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REVIEW

Catheter Ablation of Ventricular Tachycardia

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ABSTRACT

Catheter ablation is highly successful and may be considered as the first line treatment in all symptomatic idiopathic forms of ventricular tachycardia (VT). Also ablation plays an important role in recurrent VTs associated with structural heart disease and relatively preserved left ventricular ejection fraction (>35%) and/or bundle branch reentry VT. It also constitutes the preferable treatment modality in incessant VTs of any origin and in patients with implantable defibrillator (ICD) devices who present with recurrent VTs and/or an electrical storm leading to multiple ICD shocks

Catheter ablation appears to improve arrhythmia control in about two thirds of patients with structural heart disease and mappable VTs. Novel substrate and/or non-contact mapping techniques suggest that even hemodynamically unstable VTs and/or VTs of multiple morphologies can be successfully ablated. As the ablation method is not curative and there remains the risk of dying suddenly in patients with depressed left ventricular ejection fraction, the majority of patients with VT associated with structural heart disease also receive an ICD.

KEY WORDS: *ventricular tachycardia; radiofrequency catheter ablation; electroanatomical mapping; electrophysiology study; implantable cardioverter defibrillator*

INTRODUCTION

Management of ventricular tachycardias (VTs) remains a continuing challenge even in the era of implantable cardioverter-defibrillators (ICDs). Although successful in terminating VT, these devices do not prevent episodes of arrhythmia. In respect, catheter ablation offers a substantial potential to control and cure VT.

Based on ECG morphology, VTs are described either polymorphic or monomorphic.[1] Until recently, only monomorphic VTs have been a subject to catheter ablation. Our group reported probably one of the first cases of successful catheter ablation of focus triggering polymorphic VT and/or ventricular fibrillation.[2] More recently, this approach has been described in a collaborative report by Haissaguerre et al.[3] Triggering foci originate often from Purkinje fibers of the left ventricular septum and can be identified as monotopic ventricular ectopy, usually with a short coupling interval. Monomorphic VTs are characterized by a constant morphology of the QRS complex, indicating the same sequence of ventricular activation.

Arrhythmia may originate either from a focal source or be a result of reentrant activation within the circuit, usually associated with structural substrate.[1] The

ABBREVIATIONS
ICD = implantable cardioverter defibrillator
LBBB = left bundle branch block
RBBB = right bundle branch block
RF = radiofrequency
VT = ventricular tachycardia

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mechanism of focal VTs comprises abnormal automaticity, triggered activity and/or microreentry circuit. Importantly, the QRS morphology suggests the probable arrhythmogenic region. A left bundle branch block (LBBB) configuration in precordial lead V_1 implies an origin in the right ventricle or in the interventricular septum. On the contrary, a right bundle branch block (RBBB) appearance indicates origin in the left ventricle. Inferiorly directed frontal plane axis reflects an origin in the superior aspect of the left ventricle. Superior frontal plane axis suggests origin in the inferior wall of the left or right ventricle. Predominant R waves in precordial lead V_4 favours origin near the base of the heart, while dominant S waves suggest rather apical site.

Ventricular tachycardia occurring in subjects with apparently healthy heart (i.e. without identifiable structural heart disease) is referred to as "idiopathic" and is usually focal in origin. On the other hand, VT associated with myocardial scars is typically re-entrant. Rarely, VT in these patients could be focal in origin. Patients with nonischemic cardiomyopathies are prone to development of bundle-branch reentry VT.

IDIOPATHIC VT

Ventricular tachycardia in subjects without structural heart disease accounts for a minority of all VTs. Prognostically, these arrhythmias are considered benign and catheter ablation is indicated in symptomatic cases. High success rate of catheter ablation reflects focal origin of idiopathic VTs.[4] Despite the fact that the focus can be situated in any region of the ventricles, two locations prevail in clinical arena.

