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THE ROLE OF RADIOFREQUENCY CATHETER ABLATION IN THE TREATMENT OF NONISCHEMIC VENTRICULAR TACHYCARDIA

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SUMMARY – Radiofrequency catheter ablation can be used for the treatment of different patient groups with nonischemic ventricular tachycardia (VT). Concerning the small area of induced ventricular injury, a high degree success is expected in patients with idiopathic VT and in patients with bundle branch reentrant VT. The long-term success rate of radiofrequency ablation in the cure of idiopathic VT and bundle branch reentrant VT is about 90% and 100%, respectively. Radiofrequency ablation of VT in arrhythmogenic right ventricular dysplasia (ARVD) can be effective in patients with localized disease and single VT origin. In patients with more extensive ARVD and/or pleomorphic VT, other therapeutic options should be considered. In patients with idiopathic dilated cardiomyopathy, radiofrequency ablation is not curative but may be used as an adjunctive therapy to reduce the frequency and severity of VT. By using the new mapping systems, the application of radiofrequency ablation is expanded to the treatment of VTs requiring chamber compartmentalization or resulting in hemodynamic instability.

Key words: *Tachycardia, ventricular – therapy; Catheter ablation – methods*

Introduction

The therapeutic goal of radiofrequency catheter ablation in patients with ventricular tachycardia (VT) is to destroy the critical part of reentrant circuit or area from which the arrhythmia originates[1]. The susceptibility of radiofrequency catheter ablation varies depending on the substrate and pathophysiology of nonischemic VT. In various clinical settings, the efficacy of radiofrequency catheter ablation is less curative because the arrhythmogenic substrate of these tachycardias is too large, and radiofrequency energy cannot effectively reach the subendocardial or intramural tissue responsible for the emerging and maintaining of VT. Moreover, only hemodynamically well tolerated VTs allow for localization of the site of origin with conventional mapping technique during arrhythmia. This article will review the current place of radiofrequency catheter ablation in the management of patients with nonischemic VT.

Idiopathic Ventricular Tachycardia

Idiopathic VT occurs in young or middle-aged patients without structural heart disease. In the majority of patients, idiopathic VT originates from the right ventricular outflow tract or inferoposterior aspect of the left ventricle. Less commonly, idiopathic VT arises in other areas of the right or left ventricle. The mechanisms of idiopathic VT are different, including abnormal automaticity, triggered activity, and reentry. The prognosis of patients with idiopathic VT is benign, although the rare cases of sudden cardiac death have been observed.

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Table 1. Clinical results of radiofrequency catheter ablation in patients with idiopathic VT from the right ventricle

Author(s) (n)	Patients (%)	Success rate (n)	Complications (%)	VT recurrence (months)	Follow-up
Klein[1]	15	93	None	7	11
Calkins[2]	10	100	None	0	8
Coggins[3]	20	85	1 death	15	10
Chinusi[4]	13	92	None	8	28
Rodriguez[5]	35	88	None	11	30
Author[8]	17	88	None	10	56

VT=ventricular tachycardia

Idiopathic VT from the right ventricular outflow

This type usually shows left bundle branch block and inferior axis morphology on surface electrocardiogram. This tachycardia may occur in one of two forms: nonsustained repetitive monomorphic VT, or paroxysmal sustained monomorphic VT. Repetitive monomorphic VT usually is not inducible with programmed stimulation technique but isoproterenol infusion will precipitate VT. Paroxysmal sustained monomorphic VT is more often initiated and terminated by programmed ventricular stimulation, and its induction may be facilitated by isoproterenol infusion. These arrhythmias are sensitive to vagal maneuvers, verapamil and beta-blockers. Adenosine sensitivity strongly suggests that idiopathic VT from the right ventricular outflow tract is the result of cAMP-mediated triggered automaticity[2].

Radiofrequency catheter ablation is highly successful curative therapy for idiopathic VT from the right ventricular outflow tract. In the largest series, the long-term success rates have been about 90%[3-8] (Table 1). Recurrences of VT are rare and tend to occur early after ablation. These results are supported by our own experience showing a comparable success rate in 17 consecutive patients during a mean follow-up of 56 months[8]. Although we did not observe any serious complication, this therapeutic procedure is not without risk. Coggins *et al.*[5] report on a patient who died due to the consequences of cardiac tamponade caused by radiofrequency ablation in the right ventricular outflow tract, and Kay and Plumb[9] report on pericardial effusions requiring catheter drainage in two patients.

