Electroanatomic mapping characteristics of ventricular tachycardia in patients with arrhythmogenic right ventricular cardiomyopathy/dysplasia

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Abstract Background Ventricular tachycardia (VT) in arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVD) has been previously explored using entrainment mapping techniques but little is know about VT mechanisms and the characteristics of their circuits using an electroanatomical mapping system.

Methods and results Three-dimensional electroanatomical mapping was performed in 11 patients with well tolerated sustained VT and ARVD. Sinus rhythm mapping of the right ventricle was performed in eight patients showing areas of low bipolar electrogram voltage (<1.2 mV). In total 12 tachycardias (mean cycle length 382 ± 62 ms) were induced and mapped. Complete maps demonstrated a reentry mechanism in eight VTs and a focal activation pattern in four VTs. The reentrant circuits were localized around the tricuspid annulus (five VTs), around the right ventricular outflow tract (one VT) and on the RV free lateral wall (two VTs). The critical isthmus of each pertricuspid circuit was bounded by the tricuspid annulus with a low voltage area close to it. The isthmus of tachycardia originating from the right ventricular outflow tract (RVOT) was delineated by the tricuspid annulus with a low voltage area close to it. The isthmus of tachycardia originating from the right ventricular outflow tract (RVOT) was delineated by the tricuspid annulus with a low voltage area close to it. Each right ventricular free wall circuit showed an isthmus delineated by two parallel lines of block. Focal tachycardias originated on the right ventricular free wall. Linear radiofrequency ablation performed across the critical isthmus was successful in seven of eight reentrant tachycardias. The focal VTs were successfully ablated in
Ventricular tachycardia (VT) is a common complication in patients with arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVD). More severe arrhythmias are associated with ARVD since it may be responsible for sudden cardiac death in young people [1].

Several approaches have been proposed for treating VT in ARVD. Implantable cardioverter defibrillator (ICD) therapy provides a life-saving protection by effective termination of the tachycardia; however, complications may be encountered [2,3].

Modification of the substrate of VT was initially approached through surgical methods and endocardial ablation with DC shock-fulguration [4–6]. More recently, radiofrequency (RF) catheter ablation has emerged as a safe technique with an acute success rate, defined as prevention of reinduction of VT ranging from 42 to 74% [7–9].

Two approaches have been proposed for the identification of the target site for RF ablation. The first of these uses classical mapping methods such as entrainment, to confirm the reentry mechanism of the tachycardia and to characterize the circuit [7,10]. The second approach is based upon new electroanatomical mapping techniques [8–12].

The purpose of the present study is to report on VT mechanisms in a consecutive series of patients with ARVD in whom VT mapping was performed using an electroanatomical mapping system.

Methods

Patients

Between January 2000 and June 2003, 11 patients (eight men, mean age 50 ± 17 years) were referred to our centre for RF ablation of VT related to ARVD (Table 1). The diagnosis of ARVD was confirmed in all, using the recently published criteria [1].

In four patients right ventricular ejection fraction had been measured: patients 1, 2, 3 and 6. These were, respectively, 30%, 25%, 50% and 10%.

In all other patients either right ventricular angiography or echocardiography identified right ventricular dilatation or zones of dyskinesia. Left ventricular ejection fraction was 54 ± 9%, with two patients (patient 5 and 11) having a left ventricular ejection fraction below 50%. Patient 6 underwent an MRI scan showing fatty infiltration of the left ventricular apex.

Four patients had been previously equipped with an automatic implantable cardioverter defibrillator (ICD) because of resuscitated sudden cardiac death or haemodynamically unstable VT. They were referred to our institution because of either underdetection of VT (patient 11) or multiple ICD shocks (patients 5, 8 and 9) despite antiarrhythmic drugs. The remaining seven patients presented with haemodynamically well tolerated VT and symptoms of palpitations and/or presyncope. In four of them the diagnostic work-up of the VT led to the diagnosis of ARVD. The other three suffered from recurrent VTs despite antiarrhythmic drugs.

