Tachycardia-Related Channel in the Scar Tissue in Patients With Sustained Monomorphic Ventricular Tachycardias

Influence of the Voltage Scar Definition

Angel Arenal, MD; Silvia del Castillo, MD; Esteban Gonzalez-Torrecilla, MD; Felipe Atienza, MD; Mercedes Ortiz, PhD; Javier Jimenez, MD; Alberto Puchol, MD; Javier Garcia, MD; Jesus Almendral, MD

Background—Endocardial mapping before sustained monomorphic ventricular tachycardia (SMVT) induction may reduce mapping time during tachycardia and facilitate the ablation of unmappable VT.

Methods and Results—Left ventricular electroanatomic voltage maps obtained during right ventricular apex pacing in 26 patients with chronic myocardial infarction referred for VT ablation were analyzed to identify conducting channels (CCs) inside the scar tissue. A CC was defined by the presence of a corridor of consecutive electrograms differentiated by higher voltage amplitude than the surrounding area. The effect of different levels of voltage scar definition, from 0.5 to 0.1 mV, was analyzed. Twenty-three channels were identified in 20 patients. The majority of CCs were identified when the voltage scar definition was ≤0.2 mV. Electrograms with ≥2 components were recorded more frequently at the inner than at the entrance of CCs (100% versus 75%, P≤0.01). The activation time of the latest component was longer at the inner than at the entrance of CCs (200±40 versus 164±53 ms, P≤0.001). Pacing from these CCs gave rise to a long-stimulus QRS interval (110±49 ms). Radiofrequency lesion applied to CCs suppressed the inducibility in 88% of CC-related tachycardias. During a follow-up of 17±11 months, 23% of the patients experienced a VT recurrence.

Conclusions—CCs represent areas of slow conduction that can be identified in 75% of patients with SMVT. A tiered decreasing-voltage definition of the scar is critical for CC identification. (Circulation. 2004;110:2568-2574.)

Key Words: catheter ablation ■ mapping ■ coronary disease ■ tachycardia, ventricular

Slow-conduction areas are part of the substrate of the reentrant circuits in most sustained monomorphic ventricular tachycardias (SMVTs) occurring in patients with remote myocardial infarction. These areas are identified by the recording of presystolic electrograms during tachycardia.1-6 Human studies have demonstrated that these slow-conduction areas take place in narrow bundles of viable tissue that function as conducting channels (CCs) bounded by scarred tissue.7,8 Those CCs that are located in the endocardium are identified during tachycardia.9 The identification of these CCs before induction of VT may facilitate mapping and/or ablation of tolerated and unmappable VT. Scar mapping based on pacing threshold has been used for the identification of nonconducting tissue that borders viable myocardium during sinus rhythm (SR).10 To simplify the identification of these CCs during SR, we hypothesized that conducting bundles should have larger voltage amplitude than nonconducting scar. Therefore, voltage maps of the left ventricle might show channels inside the scar, provided that an adequate adjustment of voltage definition of nonconducting scar is used. Nevertheless, for CC identification, the voltage limits of scar as a nonconducting tissue are not defined yet; the limit of 0.5 mV differentiates contractile and noncontractile areas11,12 but not bundles of viable myocardium. A single cutoff voltage amplitude may not be feasible in all cases, because voltage of the electrograms at sites at which radiofrequency (RF) ablation was effective had a wide range.13-15 Therefore, analysis of different voltage levels of scar definition may be required for identification of CCs in each patient.

Recently reported data suggested that electrograms with isolated delayed components (E-IDCs) and multicomponent electrogram, highly predictive of the central isthmus of reentrant circuits,14 are more frequently detected during right ventricular apex (RVA) pacing than during SR.15 Because voltage amplitude is similar during both rhythms,14 we analyzed only maps obtained during RVA pacing.

In conclusion, the purposes of this study were (1) to assess the possibility of identifying channels inside the scar using a tiered decreasing-voltage scar definition; (2) to verify the function of channels as slow-conducting zones by means of the analysis of electrogram characteristics, activation se-
sequence, and response to pacing inside the channel; and (3) to establish the relation of these channels with documented or induced VT.