There is general agreement that the success rate of radiofrequency ablation of idiopathic VT is dependent on the accurate mapping of VT origin[3-7]. In VT arising from the right ventricular outflow tract, a 12 electrocardiogram lead concordance between pace-mapping and clinical VT is considered as the most valuable criterion for proper localization and ablation of the site of VT origin (Fig. 1). In all patients with such findings, we obtained a long-term success rate of 100%[8]. The high efficacy of radiofrequency catheter ablation of VT from the sites at which 12 of 12 pace-map was obtained is consistent with the spatial resolution of pace mapping[10] and size of lesions created during radiofrequency energy delivery. The value of endocardial activation time during VT or ventricular premature beats to guide radiofrequency ablation is of limited help because the earliest ventricular activation time is highly variable, and multiple unsuccessful ablation sites had endocardial activation time earlier than the successful site in other patients[5]. Thus, there is not a standard value in the degree of ventricular electrogram prematurity predicting successful ablation.

Idiopathic VT from the left ventricle

This type usually arises from a discrete site in the inferoposterior or inferoapical area of the left septum. These tachycardias have a characteristic right bundle branch block and superior axis morphology. They appear to be reentrant in origin, and therefore they can be readily induced and terminated by standard ventricular and/or atrial stimulation.

The radiofrequency catheter ablation has also been very successful in eliminating idiopathic VT from the left ventricle without any significant complication. In the study of When *et al.*, 17 of 20 patients with idiopathic left VT, who underwent radiofrequency ablation, were free from tachycardia after a mean follow-up period of 7 months[11]. Kay and Plumb report on successful ablation in 20 or 22 patients with idiopathic left VT[9]. In the study of Rodriguez *et al.*, 12 of 13 ablated patients with idiopathic VT from the left ventricle had no recurrences of clinical VT during a mean follow-up period of 30 months[7].