Electrophysiological study and ablation

Electrophysiological study and ablation were performed after obtaining written informed consent and after a period of fasting of 12 h. All antiarrhythmic drugs had been stopped for at least five half-lives. The ICDs were switched off during the procedure.

Under local anaesthesia two catheters were advanced via the right femoral vein into the right ventricle (RV). The first was a 5F bipolar diagnostic catheter placed at the RV apex and the second was a 7F or 8F mapping/ablation catheter (NAVISTAR®, Cordis-Webster, Diamond Bar, CA, USA, Johnson & Johnson, USA) with an 8 mm tip electrode. Bipolar electrograms were filtered at 30–400 Hz, while unipolar electrograms were not filtered. Pacing was delivered through an external stimulator (Biotronik UHS 20, Biotronik Inc, Berlin, Germany) at twice the diastolic threshold and at a pulse duration of 2 ms. Ventricular programmed electrical stimulation (PES) consisted of delivering up to three extrastimuli during spontaneous
rhythm and during paced rhythm (600 ms and 400 ms drive cycle length) at the RV apex. Failure to induce VT prompted the same protocol at the RV outflow tract and then again at the RV apex with infusion of isoprenaline.

**Mapping technique**

Three-dimensional (3D) endocardial activation maps of the RV were created with the CARTO®, Biosense-Webster (Diamond Bar, CA, USA) system and NAVI-STAR® mapping and reference catheters. This technique has been described in detail elsewhere [13–14]. The automatically assigned activation times were adjusted manually where needed, as described by Shah et al. [15]. For double potentials (DPs) the earliest peak deflection was used.

Mapping was started in sinus rhythm in eight patients in order to obtain an anatomical reconstruction of the RV. Next, the VT was induced by ventricular PES. A remap procedure was initiated upon induction of VT starting from the anatomical map created in sinus rhythm. Mapping was continued until a sufficient density of points had been obtained to understand the VT mechanism. Three patients (patients 5, 6 and 8) presented with incessant VT at the beginning of the ablation procedure. In those patients mapping was performed solely during VT. Conventional entrainment manoeuvres were not used, because in our experience frequent interruption or change in VT occurs as a result of such manoeuvres. No pace mapping was performed for any patient.

**Definitions**

A complete reentrant circuit was considered to be the spatially shortest route of unidirectional activation encompassing the full range of mapped activation times (more than 90% of the tachycardia cycle length) and returning to the site of earliest activation. During VT, a critical isthmus was defined as an area of slow conduction delineated by nonconductive tissue (a line of double potentials, a scar area, or an anatomical obstacle such as the tricuspid valve or the pulmonary valve) where the VT activation has to pass to perpetuate the tachycardia. As a consequence ablation of the critical VT isthmus should interrupt the tachycardia and prevent its re-inducibility [16].

A focal pattern of activation was defined as a wavefront radially spreading from a single site of earliest activation. The range of activation times is considerably less than the tachycardia cycle length.

Double potentials were defined as an electrogram with two distinct ventricular deflections separated by an isoelectric interval of at least 50 ms.

### Table 1

<table>
<thead>
<tr>
<th>Patient characteristics</th>
<th>Patient</th>
<th>Age (y)</th>
<th>Sex</th>
<th>ICD before RF</th>
<th>VT cycle</th>
<th>VT morphology</th>
<th>Circuit location</th>
<th>Ablation result</th>
<th>Control VPES</th>
<th>ICD after RF</th>
<th>Recurrences of ablated VTs</th>
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<td>LBBB/LA</td>
<td>Failure</td>
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W = male, F = female, ICD = implantable cardioverter defibrillator, RF = radiofrequency, VT = ventricular tachycardia, SR = sinus rhythm, RBBB = right bundle branch block, LBBB = left bundle branch block, LA = left axis deviation, TR = tricuspid ring, Focal = focal VT, Patchy = patchy VT, LA = left atrium, VPES = ventricular programmed electrical stimulation, N/A = not applicable.
Low voltage areas (LVA) were defined as areas with bipolar electrogram voltage < 1.2 mV as proposed by Boulos et al. [17].