**Methods**

**Population**

In this protocol, approved by the Research and Ethical Committee of our institution, we included 26 patients (68±9 years old; left ventricular ejection fraction, 31±10%) with remote myocardial infarction and documented SMVT (VT cycle length, 374±59 ms) who underwent a 3D mapping–guided RF ablation. Ten patients were taking amiodarone or sotalol.

**Study Protocol**

**Electrophysiological Study**

After written informed consent was obtained, the electrophysiological study was performed in the postabsorptive state (Figure 1). At least 2 to 3 quadripolar catheters were placed at the right atrium, His bundle area, and RVA or right ventricular outflow tract. The distal pair of electrodes was used for pacing and the proximal pair for local electrogram recording. Intracardiac recordings were filtered at 30 to 500 Hz and displayed simultaneously with 4 to 6 ECG leads at 100 to 200 mm/s on a physiological system recording (Midas, Hellige Biomedical or RPM Boston Scientific). Stimulation was performed with a programmable stimulator (UHS-20 Biotronik) set to deliver rectangular pulses of 2-ms duration at twice diastolic threshold. Programmed stimulation was performed to induce VT through triple extrastimuli at 2 RV sites.

**Left Ventricle Mapping**

Endocardial mapping was performed during RVA pacing at 600 ms. Mapping and ablation was performed using the CARTO (Biosense, Inc) magnetic mapping system with the Navistar catheter. The Navistar bipole consists of a 4-mm-tip electrode and a 2-mm-ring electrode separated by 1 mm of spacing. Bipolar electrograms were filtered at 30 to 400 Hz and displayed at 100 mm/s; peak-to-peak amplitude was measured automatically.

To identify CCs and E-IDCs, multiple sites were explored to obtain a filling threshold of 10 mm in the zone of interest within the low-voltage area, so the distance between mapped sites was ≤1 cm. The Carto system displays the amplitude of bipolar electrograms as voltage maps, automatically selecting the largest component. Five maps were displayed using the following lower and upper limits in the color range that represents the electrogram voltage amplitude: 0.10/0.11, 0.20/0.21, 0.30/0.31, 0.40/0.41, and 0.50/0.51 mV. Activation maps were reconstructed taking the peak of the largest deflection of every single electrogram as the local activation time. To define the activation sequence inside the CC when multiple-component electrograms were recorded, the local activation time at each site was referred to as the latest component, and the difference between entrance and inner local activation time was measured.

**VT and CC Relation**

To define the relation of CCs and E-IDCs with documented or induced VTs, pace-mapping from the CCs and E-IDCs and their spatial relationship with the sites characterized by (1) mid-diastolic electrograms during VT, (2) concealed entrainment during VT, and (3) effective RF ablation were analyzed. After an RF pulse terminated a VT, RVA pacing was performed to incorporate this ablation site to the voltage map and establish the relation to the CCs.

We assumed a relationship between CCs or E-IDCs and clinical or induced VT when the following criteria were met: (1) noninducible VT: pace-mapping reproducing the clinical QRS VT and stimulus to QRS interval (S-QRS) ≤50 ms; (2) inducible poorly tolerated VT: presence of a presystolic or mid-diastolic electrogram and a difference ≤30 ms between the interval electrogram-QRS (E-QRS) on VT and the stimulus-QRS (S-QRS) when pace-mapping was similar; (3) in well-tolerated VT: presence of presystolic or mid-diastolic electrogram, concealed entrainment, and a first postpacing interval–VT cycle length difference <30 ms. When RF pulses terminated a VT, we considered a VT-related channel if the successful ablation site was <5 mm distant from any part of the channel.

**RF Ablation Procedure**

The RF energy, from a RF Stockert-Cordis generator, was delivered in a temperature-controlled mode for 60 to 120 seconds at each ablation site with a maximal temperature target of 60°C and 50 W of maximum power delivered.

