cases, multiple morphologies of stable VT are observed and the arrhythmia may convert from one morphology to another during mapping. All these situations make it

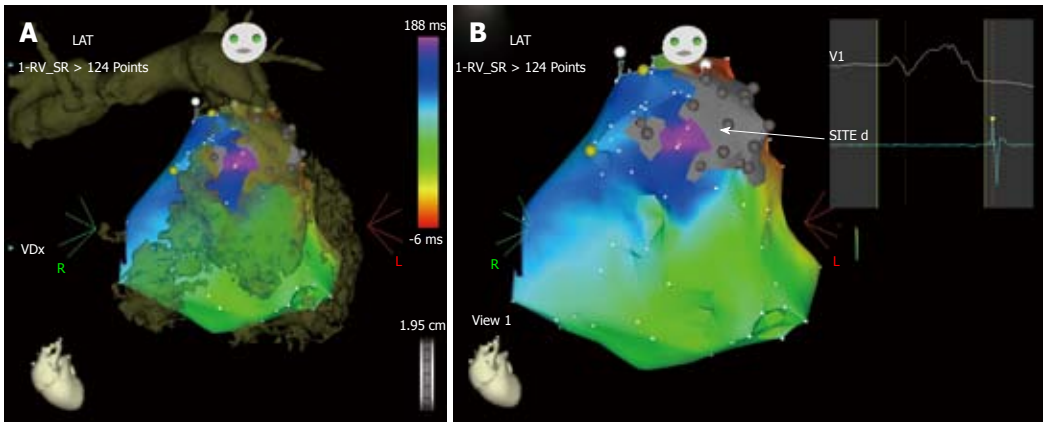


**Figure 3 Imaging integration for ablation of ventricular tachycardia (A, B).** Example of integration of a computed tomography image in the three-dimensional electroanatomic system. This patient had ventricular tachycardia years after multiple surgical corrections for tetralogy of fallot with pulmonary artery stenosis. He was referred for multiple recurrences of drug-refractory ventricular tachycardia with two morphologies, both with left bundle branch morphology and inferior axis deviation. However, no arrhythmia was inducible during the electrophysiology testing. Therefore, the ablation strategy was based on substrate mapping in sinus rhythm with imaging integration (Figure 4). A: The three-dimensional rendering of the computed tomography (image in brown) of the right ventricle and pulmonary artery is superimposed on the electroanatomic mapping of the right ventricle in sinus rhythm. The merging of the two images helps reconstruct the geometry of the heart chamber and, particularly in this case, clarifies the anatomy of the right ventricular outflow tract, identifying precisely the pulmonary valve annulus, an essential landmark for ablation. The right ventricle appears hypertrophic and markedly dilated; B: The activation map in sinus rhythm is shown in the antero-posterior projection without the integrated image. There is a markedly delayed activation in the anterior wall of the outflow tract (in purple), in a channel between two electrically silent areas (in gray) related to previous surgery (ventriculotomy and positioning of a prosthetic patch). This channel (arrow) shows late bipolar recordings, after the end of the surface QRS (panel on right hand side) and could possibly be related to one morphology of ventricular tachycardia. During sinus rhythm, the earliest activated site (in red) of the right ventricular endocardium is the ventricular septum, consistent with a breakthrough of activation from the left bundle branch in the presence of a complete right bundle branch block.

difficult or even impossible to use a strategy based on activation mapping to identify the ablation target. Consequently, over the years, alternative approaches to activation and entrainment mapping have been developed to treat these "unmappable" forms using catheter ablation. This has moved ahead the frontier of catheter ablation of VT, expanding this treatment option to a wider patient population. As shown in the right hand side of Figure 1, these strategies are necessarily based on the use of new technologies, such as three-dimensional mapping systems.