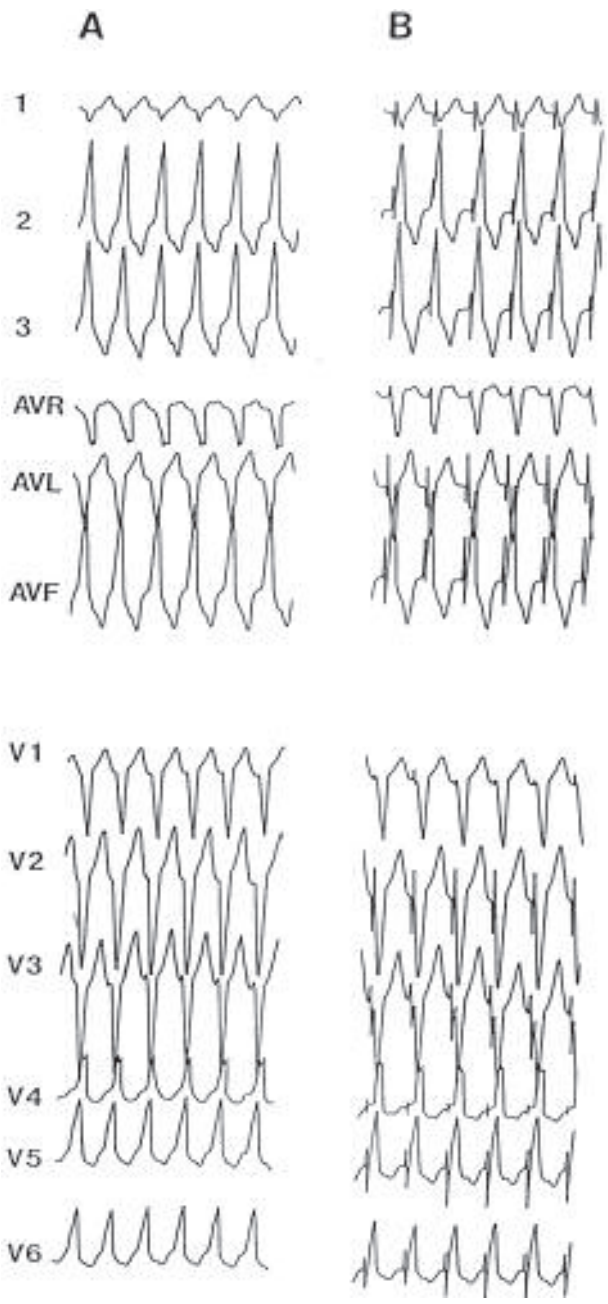

Fig. 1. Panel (A) shows a 12-lead electrocardiogram of the idiopathic VT originating at a mid-septal site of the right ventricular outflow tract, and panel (B) shows the optimal pace-map of this tachycardia. The paced QRS are identical to the QRS of clinical VT in 12 of 12 electrocardiographic leads.

An optimal pace-mapping and early endocardial activation time during VT are complementary to guide radiofrequency catheter ablation in patients with idiopathic VT originating from the left ventricle. Recently, a high-frequency deflection preceding the onset of QRS during tachycardia was recorded within the Purkinje network in the left posterior fascicle[12]. This deflection, called Purkinje potential, has been providing a very suitable site for successful ablation of idiopathic VT arising from the inferoposterior area of the left septum[7]. In our experience, Purkinje potential was identified as a target for successful ablation in one patient only. In other two patients, the earliest ventricular activation during VT and pace-mapping were sufficient for successful ablation[8].

The predictors of immediate or late unsuccessful ablation of VT from both right and left ventricles are: (a) induction of more than one VT morphology; (b) a delta wave-like beginning of QRS during VT; and (c) pace-mapping showing correlation in less than 11 of 12 electrocardiographic leads[7].

Bundle Branch Reentrant Ventricular Tachycardia

Bundle branch reentrant VT is tachycardia with a well-known anatomic circuit, which is consistent with the distal His bundle, right and left bundle branches, and the ventricular septum. It accounts for up to 6% of cases of clinical sustained monomorphic VT referred for evaluation[13]. Bundle branch reentrant VT occurs most commonly in patients with structural heart disease, especially in those with dilated cardiomyopathy or coronary heart disease. In this context, cardiomegaly, congestive heart failure, and atrial fibrillation are present in many of these patients.

Bundle branch reentrant VT has more often the left bundle branch block morphology. In this form of VT, the antegrade limb of the reentrant circuit is the right bundle branch, and the left bundle branch constitutes the retrograde limb. The bundle branch reentrant VT with right bundle branch block morphology has an opposite activation, while the reentrant circuit of the third type of bundle branch reentrant tachycardia incorporates the fascicles of the left bundle branch with incidental activation of the His bundle, right bundle branch, and ventricular myocardium. During tachycardia, the ventricular depolarization is preceded by the His-bundle, right-bundle, or left-bundle branch potentials, and spontaneous changes in the interval of these potentials precede similar changes in the ventricular to ventricular interval.

Radiofrequency catheter ablation of the right bundle branch is the treatment of choice for patients with bundle branch reentrant VT (Fig. 2). Ablation of the left bundle branch is needed in the rare cases with right bundle

