Radiofrequency catheter ablation of ventricular tachycardia in arrhythmogenic right ventricular dysplasia

Josef Kautzner, Robert Čihák, Vlastimil Vančura, Kateřina Lefflerová, Jan Bytešník

Department of Cardiology, Institute for Clinical and Experimental Medicine, Prague, Czech Republic

Background: Ventricular tachycardia (VT) in arrhythmogenic right ventricular dysplasia (ARVD) is believed to be of intramyocardial reentry origin. However, there is still limited information about the character of reentry circuits, and about the long-term benefit of radiofrequency (RF) catheter ablation.

Methods: To address this issue, 8 monomorphic, hemodynamically stable VTs were mapped in 6 patients with ARVD (5 men, mean age 48±15 years). After identification of late potentials in sinus rhythm, VT was induced by programmed ventricular stimulation. Entrainment was performed during mapping by pacing from the distal bipole of the mapping catheter at cycle length 20-50 ms shorter than that of VT. RF current was applied in areas judged to be central or exit, or (in the case when pacing was impossible) in areas with mid-diastolic potentials close to the entrainable site. The success of RF ablation was defined as termination of VT and non-inducibility of clinical (or hemodynamically stable) VT.

Results: RF ablation was successful in 5 patients, and in 7 of 8 clinical VTs. The mean procedure duration was 188 ± 112 min with the mean fluoroscopic time of 12.8 ± 10 min. During mid-term follow-up (33 ± 7 months, range 22-39), 2 recurrences of VT of different morphology were noticed within 1 year period, and RF ablation was successfully repeated in both cases. Long-term follow-up (mean 48 ± 16 months, range 26–63) revealed that three patients died: none of them suddenly, two of them defintely not of arrhythmia recurrence. One re-ablation supported by electroanatomical mapping was performed in the patient with initially unsuccessful RF ablation. Two other patients are without recurrences of arrhythmias.

Conclusions: VT in ARVD shows many characteristics of VT after myocardial infarction. RF catheter ablation of VT in ARVD guided by activation sequence and entrainment mapping appears to be effective and safe technique for selected patients.

Key words: ventricular tachycardia, arrhythmogenic right ventricular dysplasia, radiofrequency catheter ablation, entrainment mapping.

RADIOFREKVENČNÍ KATETRIZAČNÍ ABLACE KOMOROVÉ TACHYKARDIE PŘI ARYTMOGENNÍ DYSPLAZII PRAVÉ KOMORY

Východiska: Komorová tachykardie (KT) u arytmogenní dysplazie pravé komory (ARVD) je považována za arytmii typu reentry. Přesto existuje omezené množství informací o charakteru okruhů reentry, bezpečnosti radiofrekvenční (RF) katetrizační ablace a o jejím dlouhodobém přínosu.

Metody: K ozřejmení uvedených otázek bylo mapováno celkem 8 hemodynamicky stabilních, monomorfních KT u 6 nemocných s ARVD (5 mužů, průměrný věk 48±15 let). Po identifikaci oblasti pravé komory s pozdními potenciály mapováním při sinusovém rytmu byla indukována KT pomocí programované stimulace komor. Během mapování při KT bylo používáno stimulace z distálního bipólu ablačního katétru o délce cyklu o 20-50 ms kratší než při KT k posouzení tzv. entrainmentu. RF proud byl aplikován v oblastech, které byly identifikovány jako centrální zóna nebo zóna exitu okruhu reentry, nebo v oblasti s mid-diastolickými potenciály. Úspěšnost ablace byla definována jako ukončení KT a nemožnost indukce klinické (nebo jiné tolerované) formy KT.