In "unmappable" VTs, the approach most frequently used is substrate mapping during sinus rhythm or right ventricular pacing combined with pace-mapping techniques[23-25]. This method aims at localizing the substrate for possible reentry circuits based on identification of areas of low voltage and slow conduction, assuming that the clinical "unmappable" arrhythmia is sustained by reentry. Using a three-dimensional electroanatomic mapping system, bipolar voltage mapping is performed in the ventricles to identify areas of low voltage related to an old myocardial infarction or, in general, fibrosis. According to a preliminary evaluation[23], areas with bipolar voltage $\geqslant$ 1.5 mV are normal, those with bipolar voltage $\leqslant$ 0.5 mV are areas of dense scar, whereas areas with a voltage between 0.5 and 1.5 mV are the border zone between necrosis/fibrosis and the normal myocardial tissue. In a post-mortem study, these voltage thresholds correlated very well with histology, since massive (> 80%) fibrosis has been found in areas with voltage < 0.5 mV, while intermediate (21%-79%) and minimal (< 20%) fibrosis has been observed in areas with voltage 0.5-1.5 mV and > 1.5 mV, respectively[26]. Once voltage mapping has been completed, pacing maneuvers are used in the area of dense scar and along the border zone to identify a channel of low voltage and slow conduction, possibly related to the VT. In fact, this may serve as the critical diastolic isthmus of the reentry circuit. Ventricular pacing is finalized to: (1) reproduce a QRS complex morphology identical or similar to the target VT; and (2) identify an area of slow conduction as assessed by a long interval between the pacing stimulus and the paced QRS complex. In the majority of cases, a slow conducting channel related to a VT can be identified in a discrete area, so that limited ablation can be performed to transect this channel[24]. An example of a non inducible VT ablated using this strategy is shown in Figures 3 and 4. These VT-related channels are usually found in the area of dense scars with a bipolar voltage of 0.1-0.3 mV and a length of 33 ± 22 mm, on average[24,27]. Ablation aims to render unexcitable the identified VT-related channel. After ablation, stimulation protocols are used to assess the non inducibility of VT. Although every effort is made to ablate a limited area, this strategy may be more aggressive than the one based on activation mapping during a stable VT, since in some cases this technique may overestimate the ablation target. However, this does not result in worsening of ventricular function, because ablation is performed in an area of massive fibrosis, which does not contribute to ventricular contraction. In fact, a study[28] showed that the value of left ventricular ejection fraction is unchanged before and after ablation, even if a considerable amount of radiofrequency energy was applied (25 applications on average, ranging from 3 to 98). Finally, this strategy, proposed to approach "unmappable" ventricular arrhythmias both in

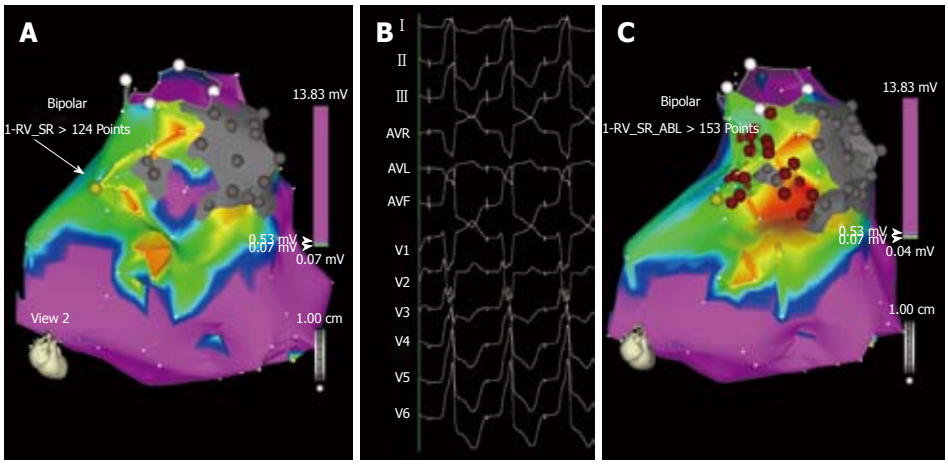


**Figure 4 Ablation of "unmappable" ventricular tachycardia based on substrate mapping and pace-mapping (A, B).** Bipolar voltage mapping of the right ventricle in the same patient as in Figure 3. A: According to the settings used for this map, shown in antero-posterior view, the purple area has a bipolar voltage > 0.53 mV, while in the myocardium surrounding the two scars (in gray) the voltage is low, between 0.05 and 0.52 mV (colors from red to blue). In this area, pace-mapping is used to identify channels of low voltage and slow conduction possibly related to the two ventricular tachycardia morphologies clinically documented. In addition to the channel of slow conduction identified between the two scars during sinus rhythm (Figure 3), a second channel of low voltage and slow conduction is now identified by pacing at 600 ms cycle length in the area marked by the yellow dot (arrow); B: In this site, pacing reproduces one of the two ventricular tachycardia morphologies. Interestingly, the interval between the stimulus artifact and the onset of the QRS complex is markedly prolonged (125 ms) and this demonstrates the presence of slow conduction in this area; C: The same map is now shown in right anterior oblique view with two lines of radiofrequency energy applications (red dots) delivered to transect the two channels: one line is deployed between the two scars and the other between one scar and the pulmonary artery annulus (circle marked by white dots). No arrhythmia was inducible at the end of the procedure and the patient had no recurrences during follow-up.