Idiopathic right ventricular outflow tract VT is the most frequent entity among idiopathic VTs. The mechanism of this arrhythmia is most probably triggered activity.[4,5] The site of origin in the outflow tract of the right ventricle leads to a specific ECG pattern of a LBBB with inferior frontal plane axis. This arrhythmia occurs either as a repetitive or paroxysmal VT and is frequently triggered by adrenergic stimulation. Catheter ablation has become first-line therapy in this condition in the majority of cases. It is guided by activation sequence mapping (i.e. searching for the earliest site of activation during tachycardia) and/or by pace-mapping (i.e. reproduction of VT morphology through pacing at the site of origin). The success rate of catheter ablation reaches approximately 85%. Inability to induce the arrhythmia and/or its epicardial or deep septal location usually account for failures. In some patients, catheter ablation could be performed from the pulmonary or aortic cusp and/or from a subaortic region.[6] In such cases, R wave transition occurs in V_1-V_3.

Idiopathic left ventricular fascicular tachycardia originates in calcium channel-dependent tissue of the Purkinje fibers on the interventricular septum.[4,7] Reentry involving the posterior or anterior fascicle is considered the mechanism of this arrhythmia. The site of origin corresponds to QRS morphology. Typically, a RBBB configuration with a superior (or inferior) frontal axis can be recorded. Catheter ablation is successful in approximately 90% of cases and is guided by mapping of a discrete Purkinje potential preceding the QRS complex during VT.[7] Inability to ablate this arrhythmia reflects catheter-caused pressure trauma to the focus.

VT ASSOCIATED WITH STRUCTURAL HEART DISEASE

These arrhythmias are caused by reentry involving a region of ventricular scar of different origin.[1] The most frequent cause of the scar is previous myocardial infarction. Other causes include arrhythmogenic right ventricular dysplasia, nonischemic cardiomyopathies, ventriculotomies after surgery for congenital heart disease, sarscoidosis and others. Dense scars create anatomical barriers and intercellular fibrosis decreases cell-to-cell coupling, resulting in slow conduction. These conditions are known to promote reentry. During VT, the morphology of the QRS complex reflects the location of the exit from the protected isthmus of slow conduction. The timing of the local activation corresponds to the beginning of the QRS complex and the wavefront then propagates through the surrounding myocardium towards the entrance site of the isthmus (outer or inner loop). Configuration of the individual circuit varies based on arrangement of the scar tissue. Often, one isthmus is shared by several circuits with different exits. In such cases, several VT morphologies can occur spontaneously and/or can be induced in the electrophysiology laboratory. In the majority of postinfarction scars, the isthmuses are localized in the subendocardial region and thus, accessible to catheter ablation. Until recently, their identification required detailed mapping during tolerated tachycardia. *Entrainment mapping* was used to confirm participation of the isthmus in the circuit.[8] Provided the pacing site is within the protected isthmus, pacing at cycle length 20-50 ms shorter speeds up VT without any change in QRS morphology (concealed entrainment) and the post-pacing interval (i.e. the first cycle after termination of pacing) equals tachycardia cycle length. At the same time, stimulus to QRS interval during entrainment pacing equals spontaneous interval between local electrogram in mapping catheter and the beginning of the QRS during VT. More recently, the so-called *substrate mapping* can be used to describe morphological substrate for VT in sinus rhythm using an electroanatomical mapping system.[9] Pacing manoeuvres can be used to reconstruct re-entrant circuit, analyzing stimulus-to-QRS intervals in different locations of the presumed isthmus (isthmuses) and looking for perfect or near-perfect match in ECG morphology with ECG during VT. Another alternative for mapping and ablation is non-contact mapping that could be used in unstable VTs to reconstruct the zone of slow conduction during one cycle of arrhythmia.[10] Rarely, arrhythmogenic foci or critical isthmuses are localized epicardially. Therefore, a technique of epicardial mapping and ablation through a direct intrapericardial approach has

been described.[11]

As ablation lesions produced with a standard 4-mm tip catheter are limited in size, irrigated-tip catheters are preferably employed for this purpose.[12] Still, a line of radiofrequency lesions is often necessary across a region of the circuit and/or its exit to ablate successfully all inducible VTs or at least clinical ones. This reflects another problem of inducibility of several different VT morphologies in an individual patient. Spontaneously occurring VTs are then considered as "clinical VTs", while other inducible VTs are referred to as "non-clinical VTs". However, some of the "non-clinical" VTs may recur after successful ablation of a "clinical" VT.