Výsledky: RF ablace byla úspěšná u 5 pacientů a u 7 z 8 klinických KT. Průměrné trvání výkonu dosáhlo 188 ± 112 minut s průměrným skiaskopickým časem 12,8 ± 10 minut. Během střednědobého sledování (33 ± 7 měsíců, rozmezí 22–39) byly zaznamenány u 2 nemocných rekurence KT jiné morfologie a obě KT byly odstraněny další katetrizační ablací. Během dělédobého sledování (48 ± 16 měsíců, rozmezí 26–63) zemřeli tři nemocní: žádný z nich náhle, dva z nich jistě ne pro rekurenci arytmie. Byla provedena jedna další ablace (pomocí systému pro elektroanatomické mapování) u pacienta s původně neúspěšnou ablací. Dva další pacienti zůstali bez recidiv arytmie.

Závěry: KT při ARVD vykazuje řadu charakteristik, které jsou podobné jako při KT po infarktu myokardu. RF katetrizační ablace KT při ARVD navigovaná pomocí mapování aktivační sekvence a entrainmentu je pro vybrané pacienty účinnou a bezpečnou metodou.

Klíčová slova: komorová tachykardie, arytmogenní dysplazie pravé komory, radiofrekvenční katetrizační ablace, mapování pomocí entrainmentu.

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Introduction

Arrhythmogenic right ventricular dysplasia (ARVD) is a distinct form of cardiomyopathy associated with reentrant ventricular tachycardias (VT) originating from the right ventricle[1]. The anatomical substrate for these arrhythmias consists of fibrolipomatous tissue that replaces predominantly right ventricular myocardium[2]. Compared to VT developing late after myocardial infarction, the efficacy and safety of radiofrequency (RF) catheter ablation remains more controversial[3]. In this respect, there is only limited information about the character of reentry circuits in ARVD and on the role of entrainment mapping techniques[4, 5].

In addition, long-term follow-up after RF ablation is available for small series of patients. The purpose of this study was to evaluate the applicability of entrainment techniques to identify appropriate targets for radiofrequency catheter ablation of VT in ARVD, and assess the success rate of RF catheter ablation, its safety and long-term efficacy.

Methods

Of the total number 109 cases of inducible sustained monomorphic VTs over a period of 24 months, ARVD was diagnosed to be the underlying disease in 8 cases (7.3 %). The diagnosis of ARVD was based on criteria of the Task Force of the Working Group on Myocardial and Pericardial Disease of the European Society of Cardiology and of the Scientific Council on Cardiomyopathies of the International Society and Federation of Cardiology[6]. The presence of excessive amount of fatty tissue in the right ventricular free wall together with its thinning was confirmed in all cases by magnetic resonance imaging. Coronary angiography excluded coronary artery disease.

Mapping with entrainment and RF ablation was performed in 6 of 8 cases (5 men, 1 female, mean age 48 ± 15 years) with spontaneous episodes of sustained monomorphic VT despite antiarrhythmic treatment. None of the patients had a history of cardiac arrest. Clinical VT was invariably of left bundle branch block morphology.

Figure 1. Late ventricular potentials recorded in sinus rhythm in ARVD patient. Figure shows surface ECG lead V2 with prominent epsilon wave (arrow) and both unipolar and bipolar intracardiac recordings from mapping catheter (MAP uni and MAP 1.2, respectively) and ablation catheter (ABL uni, ABL 1.2) together with the recording from the right ventricular apex (RVA). Of note is correspondence of late potentials in mapping and ablation catheter recordings with the epsilon wave.