RF ablation
RF energy was delivered at the critical isthmus for reentrant tachycardia and at the site of earliest activation for focal tachycardia. Target sites were chosen based on catheter stability and electro-anatomical mapping data. RF energy was delivered between the tip of the mapping/ablation catheter and a 575-cm² back plate placed under the patient’s left shoulder. RF energy was delivered through a 550-kHz RF Stockert-Cordis generator in a temperature-controlled mode for 60–120 s at each ablation site. The maximal temperature target was set at 65 °C with a maximum delivered power of 75 W. Procedural success was defined as the non-inducibility of any haemodynamically tolerated VT, using the same protocol as during baseline ventricular PES.

Results

Mapping results
Procedures lasted a mean of 210 ± 93 min (range 90–360) with a mean fluoroscopy time of 23 ± 11 min (range 11–41). A total of 12 map-pable VTs were induced (Fig. 1). Eight demonstrated a reentrant mechanism: five circuits revolving around the tricuspid annulus, two in the lateral free wall and one revolving around the RVOT. The remaining four showed a focal endocardial activation pattern. No tachycardia originating from the RV apex was observed. All but three VTs had a left bundle branch block morphology. The three right bundle branch block VTs all demonstrated a peritricuspid reentry circuit. No significant differences could be demonstrated in terms of programmed electrical stimulation characteristics in relation to the different VT circuits or VT patterns.

Sinus rhythm mapping
Eight maps were obtained in sinus rhythm, with a mean number of 84 ± 29 (range 58–144) points. Low voltage areas (LVA) were observed on all these maps. Four patients (patients 1, 2, 3, 4) presented an LVA localized close to the tricuspid annulus on the lateral free wall and/or the inferior wall of the right ventricle: area 13 in Josephson’s classification [18]. The average surface of these areas was 20 ± 9 cm² (range 3.9–29.5 cm²). Their localization corresponded to the abnormal zones (thickening, akinesia, bulging) described on magnetic resonance imaging for the three patients for whom this examination was available. In two patients a line of DPs was observed at the border of the LVA and the normal tissue.

Two other patients (patients 7 and 9) had an LVA on the RV outflow tract (posterior wall and lateral wall, respectively: Josephson’s area 17). The posterior area measured 3.24 cm² and the lateral 5.72 cm².

The remaining two patients (patient 10 and 11) had multiple LVAs. Patient 10 had an LVA area on the lateral outflow tract wall and another LVA on the RV lateral free wall close to the tricuspid annulus. These LVAs had a total surface of 11.31 cm² and the peritricuspid one was matched to the abnormal (akinesia) zone described on MRI for this patient. The last patient had patchy LVAs localized on the inferior RV wall, the lateral free wall and in the outflow tract with a total surface of 50 cm².

Characteristics of VT maps
Reentrant ventricular tachycardias
Five peritricuspid VTs (mean cycle length 405 ms) were mapped. A mean number of 88 ± 49 points was taken per map. All of those VTs revolved around the tricuspid annulus in a counterclockwise direction (in left anterior oblique view). A critical isthmus bounded by the tricuspid annulus and

Figure 1 (A) Ventricular tachycardia mechanisms and acute radiofrequency results. VTs = ventricular tachycardias, LAT = lateral, TR = peritricuspid, AP = peri pulmonary valve. The lowest numbers indicate the number of non-inducible VTs after ablation. (B) Results after a follow-up of 20 ± 13 months. Success = acute success group, failure = acute failure group, free = no VT recurrence documented, recur = VT recurrence, death = patient committed suicide.
a line of double potentials (DP) was identified in all of them. In the four patients for whom an SR map was available, this line of DP was located at the border of the LVA. In two patients the line could already be identified on the SR maps. The long axis of the isthmus was oriented parallel to the tricuspid annulus and the line of DP. The isthmus measured on average 46 ± 11 mm in length and 25 ± 7 mm in width. A typical example of a counterclockwise peritricuspid VT circuit is shown in Fig. 2.