Postinfarction VT

Hemodynamically tolerated VT allows detailed mapping (including entrainment mapping) during arrhythmia and thus, precise description of the reentrant circuit. The success rate of catheter ablation of mappable VT has been reported in the range of 70-75%, with a recurrence rate of 10-40%.[13,14] Others targeted all inducible VTs [16-18] and achieved noninducibility in approximately 30% of patients, and almost 50% of subjects had successful modification of the substrate with noninduciblity of targeted VTs. Almost two thirds of patients were arrhythmia -free during long-term follow up.

The patient with untolerated VT is a particularly difficult problem. The so-called substrate mapping seems to provide a solution. The procedure is based on the ability of electro-anatomical mapping to localize and quantify abnormal electrograms and scar tissue in a 3D reconstruction of endocardial surface of the left or right ventricle.[9,18] The areas of scar are defined as sites with low voltage amplitudes <0.1-0.5 mV, while normal tissue has the voltage of electrograms >1.5 mV. In addition, electrical nonexcitability can be used to identify "dense scars" as areas without pacing capture at 10 mA. This allows identification of relatively narrow isthmuses of slow conduction even within relatively large regions of very low voltage (<0.5 mV). At the same time, pacing at each site allows analysis of 12-lead ECG as well as interval stimulus-to-QRS interval. This approach provides detailed description of the substrate with an exit site and the central zone (Figure 1). Radiofrequency (RF) energy could be applied across the exit region, some target also late potential areas and/or connect areas of dense scars to eliminate all potential conduction channels. Pacing is used again after each RF energy application to assess lesion completeness. Our experience and that of others suggest that these approaches may eliminate all VTs in 60-80 % of cases.[19-21] However, more data from prospective trials are needed.

It remains unknown whether successful catheter ablation of post-infarction VT may result in a complete cure. Patients often have seriously depressed left ventricular function and thus, remain at risk of malignant arrhythmias even after successful catheter ablation of all inducible arrhythmias.

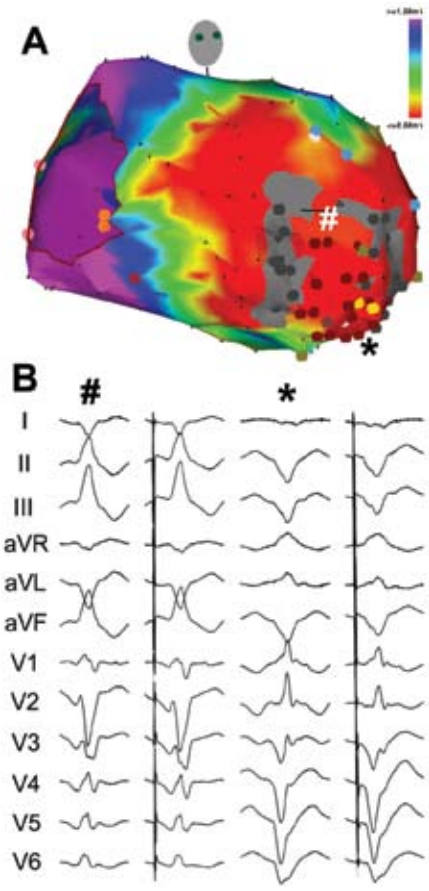

FIGURE 1. An example of a detailed electroanatomical voltage map (CARTO, Biosense Webster) of the left ventricle (A) in a patient with recurrent VT after previous anterior wall myocardial infarction (right anterior oblique projection). Red colour identifies scar, grey zones represent dense scar without pacing capture. Two morphologies of VT were present (B) that shared the same critical isthmus between two dense scars with exits on opposite sides (see markers # and * in the map and corresponding morphologies of VT with pacemaps from the exit site). Both VTs were successfully treated by a series of radiofrequency lesions between two dense scars (dark red dots).