The end points of the procedure were (1) inducibility suppression of induced clinical VT and/or (2) disappearance of clinical VT-related CCs or E-IDCs in those patients with unmappable VT. Heparin was infused throughout the whole procedure. For those patients in whom VT was not inducible or the inducibility suppression was not tested, implantation of an ICD was advocated.
Electroanatomic maps were constructed with 142 patients; VTR-CCC indicates VT-related complete CCs; VTR-ICC, VT-related incomplete CCs; VTNR-CCC, VT-nonrelated complete CCs; and VTNR-ICC, VT-nonrelated incomplete CCs.

**Definitions**

**Complete CC**
Complete CC was defined as a corridor of continuous electrograms differentiated from the surrounding scar tissue by a higher voltage. These electrograms must be surrounded by 2 scar areas or 1 scar area and the mitral annulus and connected to normal myocardium by at least 2 sites.

**Incomplete CC**
Incomplete CC was defined as a CC connected to normal myocardium by only 1 site.

**Electrogram With Isolated Delayed Component**
An E-IDC was defined as an electrogram recorded in the scar tissue showing double or multiple components separated by >50 ms by very-low-amplitude signal or an isoelectric interval. During RVA pacing, the activation time of the latest component from the stimulus artifact had to be >150 ms.

**Channel Electrogram Type**

*Type I:* Electrogram with 2 or more components in which the first component is the largest in voltage amplitude.

*Type II:* Electrogram with 2 or more components in which the first component is not the largest in voltage amplitude.

*Type III:* Electrogram with more than 2 components of similar amplitude.

*Type IV:* Electrograms with only 1 component.

Electrogram types I, II, and III are considered E-IDCs.

**Scar Definition**
We used a tiered decreasing voltage scar definition. Scar was defined sequentially by the areas with a voltage amplitude ≤0.5, 0.4, 0.3, 0.2, and 0.1 mV.

**Statistical Analysis**
Values are expressed as mean±SD. Comparisons were performed with the use of the Student t test and Fisher exact test. A probability value of P=0.05 was considered significant.

**Results**

Electroanatomic maps were constructed with 142±53 points. In this group of 26 patients, 23 CCs were identified in 20 patients; therefore, at least 1 CC was detected in 77% of cases. Twenty CCs in 20 patients were related to at least 1 clinical or induced VT. The CC dimensions were 23±11 mm in length and 9±3 mm in width. Figure 2 shows the voltage limit for scar definition at which CCs were identified; the majority of CCs were identified when the scar voltage was set at ≥0.2 mV (Figure 3). Seven CCs remained incomplete despite the use of all levels of voltage scar definition (5 associated with anteroserptal myocardial infarction), 7 additional CCs were incomplete at the highest voltage and complete at the lowest voltage, and the remaining 9 CCs were always complete. In the 12 patients with strictly inferior myocardial infarction, 5 of 9 identified CCs had a submural location between the scar and the mitral annulus;$^{18}$ in these cases, the CCs were always complete, extending from the septum to the lateral wall (Figure 3).

**Electrophysiological Characteristics and Activation Pattern of CCs: Comparison Between Entrance and Inner Electrograms**

To characterize the CCs, 79 electrograms were analyzed, 37 at the entrance and 42 at the inner part (Figure 4). We considered inner electrograms to be those located 0.5 to 10 mm from the entrance. Electrograms <5 mm apart were not evaluated separately; we usually analyzed 1 electrogram at each entrance and 2 at the inner part. Single-component electrograms (type IV) were recorded only at the entrance, and multiple-component electrograms (type III) were more frequently recorded at the inner part, P<0.0001 (Figures 3, 5, and 6). No differences were found in the activation time when the first component was compared between entrance and inner electrograms (115±47 versus 108±43 ms). Nevertheless, the last component of the inner electrograms was recorded later than the last component of entrance electrograms (200±40 versus 164±53 ms, P<0.01 (Figures 3, 5, and 6), consistent with an activation sequence from the entrance to the inner part of the channel.

The voltage of the largest deflection was higher at the entrance electrograms than at the inner electrograms (0.5±0.3 versus 0.36±0.19 mV, P=0.002). The largest deflection coincided with the first component in 28 of 37 electrograms recorded at the entrance of the channel but in only 13 of 42 electrograms recorded at the inner part (P<0.0001).