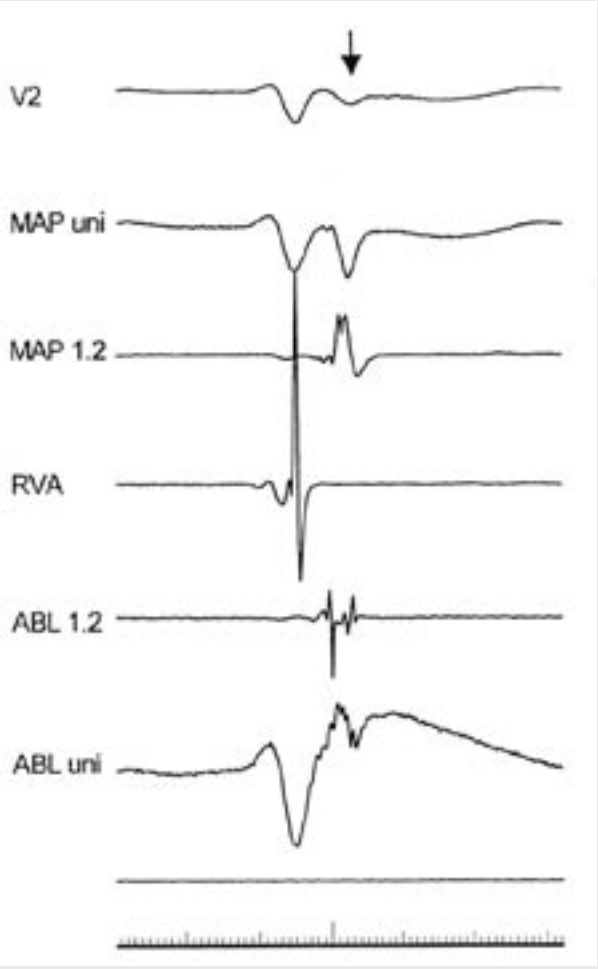
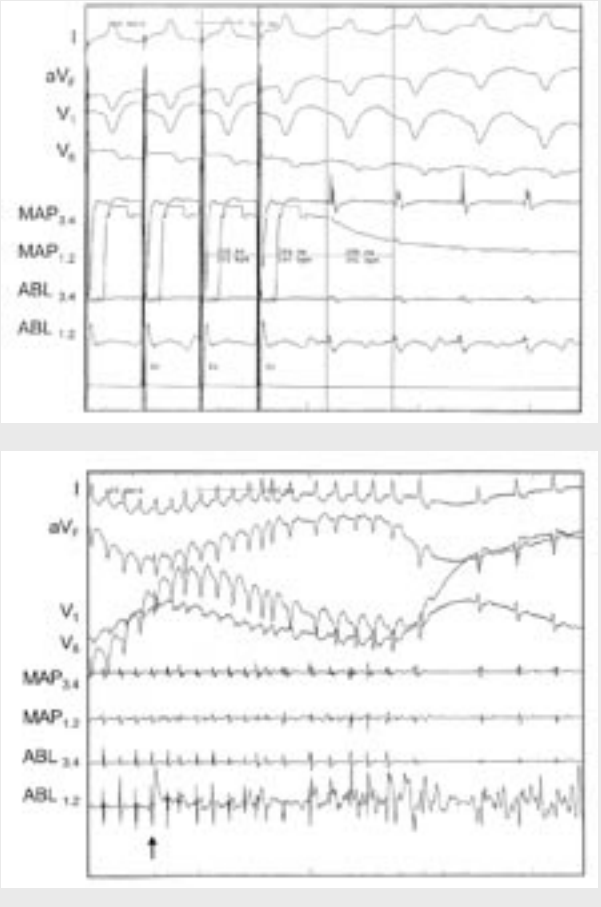


Figure 2. A. Entrainment at an exit site of the reentrant circuit in the right ventricle. VT shows left bundle branch block QRS pattern with superior axis and cycle length of 298 ms. Bipolar pacing from distal bipole of mapping catheter (MAP 1.2) at cycle length of 258 ms entrains VT and does not change the QRS morphology, i. e. VT is entrained with concealed fusion. The post-pacing interval is 304 ms and thus, approximates the VT cycle length. The stimulus to QRS interval is 70 ms, which is consistent with an exit site (i. e. less than 30 % of the VT cycle length). B. Ablation at this site (after switching mapping catheter-MAP for ablation catheter- Abl) terminates the VT (arrow shows initiation of RF application).