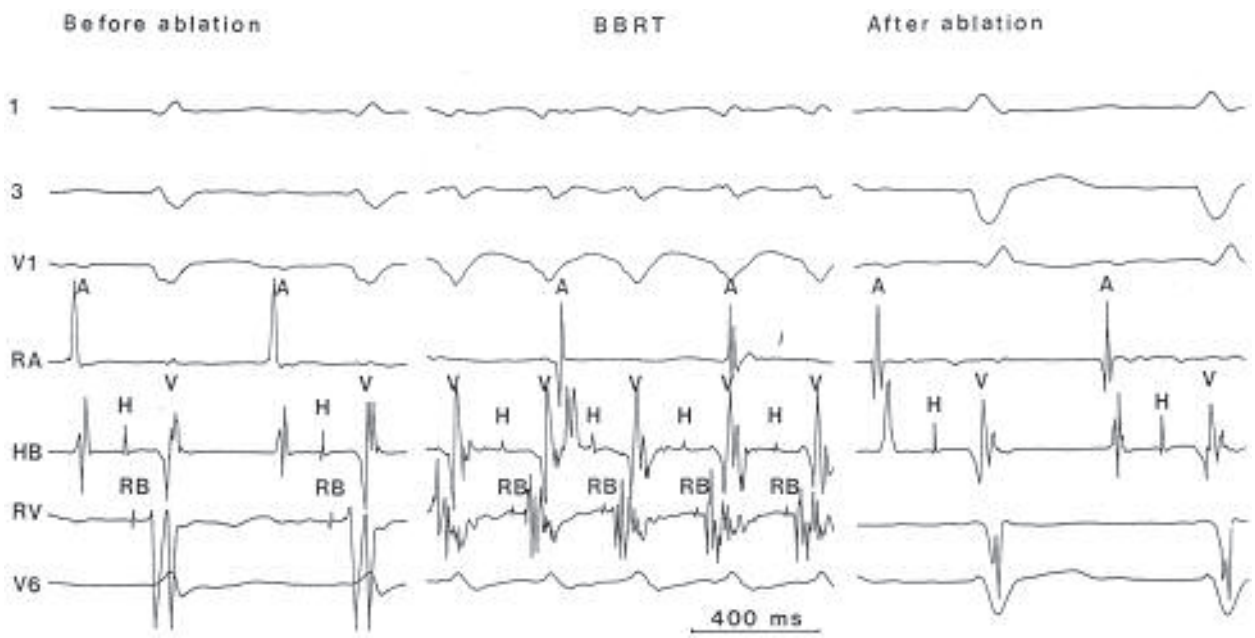

Fig. 2. Before ablation, sinus rhythm with left bundle branch block, prolonged HV interval, and a right bundle branch potential (RB) are present. Bundle branch reentrant VT (BBRT) has a left bundle branch morphology. The onset of each ventricular depolarization (V) is preceded by His (H) and right bundle branch potential. After ablation, sinus rhythm with right bundle branch block and more prolonged HV interval are present. Note the absence of right bundle branch potential at the intracardial right ventricular electrogram (RV). RA=right atrial electrogram; HB=His bundle electrogram.

branch block VT and left bundle branch block during sinus rhythm. Results from the literature and our experience show that radiofrequency ablation of the right bundle branch is highly effective in all patients with bundle branch reentrant VT[13-16] (Table 2). During intermediate and long-term follow-up, no recurrences have been reported in the presence of persistent conduction block along the ablated bundle during sinus rhythm. In spite of this, the long-term outcome of these patients was mostly influenced by the underlying heart disease. It is not surprising because the majority of these patients had significant structural heart disease with poor left ventricular function that can progress to congestive heart failure and to the development of other ventricular tachyarrhythmias. One of our patients had symptomatic VT of myocardial origin five months after ablation, which required implantation of a cardioverter-defibrillator; one patient with prolonged HV interval underwent pacemaker implantation; and two patients developed congestive heart failure as a consequence of further progression of their underlying disease[16].

Table 2. Clinical results of radiofrequency catheter ablation of bundle branch reentrant VT

Author(s)	Patients (n)	Success rate (%)	New VT (n)	Other therapy	Death (n)	Follow-up (months)
Cohen[13]	7	100	0	None	1 heart failure	12
Medhirad[14]	15	100	2	2 ICD	1 heart failure	22
				2 heart transpl	1 sudden death	
Klein[15]	6	100	2	3 PM	None	14
Author[16]	4	100	1	1 ICD, 1 PM	1 heart failure	20

VT=ventricular tachycardia; ICD=implantable cardioverter-defibrillator; transpl=transplantation; PM=pacemaker

Table 3. Clinical results of radiofrequency catheter ablation in patients with arrhythmogenic right ventriciular dysplasia

Author(s)	Patients (n)	Primary success (%)	VT recurrence (%)	Other therapy	Death (n)	Follow-up (months)
Wichter[14]	30	73	60	6 ICD 2 drugs	1 heart failure 1 sudden death	52
Asso[15]	6	66	50	1 ICD 2 drugs	None	22
Serrano[16]	15	58	42	2 ICD 3 drugs	1 heart failure	34
Author	6	50	66	2 ICD 2 drugs	None	60

VT=ventricular tachycardia; ICD=implantable cardioverter-defibrillator

Arrhythmogenic Right Ventricular Dysplasia

Arrhythmogenic right ventricular dysplasia (ARVD) is a heart muscle disease of unknown etiology, characterized by replacement of the right ventricular myocardium by fibrofatty tissue. The most common areas of ARVD are the apex, the infundibulum, and the inferior wall of the right ventricle. Clinical presentation of ARVD is usually related to ventricular arrhythmias that have a left bundle branch morphology. Based on electrophysiological testing, the mechanism of VT is most often reentry due to zones of slow conduction in areas of the involved myocardium. However, triggered activity due to delayed afterdepolarizations and abnormal automaticity have also been suggested as a mechanism of ventricular arrhythmias[17], particularly in patients with localized or so-called 'concealed' manifestations of ARVD.