**Pacing From CCs**

Pacing was attempted from the central inner part of the channel. The stimulus-QRS interval from the channel was 110±49 ms (Figures 5 and 6). Of 15 complete VT-related channels, VT was reproduced in 12 leads in 7, fewer than 12 in 4, and not done in 4, because of capture failure in 2 and nonstability in another 2. Of 5 incomplete VT-related channels, VT was reproduced in 12 leads in 3, 11 leads in 1, and in 1 CC no local capture was obtained.

**VT and Channel Relationship**

The induced channel-related VT cycle length was 365±77 ms; in 12 episodes, the VT cycle length was ≤350 ms and in 9, ≤320 ms (Figures 5 and 6). Seven channels were associated with 7 clinical VTs by activation-entrainment mapping, 2 channels with 2 nonclinical induced VTs by activation-entrainment mapping, 7 channels with 7 clinical VTs by activation-entrainment and pace mapping, and 2 channels...
with 2 clinical VTs only by pace mapping. One channel was associated by pace mapping with a clinical VT and with a nonclinical VT by activation-entrainment mapping. One channel was associated with a clinical VT and with a nonclinical VT by activation mapping. The remaining 3 channels were not associated with any induced or clinical VT.

**Submitral CCs**

Clinical VTs associated with submitral channels presented the morphologies described by Wilber et al., left bundle-branch block with left superior axis (rS in V1 and aVR and R in V6, I, and aVL) in 4 patients or a right bundle-branch block and right superior axis morphology (R in V1 and aVR, QS in V6, I, and aVL) in 1 patient; similarly induced VT presented predominantly a left bundle-branch block morphology, 5 of 6 VTs. In only 2 cases did pacing from the channel reproduce the VT morphology exactly. Pacing from submitral channels usually gave rise to alternating morphology between left and right bundle-branch block morphology (Figure 6). The remaining VTs were related to channels by activation/entrainment mapping.

**RF Ablation Results**

A mean of 14±8 RF lesions were applied per patient. RF pulses were delivered over electrograms located in all VT-related channels in 20 patients, 11±6 RF lesions per channel. In 14 channels of 14 patients, the ablation was performed during tachycardia that terminated in all cases but in 1 remained reinducible despite multiple lesions. In the remaining 6 VT-related channels, lesions were delivered during SR because of noninducible documented VT in 2 and nontolerated VT in 4. In 3 of these 4 patients, VT inducibility was suppressed, and it was not tested in 1. In 2 of these 20 patients, additional RF pulses were delivered over E-IDCs not channel related.

In the remaining 6 patients, VT ablation was anatomically guided toward VT-related E-IDCs.15

After ablation, previously inducible VT became noninducible in all but 1 patient, ventricular fibrillation was induced in 2 patients, and a fast nonclinical VT in 8 patients (VT cycle length, 280±50 ms). No complications were observed during the procedure.

After the RF ablation procedure, 19 patients were discharged with an ICD. In 3 patients with VT inducibility
suppression and ejection fraction >35%, ICD implantation was not advised. Despite the fact that some VTs were not inducible or nonsuppressed, 3 patients were not considered to be candidates to receive an ICD on the basis of very short life expectancy. One patient with noninducible VT refused ICD implantation.

Follow-Up
During a follow-up of 17±11 months, there were only 7 recurrence episodes in the whole group. Five patients experienced a single VT recurrence, with cycle length shorter (≤310 ms) than the ablated VT in 4 patients. One patient experienced 2 VT recurrences before dying of congestive heart failure. Two additional patients died of congestive heart failure and 2 of noncardiac causes without any VT recurrence.

Discussion
We describe a new method of identifying CCs during RVA pacing that can be feasibly applied for RF ablation procedures in patients with mappable and unmappable VT.