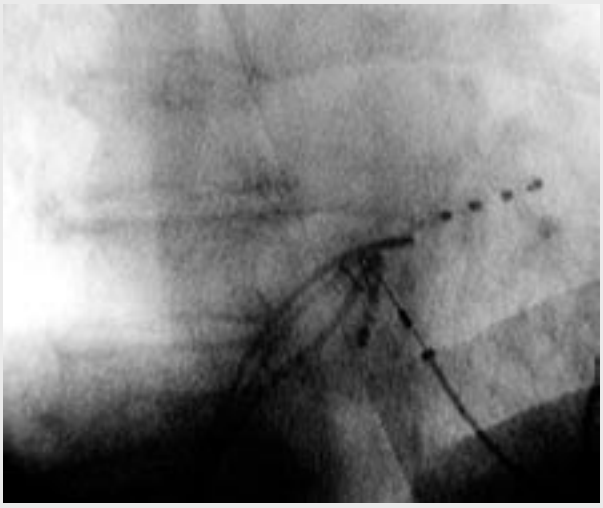


Endocardial mapping was performed using two steerable 7F ablation catheters (Celsius, Cordis Webster, Inc, Baldwin Park, CA). The catheters were introduced into the right ventricle via femoral vein together with one diagnostic quadripolar catheter (Response, Daig Corporation, Minnetonka, MN) that was placed in the right ventricular apex for pacing. Catheter mapping in sinus rhythm was used to identify areas of abnormal electrograms and late potentials (Fig. 1). VT was then induced by programmed ventricular stimulation and fractionated potentials were searched in diastole using a rowing technique with two ablation catheters. Entrainment was performed by pacing from the distal bipole of the ablation catheter at cycle length 20–50 ms shorter than that of VT (Fig. 2A)[7]. RF current was applied in areas judged to be central or exit, or (in the case when pacing was impossible) in areas with mid-diastolic potentials close to the entrainable site. Whenever RF application terminated VT, the lesion was enlarged during sinus rhythm. If RF current did not terminate VT, the catheter was moved to a new site. This approach was repeated until clinical VT was non-inducible. The success of RF ablation was defined as termination of VT and non-inducibility of clinical (or hemodynamically stable) VT. During subsequent follow-up, all patients continued to take antiarrhythmic treatment they received before the ablation procedure.

Results

Abnormal, low-amplitude intracardiac late potentials were identified in the region of right ventricular free wall in all patients. In two patients with discernible epsilon wave on surface ECG these potentials corresponded with the timing of the epsilon wave. A total number of 8 sustained monomorphic and hemodynamically tolerated VTs were induced in 6 patients. Two patients had two VTs of different morphology and cycle length inducible. The mean VT cycle length of VTs was 345 ± 27 ms. RF ablation targeting central or exit sites within the reentry circuit was successful in 7 VTs, i. e. in 5 patients (Fig. 2B). In one patient with tolerated VT (cycle length of 370 ms), RF ablation aimed at mid-diastolic potentials within the reentry circuit did not terminate VT. Sites where RF ablation terminated VT tended to cluster on the right ventricular free wall close to the tricuspid annulus (7 of 8 VTs) (Fig. 3).

Figure 3. Fluoroscopic image (RAO view) of one of the predominant ablation sites adjacent to the tricuspid annulus. The location of the reentrant circuit is marked by two ablation catheters residing at lateral wall of the right ventricle, while quadripolar pacing catheter is placed in the right ventricular outflow tract.



Fast VTs that required immediate termination with DC shock were induced in 3 patients after RF ablation. These were not considered as clinically relevant. The mean procedure duration was 188 ± 112 min with the mean fluoroscopic time of 12.8 ± 10 min. There were no complications associated with the procedure or afterwards.

During mid-term follow-up (mean 33 ± 7 months, range 22–39), 2 recurrences of tolerated monomorphic VT of different morphology were noticed within one-year period in 2 patients. One of them had no inducible VT following the first RF ablation, while the second one had fast VT inducible. RF ablation was successfully repeated in both of them, however, the second patient had still fast VT readily inducible. An ICD was implanted in this patient and no recurrence of VT was observed during next 24 months of follow-up. The patient with initially unsuccessful RF ablation had no recurrence of VT over a period of 38 months.