Two VTs (mean cycle length 430 ms) exhibited a reentry circuit in the lateral wall. These VTs (for which, respectively, 175 and 81 points were taken) revolved on the RV lateral free wall passing through an isthmus bounded by two lines of DPs. The long axis of the isthmus was parallel to the tricuspid annulus approximately 6 cm from it. The isthmus measured on average 15 mm in length and 12 mm in width (Fig. 3). No SR maps were performed for these patients.

The last reentrant VT exhibited a circuit revolving around the RV outflow tract (RVOT) with an activation wave front rotating around the pulmonary valve in a counterclockwise fashion (superior view). The activation wave front ran through a critical isthmus (26 mm in length and 19 mm in width) delineated by the tricuspid annulus and an LVA on the posterior wall of the outflow tract.

Focal ventricular tachycardias
A mean number of 114 ± 54 points was taken for each focal VT (mean cycle length 363 ms). The site of earliest activation was localized in the RV lateral free wall in all of these patients (Fig. 4). In one patient (patient 5) no SR map was performed. In the other three (patients 9, 10 and 11) none of the focal VTs originated in an LVA were identified during SR (Table 1).

Ablation results
According to the procedural end point, the ablation was successful in nine (75%) of 12 VTs (Table 1).

Reentrant ventricular tachycardias
Ablation was performed during VT in four patients having a peritricuspid reentrant mechanism with
tachycardia termination during RF delivery. In one patient the ablation was made in sinus rhythm because of catheter instability during tachycardia. On average, 9.4 RF applications (range 1–28) were needed to achieve a continuous line transecting the critical isthmus. All peritricuspid VTs were successfully ablated.

A mean of 15 RF applications (respectively, 13 and 17) were made during VT in order to transect the critical isthmus of the lateral free wall VTs. Ablation was successful in one patient and failed in the other (VT still inducible at the end of the procedure).

Ablation of the outflow tract VT was performed during tachycardia (three RF applications) and was successful. The RF line connected the tricuspid annulus to the abnormal area in the posterior wall of the outflow tract.

Focal ventricular tachycardias

A mean of 14 RF applications (range 2–22) was performed at the site of earliest activation for the focal VTs with the tachycardia termination during the RF delivery. In two patients the VT was still inducible at the end of the procedure and these ablations were considered to be failures.

Follow-up

During a median follow-up of 36 months (range 9–50 months) one patient (patient 11) died because of a nonmedical cause (suicide). He had initially presented a focal pattern VT and the ablation had failed. Ventricular PES was performed 4–6 weeks after the ablation procedure in five of seven patients successfully ablated and without an ICD (Table 1 and Fig. 1B).

Reentrant ventricular tachycardias

Among the five peritricuspid VTs, two patients received an ICD after the ablation procedure. One patient (patient 2) recurred early after the ablation (1 week). In the second, a VT with a different ECG morphology and cycle length was induced by ventricular PES. Eleven months later he presented a spontaneous VT with a different morphology from the ablated VT. The remaining three patients had a negative ventricular PES and they were discharged without antiarrhythmic drugs.

In the patient with the successfully ablated lateral reentrant VT (patient 8) the antiarrhythmic drugs (Amiodarone and Nadolol) were not discontinued.
after the ablation because of prior syncopal VT. Fourteen months after the ablation an episode of VT with a different cycle length from the ablated VT was recorded by the ICD. No surface ECG morphology of this VT is available.