The natural history of ARVD is a function of both cardiac electrical instability and progressive ventricular function. Therefore, performing radiofrequency ablation in the treatment of VT should be considered in relation to clinical manifestation and clinicopathologic phase of ARVD.

There are not many data on radiofrequency ablation of VT in patients with ARVD[18-20] (Table 3). In the available studies, the primary success, defined as noninducibility of clinical VT after the procedure, was obtained in 50% to 73% of patients. However, VT recurrences were very often clinical in patients with primary failure of ablation, and nonclinical occurring late after catheter ablation (Fig. 3). The analysis of these data shows that patients with localized ARVD and single morphology of VT have more favorable primary and long-term results of catheter ablation than patients with extensive ARVD and/or pleomorphic VT. In the study of Volkmer *et al.*[21], radiofrequency ablation was primarily successful in ten of 11 (90%) patients with focal VT, and in 28 of 34 (82%) patients with macroreentrant VT. However, there is no proof from the available data that catheter ablation offers a cure or long-term prevention in this subgroup of patients with ARVD.

Different endocardial catheter techniques have been used for localization of the site of VT origin in patients with ARVD. In patients with localized forms of ARVD and VTs due to a nonreentrant mechanism, pace-mapping appears to be of similar value for ablation as in patients with idiopathic VT. However, in the later phases of ARVD with more extensive clinical manifestations (overt phase, right ventricular failure), pace-mapping has proved to be less accurate and therefore insufficient for ablation. In these patients, areas of slow conduction have been shown to be suitable target sites for primary success of ablation. Slow-conduction tissue may be identified during endocardial catheter mapping by fractionated electrograms during sinus rhythm or VT, or by detecting the earliest electrical activity preceding the QRS complex (Fig. 3), mid-diastolic potentials, or continuous electrical activity during VT.

Several authors have shown that entrainment mapping can also be used in patients with ARVD and reentrant VT[18,22,23]. To be classified as a reentry circuit site, a post-pacing interval within 30 ms of the VT cycle length, or a stimulus-QRS interval during entrainment with concealed fusion within 20 ms of the electrogram-QRS interval is required (Fig. 4). The reentry sites are further classified by the stimulus-QRS duration as a percentage of the VT cycle length: exit sites have a stimulus-QRS duration less than 30% of the VT cycle length, central/proximal sites have a stimulus-QRS duration of 30% to 70% of the VT cycle length, and longer stimulus-QRS intervals indicate