Long-term follow-up (mean 48 ± 16 months, range 26-63) revealed that three patients died: one patient died 32 months later due to cerebrovascular accident, the other died of progressive heart failure 51 months later, and the third patient died 26 months after catheter ablation due to unknown cause. One re-ablation supported by electroanatomical mapping was performed in the patient with initially unsuccessful RF ablation. Two other patients are without recurrences of arrhythmias, one of them had an ICD implanted for persistent inducibility of non-clinical fast VT.

Discussion

This study provides another piece of evidence that the mechanism for VT in ARVD is intramyocardial reentry. First, areas of abnormal late potentials (corresponding with the presence of epsilon wave on the surface ECG in some patients) were demonstrated in sinus rhythm. Second, entrainment techniques allowed identification of reentrant circuits using the criteria derived from mapping of post-infarction monomorphic VTs. Third, RF catheter ablation targeting central-to-exit sites within the reentrant circuits was initially successful in 7 of the 8 tolerated VTs.

However, as we used a limited mapping approach of the reentrant circuits we cannot analyse which portion of the circuit was the most critical for successful RF ablation. Based on our successful strategy to apply RF current at areas judged to be central or exit, or (in the case when pacing was impossible) in areas with mid-diastolic potentials very close to the entrainable site, it appears that these sites within the circuit predict success of RF ablation. In this respect, Ellison et al. showed that RF ablation in exit zones was associated with the success in approximately 25 % ablation sites[4]. However, the same proportion of successful VT terminations during RF energy application was achieved also in outer loop sites. Harada et al. reported the highest success of RF

ablation in terminating VT in ARVD when targeting sites proximal to exit as opposed to exit sites and/or outer loop sites[5]. Current practice of using an electroanatomical mapping system in a support of the procedure is expected even better characterize arrhythmia substrate and further increase ablation success rate[8]. In addition, it should also help to characterize the extent of scar within the right ventricle and anotate points of interest directly into a 3-D map.

Similarly as in reports of others[4, 8], successful ablation sites in our patient population clustered around the tricuspid annulus. Such a preferential location of critical isthmus sites appears to be analogy to the situation in the left ventricle after inferior wall myocardial infarction[9, 10]. These observations together with higher success rate of RF catheter ablation in preferential locations suggest that the annulus may serve as a natural anatomical barrier for a reentry circuits. It may also mean that such peri-annular location of the reentry circuit manifest itself as well tolerated VT. That selected cohort of patients may mostly benefit from RF catheter ablation.

Although the number of patients in this study is small and not sufficient to analyze safety issues and overall efficacy, the results appear to confirm experience of others[4, 5]. Regarding the risk of procedural complications, no perforation was observed despite significant thinning of the right ventricular myocardium as documented by magnetic resonance imaging. Mid-term follow up in our study group was on average 33 months and during this period, two recurrences of well tolerated VT were noticed. These clinical VTs of different morphology were again successfully treated by RF catheter ablation. Such recurrence rate is lower than that reported by Shoda et al. who observed recurrent VTs with different morphology in 5 of 10 patients over a period of 8–20 months[11]. Long-term follow-up (average 48 months) revealed three deaths, none of them sudden. One re-ablation supported by electroanatomical mapping system was performed in the patient with initially unsuccessful RF ablation. Two other patients are without recurrences of arrhythmias, one of them had an ICD implanted for persistent inducibility of non-clinical fast VT. Based on previous experience that patients with ARVD who present with recurrent VTs have quite favourable outcome on antiarrhythmic drugs[1], we believe that RF catheter ablation of clinical, well tolerated VTs in association with continuation of pre-ablation antiarrhythmic therapy is reasonably safe approach in selected patients.

In conclusion, VT in ARVD shows many characteristics of VT after myocardial infarction. RF catheter ablation of VT in ARVD guided by activation sequence and entrainment mapping appears to be effective and safe technique. In association with antiarrhythmic drugs, RF catheter ablation is acceptable strategy for selected patients with recurrent and hemodynamically well tolerated VTs.

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Josef Kautzner, MD, PhD, FESC
Department of Cardiology, Institute for Clinical and Experimental Medicine
Vídeňská 1958/9, 140 21 Prague 4
e-mail: josef.kautzner@medicon.cz

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