Therefore, ablation is mostly used in an attempt to control refractory arrhythmias in patients with ICDs. Impaired left ventricular function also explains higher risk of complications such as stroke, myocardial infarction, heart block and/or perforation (approximately 5-8 %). Mortality risk may reach almost 3%.[22]

Arrhythmogenic right ventricular cardiomyopathy

Ventricular tachycardias associated with this clinical entity are re-entrant in origin and associated with fibrous and/or fibrofatty tissue in the right ventricle. Therefore, the typical ECG morphology is a LBBB configuration. Principally, the

same techniques as in post-infarction VTs can be used to characterize the reentry circuit. Based on our experience, catheter ablation appears to be successful in long term control of stable VTs in the presence of more significant right ventricular scarring and nonsyncopal VTs.[23]

Non-ischemic cardiomyopathy

Mechanism of VTs in nonischemic cardiomyopathies varies and includes scar-related reentry, focal origin and/or bundle branch reentry. This was emphasized in a study by Delacretaz et al[24] who reported re-entrant VTs in 62%, ectopic VTs in 27% and bundle branch reentry in 19% cases. Success rate reached 62 to 86%, according to the type of arrhythmia.

Previous surgery for congenital heart disease

Successful ablation of scar-related VTs after previous surgery for congenital heart disease has been reported anecdotally. Based on our own experience, re-entrant circuits can be identified accurately using electro-anatomical mapping. The obtained 3D map is then used as a guide for subsequent catheter ablation aiming at channels between dense scars in the right ventricle (Figure 2).

Bundle branch reentry

This specific arrhythmia accounts approximately for 5% of all monomorphic VTs in patients referred for electrophysiologic studies.[25] Reentrant circuit is composed of conduction system and transseptal intramyocardial conduction (Figure 3). Excitation spreads anterogradely through the right bundle branch, then transseptally to the left and retrogradely via the left bundle branch. Resulting ECG morphology is LBBB appearance with narrow rS complex contrasting with the enormous width of the whole QRS complex. Rarely, the impulse travels in opposite direction. This VT occurs predominantly in subjects with dilation of the heart and impaired intraventricular conduction (often LBBB pattern in sinus rhythm and prolonged HV interval). Catheter ablation is aimed at the right bundle and leaves this particular form of VT non-inducible. However, it can coexist with scar related VTs and many subjects will end up with an ICD even after successful ablation.

CONCLUSIONS

Catheter ablation may be considered as the first line treatment in all symptomatic idiopathic VTs, in recurrent VTs associated with structural heart disease and relatively preserved left ventricular ejection fraction (>35%) and/or bundle branch reentry VT.[26] It is also preferable treatment modality in incessant VTs of any origin.