ischemic and non-ischemic cardiomyopathy, may have some limitations in the non-ischemic cardiomyopathy setting. In these cases, the distribution of the areas of fibrosis identified by voltage and slow conduction shows a patchy pattern and fewer protected channels/isthmi, definitely different from a postinfarction scar. This undermines the feasibility and efficacy of this approach in non-ischemic cardiomyopathy patients[29,30].

As reported in Figure 1, activation mapping of only a few tachycardia beats using noncontact mapping[31-33] or using a ventricular assist device for temporary hemodynamic support[34] is another possible, but rarely used, strategy to approach "unmappable" VT. Using a multi-electrode array mounted on an inflatable balloon, noncontact mapping reconstructs isopotential activation maps of a single VT beat[31]. Analysis of the map allows identification of the arrhythmogenic substrate, which, subsequently, can be ablated. Suppression of arrhythmia inducibility correlates with a favorable outcome during follow-up[31-33]. The limitation of this approach mainly consists of possible sub-optimal identification of the diastolic pathway in the reentry circuit. Especially in cases with enlarged heart chambers, noncontact mapping may be unable to detect low amplitude potentials, which are typically found in the critical isthmus of slow conduction. When this isthmus is not localized, ablation in the exit site from the critical isthmus where higher voltage is present is a possible alternative, but usually less effective[31].

As previously mentioned, activation and entrainment mapping to localize the arrhythmogenic substrate of untolerated VT is also possible using a ventricular assist device[34]. Currently, limited experience has been gathered in this field. However, this approach is feasible. The left ventricular assist device is percutaneously positioned before the procedure and is able to maintain a cardiac output during the VT for enough time to identify the arrhythmogenic substrate. Nevertheless, it is important to underline that some patients may require continuation of ventricular assistance after the procedure for a prolonged time period. Therefore, these cases should be managed in cooperation with a heart failure specialist and a cardiothoracic surgeon[34].

Finally, frequently recurrent very fast VT and even ventricular fibrillation can be completely or partially suppressed by catheter ablation, if their recurrence is invariably triggered by a specific ventricular ectopy. Both in patients with ischemic heart disease and in patients apparently without structural heart disease[35,36], when the origin of the triggering premature ventricular beat can be localized, ablation of this area results in long lasting suppression of the fast VT or fibrillation, minimizing ICD interventions during follow-up.

## Results

It may be complex to evaluate the results of catheter ablation of VT in patients with structural heart disease. The reasons are multiple: (1) there is still a paucity of data and the published studies include a limited number of cases with non uniform underlying heart disease and, usually, only short- or mid-term follow-up data; (2) the clinical outcome in terms of prevention of VT recurrences may differ from the success of the procedure, defined as acute suppression of the inducible VT; long-term clinical outcome may also depend on the type of underlying heart disease; (3) new techniques and technologies have been introduced and applied over the last 10 years; there-

fore, early studies may underestimate the results now achievable, while the latest studies might overestimate the results because of the limited follow-up; (4) a given technique (e.g., ablation based on substrate mapping) can achieve good results in a subset of patients (ischemic cardiomyopathy), but sub-optimal results in other patients (non-ischemic cardiomyopathies); (5) single-center studies involving experienced operators may report better results, but in a limited patient population and for multicenter studies the opposite may be true; and (6) currently, meta-analyses include a limited number of studies with inhomogeneous patient populations and different ablation techniques with limited data on mortality. In the following section, data on the largest and most significant studies on VT ablation will be reported, including single-center studies, multicenter observational studies and prospective multicenter randomized trials. It would take too long to report detailed results of each subset of patients with different heart diseases. These data have been extensively reported in two recently published consensus documents[37,38].