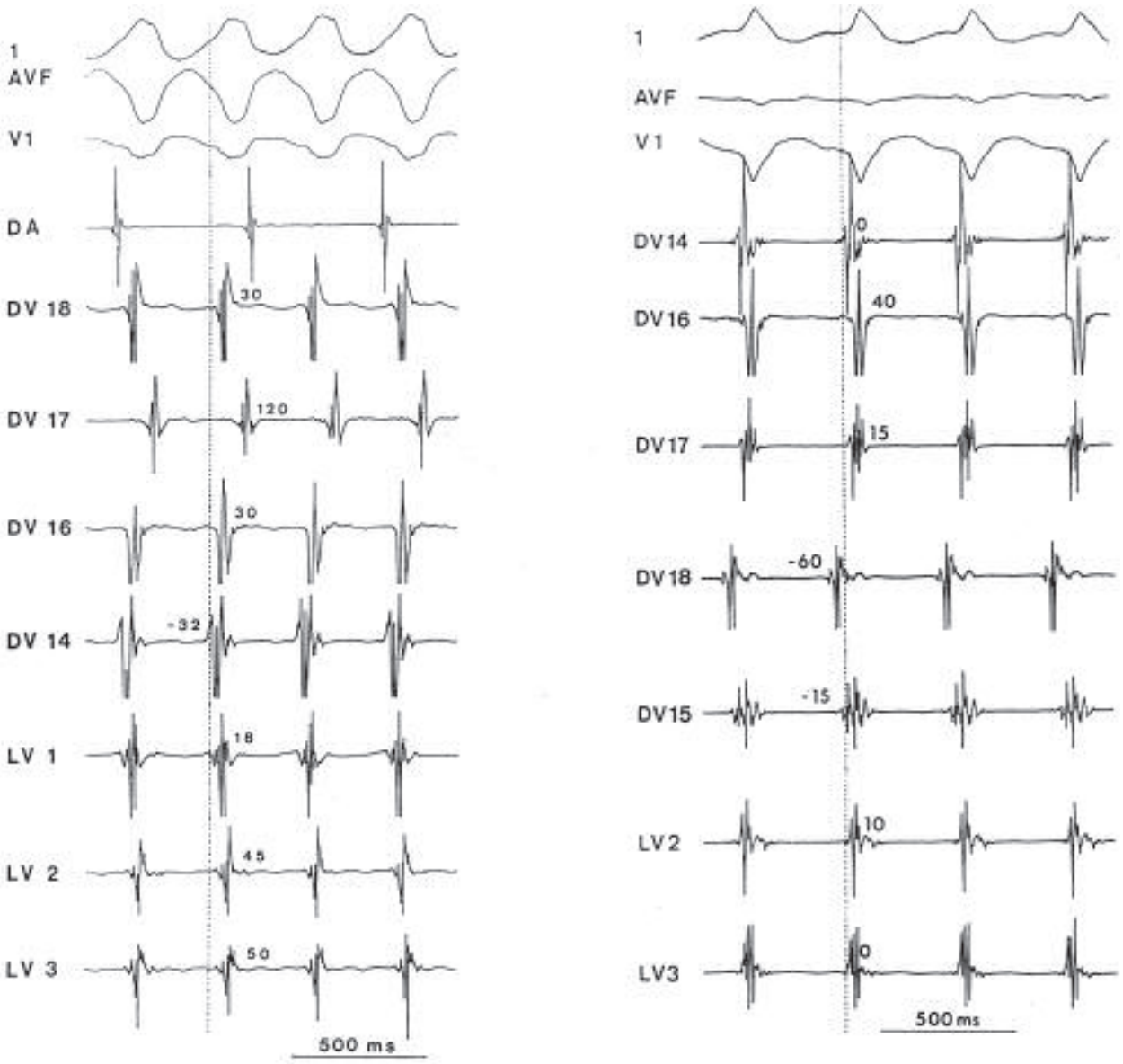

Fig. 3. Ten months after successful ablation of clinical VT originating at the apex of the right ventricle (DV 14, left panel), the patient developed a new form of VT with the earliest electrical activity at the anterior region of the right ventricle (DV 18, right panel). 1, AVF, and V1=surface electrocardiographic leads; DV 14 – DV 18=VT activation sites in the right ventricle; LV 2 and LV 3=VT activation sites in the left ventricle.

inner loop sites. If there is entrainment with concealed fusion but the postpacing interval is greater than 30 ms of the VT cycle length, the site is an adjacent bystander. Using these criteria, Harada *et al.* eliminated VT in six of seven patients with ARVD[22]. However, radiofrequency ablation was more successful in the elimination of VT at reentry circuit sites proximal to the exit as opposed to outer loop sites and exit sites, suggesting that critical isthmus for ablation of VT in ARVD may be distant to the reentry circuit exit.

Volkmer *et al.* have recently reported on the successful use of the electroanatomical mapping system CARTO in radiofrequency ablation of VT in five of six patients with ARVD[21]. Major advantages in the use of this technology