Figure 5. Top, Right anterior view of septal and anterior walls of left ventricle showing an incomplete channel related to an anterior myocardial infarction. Left, electrograms recorded at sites identified by white numbers are shown. From 1 to 3 electrograms were recorded in scar tissue; 4 is electrogram recorded at entrance and 5 and 6 electrograms from inner part. Activation time of last component (*) of inner electrograms was longer than last component of entrance electrogram; regarding first component, no differences were observed between entrance and inner electrograms. Lack of differences might be explained because first component of inner electrograms is a far-field signal from entrance, as suggested by lower voltage amplitude. Blue dot is site considered as entrance of channel, and green dot shows site from which pace-mapping was made (A, bottom, exactly reproduced induced VT with a long stimulus QRS interval (B, bottom). Dark blue dot represents site at which a mid-diastolic electrogram was recorded during tachycardia (C, bottom), and RF ablation stops VT.
We hypothesized that these CCs of surviving bundles must have a larger voltage than the surrounding scar tissue; therefore, a careful step-by-step adjustment of voltage scar definition could identify these bundles. Our data demonstrate that the scar tissue is not homogeneous and that there are areas of larger voltage shaped like a corridor of continuous electrograms, which connect with the tissue that surrounds the scar. The voltage scar definition is determinant for a correct identification of CCs. A voltage level of 0.5 mV precludes the discrimination of electrograms that identify slow-conduction area in most cases, probably because the amplitude of these electrograms is usually $\leq 0.5$ mV, as observed at sites at which the RFA was effective.\textsuperscript{13–15} Because the voltage of RFA target sites fluctuates over a wide range, the only way to detect these sites is to compare different voltage scar definitions.

Several data support these channels as being protected areas of slow conduction and not an artifact: (1) local capture during pacing with a long stimulus-QRS interval; (2) multiple-component electrograms are more frequently recorded at the inner part than at the entrance, where single-component electrograms are currently recorded; and (3) the activation time of the last component is longer at inner than at entrance electrograms, suggesting propagation from the entrance to the inner part.

**Channels and VT Relation**

Although no activation mapping during tachycardia was systematically obtained, there is consistency in the relation of the VT circuit and CC that is supported by pace and activation mapping and the relation to the effective RF lesion. The fact that pace mapping from CCs did not reproduce the VT.
morphology in some cases can be explained by the exit from the channel through more than 1 connection to normal myocardium.

Submitral Channels
All patients with inferior myocardial infarction and a documented VT showing the characteristic QRS morphology presented a CC between the mitral and inferior scar that always extended from the lateral to the septal wall. In these cases, the scar, when set at 0.5 mV, reaches the mitral annulus in all cases, meaning that the tissue is abnormal, not an area of normal myocardium not affected by the infarction.

Advantages of Ablation of the Isthmus
CC recognition on voltage maps is a fast method for VT substrate identification; this method is feasible to the majority of VT patients. Eighty-six percent of channels are related to induced or clinical VT, which implies high arrhythmogenic potential. CCs are relatively small compared with the scar areas; therefore, this method could permit focus on the diagnostic techniques and ablation to defined areas; in those patients without CCs on the voltage map, a more time-consuming approach could be used. This procedure would allow the ablation of some nontolerated or noninducible VT not approachable by conventional entrainment mapping. Regarding the ablation guided by E-ICD searching, what additional advantage is afforded by channel identification? Although E-IDCs were recorded in all CCs, the identification of the channel and its narrowest part may reduce the extension of VT substrate to be ablated.

Limitations
CCs were not found in 24% of patients. This limitation can be explained by the following possibilities: (1) scar areas might be too narrow to be detected by our mapping system, (2) the boundaries of the isthmus could be functional lines of block not detected during RVA pacing or SR, or (3) the dispersion of voltage in some scar areas may appear only when activated at the VT rate. The electrode size may determine the extension of the local recording area, thus influencing the discrimination capability for CC identification; our results therefore cannot be extrapolated to a size of tip electrode different from 4 mm. Some fast nonclinical VT seems not to be related to these channels. Although left ventricular ejection fraction was not systematically evaluated after the ablation procedure, a reduction in the ventricular function seems very unlikely, because all lesions were located inside the dense scar.

References
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