In general, catheter ablation is effective in terminating a target morphology of VT and limiting arrhythmia recurrences. Acute suppression of VTs by radiofrequency energy application has been reported in 75% to 95% of patients, with a recurrence rate of up to 35%[37,38]. Although the recurrence rate seems high, it is much lower than in patients receiving only antiarrhythmic drugs, according to a recent meta-analysis[39]. This meta-analysis considered 154 potential studies on catheter ablation of VT associated with structural heart disease and included only 5. These studies spanned 11 years with a total of 457 patients. When catheter ablation used as adjunctive therapy to antiarrhythmic drugs was compared to antiarrhythmic drugs only, there was a statistically significant 38% reduction in the number of patients with VT recurrences in the group undergoing catheter ablation. The difference in mortality between the two groups could not be demonstrated in this meta-analysis.

Catheter ablation of VT associated with structural heart disease has been a promising therapeutic option since the beginning. In the early phase, two studies[20,21] reported encouraging results on VT ablation in patients with structural heart disease (mainly ischemic cardiomyopathy), based on activation and entrainment mapping. The success rate was 73%-83%, recurrence rate about 25%, with a complication rate of 7% and limited use of ICD (20% of patients). These results seem surprisingly good considering that at that time the technology of three-dimensional mapping and irrigated tip ablation, now routinely used, was not available. The selection of patients and prior experience in antiarrhythmic surgery for VT in these centers could have been crucial in achieving good results.

In single center reports, ablation based on the substrate mapping strategy in patients with "unmappable" VT have produced similar results in terms of recurrence rate during a short/mid-[23,24,40,41] and long-[42] term follow-up. Although the percentage of patients with recurrences varied between 17% and 36% in these studies, a considerable reduction in the frequency of ICD interventions was observed post-ablation. In a single center non-randomized study[43], substrate mapping during sinus rhythm and activation mapping during VT was compared in patients with coronary artery disease. The results of the two strategies in term of clinical outcome were similar: success rate during follow-up was 80% and 71% for the activation and the substrate mapping groups, respectively. However, as previously mentioned, the results achieved using substrate mapping for ablation of "unmappable" VT very much depended on the type of underlying heart disease. In fact, the success rate of ablation based on substrate mapping in terms of prevention of recurrences during follow-up was 82% and only 50% in ischemic and non-ischemic cardiomyopathy patients, respectively[30]. The poorer clinical outcome in the latter group of patients mainly depended on the different characteristics of the arrhythmogenic substrate in patients with non-ischemic cardiomyopathy, which is more difficult to localize by substrate mapping.

Recently, two prospective multicenter observational studies have been published. They report the results on catheter ablation of postinfarction VT using electroanatomic three-dimensional mapping and irrigated-tip radiofrequency energy ablation[44,45]. In the first study[44], 231 patients in 18 centers over a period of approximately 4 years were enrolled. They had a median left ventricular ejection fraction of 25% and a median of 11 VT episodes in the last 6 mo. The prevalence of heart failure and atrial fibrillation was 62% and 29%, respectively. Before ablation, amiodarone failed in 70% of patients and 94% of them had an ICD. During the ablation procedure, a median of 3 VT morphologies per patient were treated for a total of 864 morphologies ("unmappable" morphologies were present in 69% of patients). The primary end-point (freedom of VT in the following 6 mo) was reached in 53% of patients and a reduction in the frequency of VT recurrence of at least 75% was obtained in 67% of patients. Procedure-related mortality was 3% with an additional 7.3% of non fatal complications. The second study[45] achieved similar results in a smaller patient population. In 8 European centers during approximately 3 years, 63 patients underwent ablation of 164 mappable or "unmappable" VT morphologies. Two thirds of the patients had an ICD before ablation. Success was obtained in 81%, but 49% of patients had VT recurrences during follow-up. However, 79% of patients with recurrences had a significant reduction in episodes from 60 ± 70 prior to ablation to 14 ± 15 after ablation, during the same time period. Procedure-related complications were observed in 7.9% of patients. The results of these two studies suggest that suppression of postinfarction VT can be reproducibly obtained in different centers by catheter ablation during the procedure and results in a significant reduction of arrhythmia episodes during follow-up. It is important to take into account that these

studies included severely ill patients, with multiple and also unmappable tachycardia morphologies refractory to amiodarone. Considering the time period in which these studies were carried out (between 1999 and 2003), these results may possibly improve in the near future.