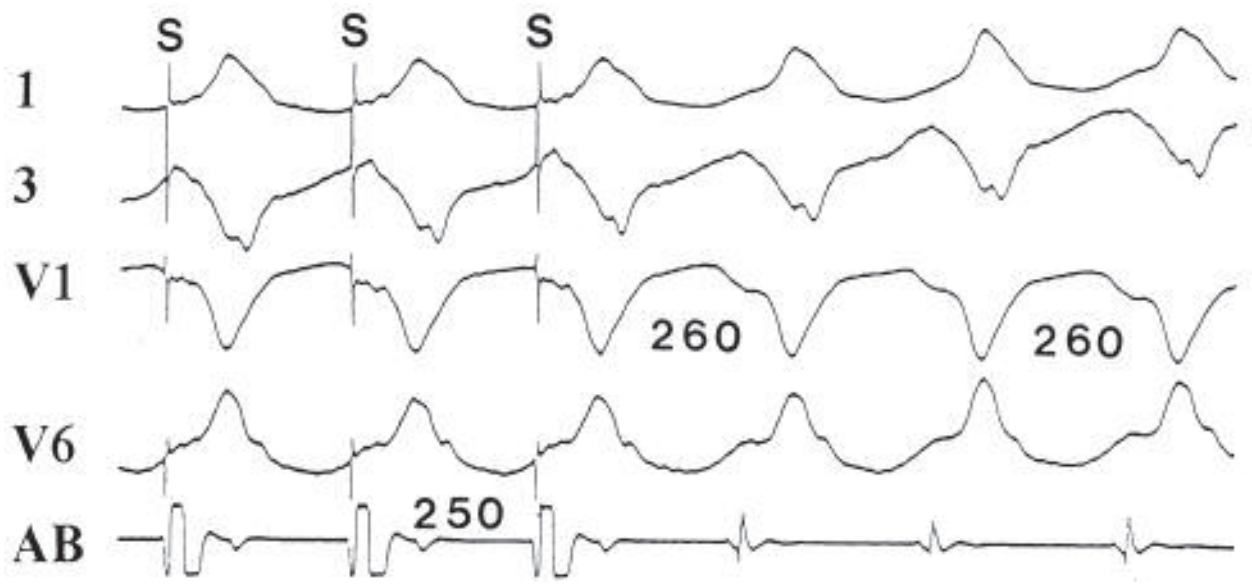

Fig. 4. Entrainment at the exit site is shown. Ventricular pacing (S) at cycle length of 250 ms does not change the QRS morphology of VT, and entrains the tachycardia with concealed fusion. The last post-pacing interval (260 ms) is identical to the VT cycle length, consistent with a reentry circuit site. The stimulus-QRS interval (50 ms) is the same as the electrogram-to-QRS interval during VT, and represents 19% of the VT cycle length, consistent with an exit site. 1, 3, V1 and V6=surface electrocardiographic leads; AB=ablation catheter at the right ventricular endocardial site.

were identification of the zone of slow conduction in macroreentry tachycardias and its relation to the surrounding electrical barriers such as scar tissue or tricuspid annulus system. This allowed for the application of linear lesions in order to connect these electrical barriers and to abolish the arrhythmogenic substrate.

The major limitation of catheter ablation in the treatment of VT secondary to ARVD refers to the progressive nature of the underlying disease predisposing for the occurrence of new arrhythmogenic foci during long-term follow-up despite successful ablation of clinical VT. In such patients, a combination of catheter ablation with other therapeutic options such as drugs or implantable cardioverter-defibrillator should be considered. In our experience, two of six ablated patients underwent implantation of cardioverter-defibrillator due to recurrences of nonclinical VT.

Idiopathic Dilated Cardiomyopathy

Idiopathic dilated cardiomyopathy (DCM) is a well recognized disease characterized by dilatation of both ventricles, and by clinical manifestation of congestive heart failure and serious ventricular arrhythmias. The mechanisms of sustained monomorphic VT in idiopathic DCM are less well understood than in the setting of myocardial infarction. A high degree of interstitial fibrosis, which was observed in patients with idiopathic DCM and inducible VT, is accompanied by a decrease in electrical coupling between adjacent myocytes[24]. This may in turn lead to slowing in conduction and susceptibility to reentrant arrhythmias. Additionally, several electrophysiologic properties, including inducibility of VT with critically timed extrastimuli and entrainment mapping, may be indicative of reentrant mechanisms of a subgroup of tachycardias in idiopathic DCM. In these patients, endocardial scarring, interstitial fibrosis, and other histopathologic characteristics may therefore constitute a 'final pathologic common pathway' for the occurrence of sustained monomorphic VT[25].