Catheter ablation appears to improve arrhythmia control

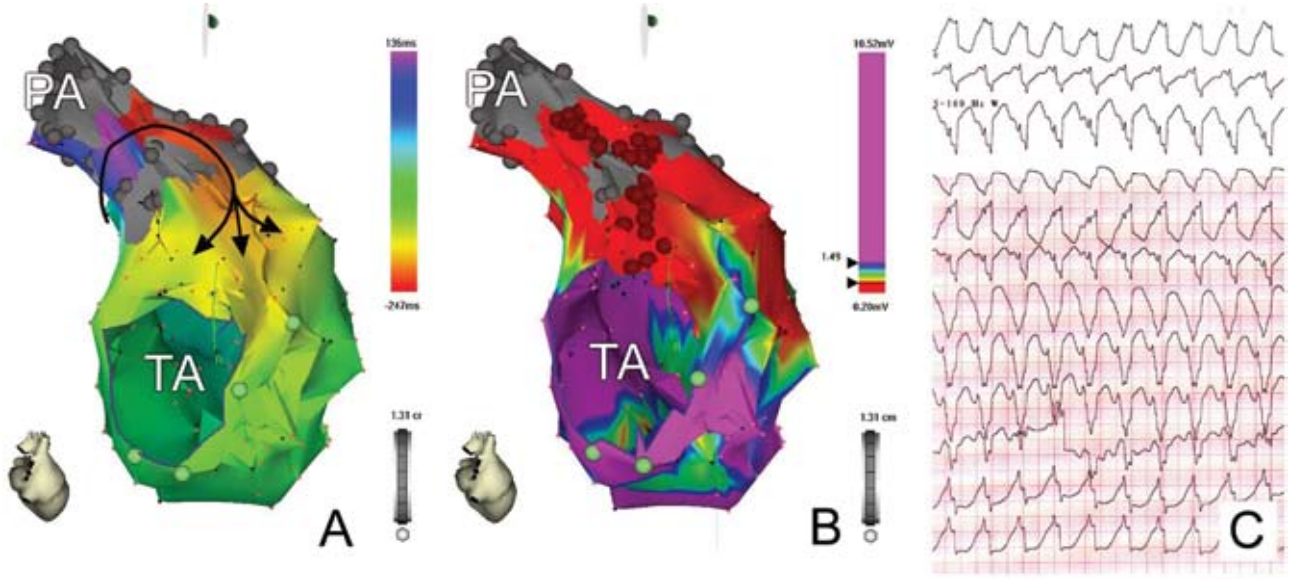

FIGURE 2. Both activation (A) and voltage (B) map of the right ventricle viewed from posterior aspect (facing tricuspid annulus – TA) in a patient after radical surgery for tetralogy of Fallot. Maps show dense scars corresponding to ventricular patch (lower grey area) and to a patch after ventricular outflow tract repair (upper grey area). Activation map was obtained during clinical VT (C) and reveals propagation of the wavefront via narrow channel between both scars (arrows). Corresponding voltage map shows that the whole surrounding area is a scar with low voltage of the signal (red colour). VT was successfully treated by lesions placed within the narrow channel. Then, a line of block was created from this region to the tricuspid annulus (dark red dots) to prevent induction of other forms of VT.

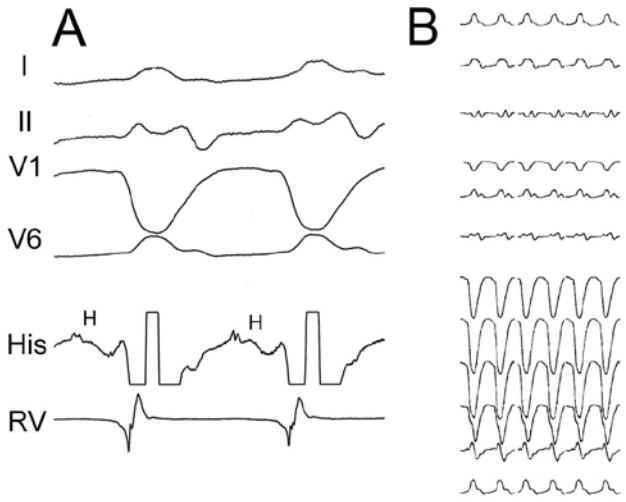

FIGURE 3. Bundle branch reentry tachycardia. Panel A shows selected leads of the surface ECG as well as intracardiac recordings from the His bundle region (His) and the right ventricular apex (RV). Panel B demonstrates typical ECG pattern of the arrhythmia with left bundle branch block appearance. Note that every ventricular electrogram is preceded by His bundle deflection (H) with a HV interval that is longer than that during sinus rhythm (75 ms). This VT was rendered non-inducible following catheter ablation of the right bundle.

in about two thirds of patients with structural heart disease and mappable VTs. Novel substrate and/or noncontact mapping techniques suggest that even untolerated VTs and/or VTs of multiple morphologies can be successfully ablated.[26-31] As the resulting noninducibility of VTs may not decrease the risk of dying suddenly in these patients with depressed left ventricular ejection fraction, the majority of patients with VT associated with structural heart disease remain recipients of ICD. On the other hand, catheter ablation is a method of choice in subjects with ICD and frequent arrhythmias.

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