Two other prospective multicenter randomized trials were designed to assess the adjunctive benefit of catheter ablation in patients with previous myocardial infarction who received an ICD for secondary prevention of VT[46,47]. In both studies, enrolled patients were 1:1 randomly assigned to receive only an ICD or catheter ablation and an ICD. In the first study[46], named SMASH-VT, ablation was based on substrate mapping and the primary end-point was survival free from any appropriate ICD therapy. Over 46 mo, 128 patients with both mappable and "unmappable" VT were enrolled in three centers and followed up for 22 ± 5 mo. In the group of patients who received ablation + ICD, survival free from ICD therapy was significantly higher than that in the group who received only ICD and, interestingly, mortality was not increased in the first group compared to the second group. The second study[47], named VTACH, enrolled 107 patients with postinfarction mappable VT in 16 European centers. The ablation procedure was based on activation or substrate mapping with the use of three-dimensional mapping systems in all cases. During a mean follow-up of 22 ± 9 mo, time to recurrence of VT or VF, the primary end-point, was longer in the ablation + ICD group than in the ICD only group, with no deaths within 30 d of the procedure. Interestingly, at 2 years, survival free from VT or VF was better in the ablation group as compared to the control group. Moreover, the benefit of ablation was mainly observed in the group of patients with an ejection fraction between 30% and 50%, in which the difference in survival between the two groups reached high statistical significance. These two randomized studies confirm that, in patients with postinfarction mappable or "unmappable" VTs and impaired left ventricular function, early ablation based on activation or substrate mapping, far from being overtreatment, significantly reduces the appropriate ICD interventions with no impact on mortality related to the ablation procedure.

### Catheter ablation of arrhythmic storms

Electrical storms are frightening events, characterized by multiple episodes in a relatively short time of intractable ventricular arrhythmias resulting in multiple appropriate ICD shocks with poor short- and long-term prognosis. Multiple reports highlight the crucial role of catheter ablation to control these arrhythmic storms and restore stable sinus rhythm[48-51]. Electrical storms usually occur in very sick patients with poor left ventricular function (ejection fraction < 30%), multiple co-morbidities, multiple morphologies of VT with heart rate of approximately 160 beats per minute. In some cases, these patients require immediate attention and the catheter ablation procedure is performed within the first 24 h after hospital admission[51]. During the same admission, multiple procedures (up to 3) may be necessary to control the electrical storm[48]. Electrical storms can be suppressed in 84%-100% of cases and during a mid-term follow-up the percentage of patients free from electrical storms and from any VT was 74%-94% and 48%-69%[48-51], respectively. Interestingly, the worst results in term of suppression of the electrical storm and, in general, of VT recurrences were obtained in patients presenting with an electrical storm associated with cardiogenic shock, despite the use in this subset of patients of the most sophisticated techniques and technologies[48]. Occasionally, inability to terminate the clinical incessant VT may result in electromechanical dissociation and death during the ablation procedure[51]. After the procedure, recurrence of electrical storm may require drastic countermeasures, such as implantation of ventricular assist device as a bridge to heart transplantation[50]. During follow-up, the mortality rate can be as high as 30%[49,50] and is mainly due to progression of heart failure. Low ejection fraction, increased left ventricular end-diastolic diameter and renal insufficiency are predictors of death[50].

**Table 2 Common complications related to catheter ablation of ventricular tachycardia**

| |
|---|
| Peripheral vascular injuries |
| Thromboembolic events |
| Pericardial effusion |
| Cardiac perforation and tamponade |
| Injuries to valve and subvalvular apparatus |
| Atrioventricular or bundle branch block |
| Injuries to coronary arteries and myocardial ischemia |
| New onset ventricular arrhythmias |
| Cardiogenic shock |
| Death |

### Complications

In several studies on catheter ablation of VT, the reported complication rate was about 7%, higher than that for ablation of supraventricular arrhythmias. This is mainly due to the associated severe underlying heart disease in many of these patients. Major and minor complications related to VT ablation are reported in Table 2. Importantly, procedure-related death has a non negligible prevalence, accounting for up to 3%[44]. In these patients, death is mainly related to the occurrence of uncontrollable fast VT leading to irreversible cardiogenic shock and cardiac arrest. Death can occur during or soon after the ablation procedure. Irreversible cardiogenic shock may also be observed after successful ablation for a long-lasting incessant VT, when the patient is in stable sinus rhythm and is related to the end-stage condition of the patient[20]. Clinical experience is required to select appropriate candidates for the procedure and to avoid ablation in those who are too sick to benefit. Moreover, expertise and team work with a heart failure specialist is needed to manage patients with critically depressed left ventricular function before, during and after the procedure.