Data on the feasibility of radiofrequency catheter ablation in patients with idiopathic DCM are still limited. In the study of Kottkamp *et al.*, radiofrequency catheter ablation for the treatment of VT was attempted in eight patients with idiopathic DCM and mean ejection fraction of 30%[25]. Three patients suffered aborted sudden death, two experienced syncope, four had incessant VT, and five patients had chronic recurrent VT inducible with pro-

grammed ventricular stimulation. After the application of radiofrequency energy, the clinical VT was rendered noninducible in all patients with incessant VT, and in two of five patients with chronic recurrent VT. However, in six patients, the nonclinical VTs with other ECG morphologies were inducible after radiofrequency ablation. During a mean follow-up of 8 months, seven of eight patients were on antiarrhythmic drugs, four patients received cardioverter-defibrillator, one underwent heart transplantation, and one patient who was resuscitated from ventricular fibrillation died due to congestive heart failure. Thus, a complete prevention of VT could only be achieved in two of eight patients.

Recently, Marchlinsky *et al.*[26] have reported on radiofrequency ablation of unmappable, monomorphic VT in six patients with implanted cardioverter-defibrillator and nonischemic DCM using the CARTO magnetic mapping system. After the application of multiple (median, 55) linear radiofrequency lesions, three patients had VT episodes at 3, 9 and 13 months, respectively, but only one of these patients had frequent VT that was amenable to pacing therapy. The results of these studies indicate that radiofrequency current application for ablation of VT in patients with nonischemic DCM is feasible and may be a valuable adjunctive therapy in patients who cannot be appropriately treated by conventional treatment modalities.

Conclusion

Initially successful radiofrequency catheter ablation of idiopathic VT has high long-term efficacy without producing proarrhythmic or cardiodepressing effects. Therefore, this procedure should be considered as an alternative option for symptomatic patients with idiopathic VT.

Radiofrequency catheter ablation of the right bundle branch is a safe and highly effective therapeutic option for definite cure of bundle branch reentrant VT. Long-term prognosis of these patients depends mainly on the underlying heart disease and the treatment of other, myocardial VT.

Radiofrequency catheter ablation of VT in patients with ARVD can only be successful in the early phase of disease when the arrhythmogenic area is not too large. Later, because of further progression of ARVD, the results of radiofrequency ablation are not satisfactory, and other therapeutic options should be considered.

The primary efficacy of radiofrequency catheter ablation in the treatment of VT in patients with idiopathic DCM is low. Therefore, radiofrequency catheter ablation may only be used in these patients as an adjunctive therapeutic option to reduce the frequency and severity of VT.

Recently, two innovative mapping systems have been introduced in clinical practice, allowing for three-dimensional reconstruction of electrical activation within a target area, i.e. a nonfluoroscopic and noncontact mapping systems[27]. These two new strategies should be most suitable to guide mapping and radiofrequency ablation of VTs, requiring chamber compartmentalization, or resulting in hemodynamic instability[21,26].

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Sažetak

ULOGA RADIOFREKVENTNE KATETERSKE ABLACIJE U LIJEČENJU NEISHEMIJSKE VENTRIKULSKE TAHIKARDIJE

D. Petrač

Radiofrekventna kateterska ablacija može se rabiti u liječenju različitih skupina bolesnika s neishemijskom ventrikulskom tahikardijom (VT). Zbog malog područja izazvane ozljede miokarda visok stupanj uspjeha očekuje se kod bolesnika s idiopatskom VT i bolesnika koji imaju VT s kruženjem preko grana. Dugoročna uspješnost radiofrekventne ablacije u izliječenju idiopatske VT je oko 90%, a VT s kruženjem preko grana 100%. Radiofrekventna ablacija VT kod aritmogene displazije desnog ventrikula (ADDV) može biti djelotvorna u bolesnika s lokaliziranom bolešću i jednim izvorištem VT. U bolesnika s proširenom ADDV i/ili više izvorišta VT treba razmotriti druge načine liječenja. Kod bolesnika s idiopatskom dilatacijskom kardiomiopatijom radiofrekventna ablacija ne dovodi do izliječenja VT, ali se može primijeniti kao dodatni terapijski postupak radi smanjenja učestalosti i težine napadaja tahikardije. Upotrebom novih sustava za endokardijalno 'mapiranje' primjena radiofrekventne ablacije se proširila na liječenje VT koje zahtijevaju razdvajanje pojedinih dijelova ventrikula ili su hemodinamski nestabilne.

Ključne riječi: *Tahikardija, ventrikulska – terapija; Kateterska ablacija – metode*