The patient with reentry around the RVOT complained of short (up to 15 min) episodes of palpitations. Ventricular programmed stimulation was not performed and no ECG was available during these episodes. He received beta blocking agents that resulted in relief of the symptoms.

Focal ventricular tachycardias
Ventricular PES was performed 4 months after the ablation procedure in one (patient 10) of the two patients in whom a focal VT had been successfully ablated. A VT with the same morphology but a shorter cycle length was induced. This VT was not haemodynamically tolerated and the patient subsequently received an ICD. No spontaneous VT was recorded for this patient during 37 months follow-up.

Complications
No complications related to the ablation procedure occurred.

Discussion
This report describes the electroanatomical characteristics of VT mapped in a consecutive series of patients suffering from ARVD.

In our series, eight VTs (67%) exhibited an activation pattern compatible with a reentrant circuit. Of these, five circuits (45%) revolved around the tricuspid annulus, two circuits (19%) were localized on the lateral free wall and one (9%) was a reentry circuit around the pulmonary valve. The critical isthmus of each reentrant circuit was either bounded by an LVA on one side and an anatomical barrier (tricuspid annulus) on the other side, or by two parallel lines of double potentials. The LVAs were identified during SR mapping and corresponded to the
localization of abnormal zones described by MRI in the patients in whom this examination was performed.

Four VTs (33%) exhibited a focal activation pattern and were localized on the lateral RV free wall. These VTs originated in areas of normal myocardium (normal bipolar voltage).

Radiofrequency ablation guided by mapping of the endocardial activation was acutely successful in seven of eight reentrant VTs (88%) and in two of four focal VTs (50%). All the peritricuspid VTs were successfully ablated. Among the seven reentrant VTs successfully ablated, one patient suffered from a recurrence of the originally ablated VT. Two other patients had a recurrence of a different VT.

Reentry in areas of abnormal myocardium is a possible cause of ventricular tachycardia in ARVD as previously demonstrated by entrainment mapping [7,9,10]. The majority of reentrant circuits clustered around the tricuspid annulus and the RVOT [7,10–12]. Ventricular tachycardias in patients with ARVD exhibiting a focal activation pattern have already been described by Reithmann et al. [8]. Our mapping observations are consistent with the available reports in the literature.

The repetitive initiation of these VTs by ventricular programmed stimulation suggests a reentrant mechanism. An epicardial reentrant circuit with a defined endocardial exit might explain the focal activation pattern of the RV endocardium in one third of VTs in our series. This kind of activation pattern has in fact been described in a case of nonischaemic dilated cardiomyopathy [19].

Epicardial mapping performed during surgery for VT in ARVD has demonstrated areas of low amplitude potentials and delayed activation in SR. During VT an epicardial reentrant circuit has been described [20]. These data support our explanation for the endocardial focal pattern of activation.

In our series, the VTs located at an LVA detected during SR mapping exhibited an endocardial re-entry pattern. For the VTs originating in areas of normal myocardium the circuit could not be entirely mapped on the endocardial surface of the RV.

The ECG morphology of the VTs was predominantly LBBB (nine of 12 VTs), but as previously reported, RBBB can be observed and does not exclude a right ventricular origin [21,22]. We did not observe a correlation between any specific ECG morphology and a particular activation pattern, although all RBBB morphology VTs exhibited a peritricuspid circuit.

Limitations

Our data represent a single-centre experience and cannot be generalized.

The mapped VTs were haemodynamically well tolerated and, thus, we cannot infer that the mechanisms of unstable VTs in ARVD are the same.

Another limitation is that a follow-up PES was not systematically performed after RF ablation to assess the short-term efficacy of the procedure.

Conclusions

Electroanatomical mapping provides a helpful tool in reconstructing the VT circuits in patients with ARVD and haemodynamically stable VTs. LVAs can provide a clue as to the potential mechanism of the VT. RF ablation guided by the mapping results is feasible, especially in patients exhibiting a re-entrant mechanism of their VT.

References