## NEW TECHNIQUES AND TECHNOLOGIES FOR CATHETER ABLATION OF VT

As previously mentioned the best results for catheter ablation of VT associated with structural heart disease are obtained using three-dimensional mapping and irrigated tip ablation. The first technology allows accurate characterization of the arrhythmogenic substrate during activation and/or substrate mapping, while the second is of crucial importance in delivering radiofrequency energy in an efficient and safe way and to abolish the arrhythmogenic substrate. Currently, these technologies are routinely used in the vast majority of centers. Over the last decade, new techniques and technologies have been developed and introduced in clinical practice. They have proved effective in improving procedure parameters and/or clinical outcomes and can be particularly useful in the management of cases refractory to standard ablation.

### *Epicardial approach*

Percutaneous access to the pericardial space using a regular vascular sheath was originally described by Sosa[52]. Alternatively, the epicardial space can be accessed using a minimally invasive subxiphoid surgical approach[53,54]. An epicardial or sub-epicardial arrhythmogenic substrate is expected in approximately 10% and 30% of cases with ischemic and non-ischemic cardiomyopathies, respectively[55]. In a multicenter study, epicardial ablation was performed in 13% of patients undergoing VT ablation[56]. An epicardial origin of VT should be suspected when the QRS morphology shows a delta wave and/or a prolonged intrinsicoid deflection. Recently, it has been reported that in patients with a left ventricular cardiomyopathy, endocardial unipolar voltage mapping is able to identify a possible epicardial arrhythmogenic substrate for the greater field of view of the minimally filtered unipolar recordings as compared to bipolar recordings[57]. Epicardial access is clearly indicated when an endocardial approach including transvenous epicardial mapping through the coronary sinus and its sub-branches has failed. Percutaneous access to pericardial space is obtained usually under general anesthesia using a needle designed for epidural access. After positioning of a regular vascular sheath, an ablation catheter can be inserted and manipulated to map the epicardial surface of the heart. Irrigated-tip ablation is usually preferred, but this requires periodic aspiration of the saline used for irrigation, because it accumulates in the pericardial space. Epicardial fat, detected during mapping as a low voltage area, may affect the quality of mapping data and, at the same time, prevent radiofrequency energy delivery directly to the epicardial surface. In the case of intramural arrhythmogenic substrate, embedded deep in the layers of the left ventricular wall, a combined epi- and endocardial ablation may be required. Three publications focusing on epicardial access for VT ablation report data on a total of 247 epicardial procedures[56,58,59]. Success in abolishing VT was obtained in 76%-78%, and recurrences during follow-up were observed in 26%-47% of cases. Complications related to epicardial access were pericarditis and right ventricular puncture with pericardial bleeding, while complications related to epicardial ablation were phrenic nerve injury and coronary artery occlusion. To avoid these latter complications, it is mandatory to localize the phrenic nerves by pacing and coronary arteries by angiography before radiofrequency energy is delivered. It is important to underline that in these two studies[56,59] in roughly one fourth of cases undergoing or referred for an epicardial procedure, ablation was performed only at an endocardial site because it was eventually considered the most suitable site for ablation. For this reason, and due to the difficulties that can be encountered in accessing the pericardial space, and the complications including death[58,59] that may be observed, the need for an epicardial procedure should be carefully evaluated after failure of the endocardial approach.

### *Imaging integration*

During the procedure, a pre-acquired three-dimensional rendering of a computed tomography or magnetic resonance scan of the heart can be imported in the electroanatomic system[60]. Once the three-dimensional image is correctly integrated, it is possible to navigate the mapping/ablation catheter in the high resolution image of a given heart chamber. In this way, complete reconstruction of the ventricular chamber is facilitated during activation or substrate mapping[61].

Integration of a computed tomography image is particularly useful, because it shows the course of the coronary artery, the size of the ventricular chamber and strategic landmarks. Visualization of the coronary vessels is necessary when an epicardial approach is performed and helps avoid ablation close to a coronary artery branch, which may result in acute myocardial ischemia[62-64]. In patients with peculiar anatomy, such as patients with multiple prior surgical intervention for correction of a congenital heart disease or with large aneurysms[64], a pre-acquired computed tomography image integrated into the electroanatomic map helps orient mapping. Figure 3 shows imaging integration of a computed tomography scan of the right ventricle in a case of post-Fallot VT. During substrate mapping in sinus rhythm, the computed tomography image guided reconstruction of the right ventricular outflow tract and allowed correct identification of the plane of the pulmonary valve, an essential landmark in the approach of this type of VT. This also facilitated localization of the channels of slow conduction related to the VT.

Delayed gadolinium-enhancement magnetic imaging identifies areas of fibrosis in the ventricular myocardium in patients with structural heart disease. When integrated in the electroanatomic systems, it provides topography and transmural extent of the fibrotic tissue and, hence, of the low voltage areas, simplifying the identification of the arrhythmogenic substrate during voltage mapping in sinus rhythm[65,66]. This is expected to significantly shorten mapping time, increase the success rate and reduce the

complication rate. In general, ICD and claustrophobia are considered contraindications for magnetic resonance imaging. However, using appropriate precautions this imaging modality is possible even in ICD patients, who represent a vast proportion of candidates for VT ablation[38,66].

### Remote magnetic navigation

A sophisticated device which consists of two magnets on each side of the patient's torso makes it possible to maneuver a special catheter with magnetic sensors by changing the direction of the magnetic field around the patient's chest[67]. The ablation/mapping catheter is maneuvered by the operator *via* a computerized system, which can be located remote from the patient's bed, to avoid radiation exposure for the operator. One of the advantages of this system is the peculiar flexibility of the catheter, which therefore can be magnetically guided and stably positioned in sites difficult for standard catheters, such as the coronary cusps and the right ventricular outflow tract[68,69]. Single center studies show that in VT patients, endocardial and epicardial mapping and ablation using this system is safe and feasible with minimal radiation exposure for the patient[70,71]. It has been recently reported that remote magnetic navigation may be more effective than manual ablation[72]. On the other hand, the disadvantages of this system are mainly represented by costs and the need for appropriate location and magnetic shielding.

### Alternative energy sources

As previously mentioned, irrigated tip ablation is a standard procedure both for endocardial and epicardial VT ablation. In antiarrhythmic surgery for VT, cryothermal energy was used to complement endocardial resection[4]. When applied percutaneously, cryothermal energy produces a more discrete lesion with less collateral damage to adjacent structures as compared to radiofrequency energy. Percutaneous cryoablation of VT associated with postinfarction ischemic cardiomyopathy has proved effective and safe, although a higher recurrence rate may be encountered, probably related to the smaller lesion size[73].

Ethanol injection in a coronary artery branch was proposed more than two decades ago to produce a controlled necrosis of the ventricular myocardium where the VT originates. This option can be used today[74], but it should be limited to very selected cases refractory to other approaches due to the high risk of complications including reflux of ethanol in other coronary branches, which results in a large necrosis of the ventricular myocardium.

Currently, ultrasound, laser, and radiation energy are under investigation to evaluate whether these energy sources can produce a larger lesion in a safe way, in order to treat patients refractory to conventional ablation. Gene therapy to modify the electrical properties of the arrhythmogenic substrate to prevent ventricular arrhythmias is currently under investigation, however, we are still far from having a clinical application[75].

## CONCLUSION

Catheter ablation is effective and safe in abolishing recurrent VTs associated with structural heart disease, although it may not be a curative and stand-alone therapy. New techniques and technologies introduced over the last decade have made ablation possible even in cases of "unmappable" VT and, in general, they have improved the success rate of VT ablation. The complication rate, including death, is significantly higher than that for supraventricular arrhythmias. This is mainly due to the severity of the underlying heart disease and clinical conditions before the ablation procedure. Finally, in patients with a structural heart disease, VT ablation should not be the last resort after a long history of multiple ICD shocks despite high-dose amiodarone. Recent data show that the VT-free survival is significantly higher in patients who are referred early for ablation of recurrent VTs[76].

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