Isthmus Characteristics of Reentrant Ventricular Tachycardia After Myocardial Infarction

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Background—The reentrant mechanism of postinfarct ventricular tachycardia (VT) has been documented by surgical mapping analysis, but little is known about postinfarct VT circuits and the characteristics of their related protected isthmus with the use of 3D catheter mapping systems.

Methods and Results—A 3D electroanatomic mapping was performed in 21 patients with well-tolerated, postinfarct, sustained VT. In total, 33 episodes of tachycardia (mean cycle length 432±74 ms) were induced and mapped. Complete maps demonstrated macroreentrant circuits with 1 loop (n=8) or 2 loops (n=25) rotating around a protected isthmus bounded by 2 approximately parallel conduction barriers that consisted of a line of double potentials, a scar area, or the mitral annulus. A total of 26 critical isthmus were identified for the 33 VTs mapped, with the same isthmus being shared by 2 to 4 different tachycardic morphologies in 5 patients. On average, isthmus were 31±7 mm long (ranging from 18 to 41 mm) and 16±8 mm wide (ranging from 6 to 36 mm) and harbored diastolic electrograms. The isthmus axis was oriented parallel to the mitral annulus plane in perimitral circuits and perpendicular to the mitral annulus plane in all other circuits. Linear radiofrequency ablation performed across the most accessible part of the isthmus prevented the recurrence of tachycardia in 19 patients (90%) with a follow-up at 16±8 months.

Conclusions—Detailed 3D electroanatomic mapping is helpful in reconstructing postinfarct VT circuits and in defining the characteristics of their related protected isthmi. The wide range of isthmus width values supports the need of linear radiofrequency lesions to eliminate the reentrant substrate of postinfarct VTs. (Circulation. 2002;105:726-731.)

Key Words: catheter ablation ■ mapping ■ myocardial infarction ■ tachycardia

In the thrombolytic era, life-threatening ventricular arrhythmias are reported at an annual incidence of 2% to 5%, after myocardial infarction (MI). These are often treated with an implantable defibrillator in the presence of ventricular fibrillation or poorly tolerated ventricular tachycardia (VT), whereas well-tolerated VT may be considered for mapping and endocardial radiofrequency (RF) ablation.

Several studies3–4 have reported on the RF ablation of VT in post-MI patients with the use of the so-called conventional electrophysiological approach to identify the critical part of the arrhythmia. A reentrant mechanism of the VT is usually suspected when the procedure is successful, but the VT circuit is not reconstructed by the conventional approach. The aim of the present study was to depict postinfarct VT circuits and the characteristics (location and size) of their related protected isthmus by use of an electroanatomic mapping system.

Methods

Patients

Between April 1998 and March 2001, 21 patients (19 men, mean age 66±9 years) admitted with a sustained monomorphic VT and history of a remote anterior (n=6), inferior (n=12), or both anterior and inferior (n=3) MI underwent a 3D mapping–guided RF ablation performed during VT. Left ventricular ejection fraction (mean 0.34±0.08, ranging from 0.17 to 0.50) was measured by echocardiography, which detected an anterior aneurysm in 4 patients and an inferior aneurysm in 5. Ten patients had been previously equipped with an automatic implantable cardioverter-defibrillator (ICD) and were referred to our institutions because of a VT that either circumvented the ICD algorithms or was resistant to antitachycardic pacing. Six patients were admitted because of an incessant VT (all 6 were ICD bearers).

This population was selected from 35 consecutive patients investigated in our laboratories during the same period for RF ablation of post-MI VT. The 21 selected patients are those with clinically well-tolerated VTs exhibiting endocardial circuits on the 3D maps. The 14 remaining patients did not undergo a 3D-mapping investigation (n=5), had a 3D mapping performed during sinus rhythm only (n=6), or had no evidence of an endocardial reentrant VT mechanism (n=3) from either conventional electrophysiological pacing maneuvers or 3D mapping data.

Electrophysiological Study, Mapping, and Ablation

Electrophysiological study, mapping, and catheter ablation were performed after informed consent was obtained. Implanted defibrillators were switched off during the procedure.
Briefly, a bipolar catheter was inserted via the femoral vein and positioned at the right ventricular apex. With this catheter, ventricular programmed electrical stimulation (PES) was used to induce VT, with the application of up to 3 extrastimuli during spontaneous rhythm and during paced rhythm (600-ms and then 400-ms basic cycle length). PES was delivered through an external stimulator (Biotronik UHS 20, Biotronik Inc) with a 2-ms pulse width at twice the diastolic threshold. Failure to obtain the clinical VT promoted the same protocol in the right ventricular outflow tract.

The 3D mapping started with the onset of VT. Left ventricular mapping was performed with an 8F or 7F mapping/ablation catheter (NAVI-STAR, Cordis-Webster, Johnson & Johnson) that had a 4- or 8-mm-tipped electrode. Access to the left ventricle was achieved retrogradely across the aortic valve or, in 2 patients, through transesophageal septal puncture.

Conventional mapping, including entrainment maneuvers and postspacing interval analysis, was performed after the induction of clinical VT. Pacing sites with a postspacing interval not exceeding the cycle length by >20 ms were considered to be part of the circuit. Pacing for entrainment resulted in either interruption of the VT or transformation into another morphology in 5 of the 8 initial patients. The clinical VT could be induced again but with considerable effort in 4 of these 5 patients. Subsequently, conventional mapping was not performed systematically.

Systemic anticoagulation was achieved with heparin (initial bolus of 50 U/kg IV followed by 1000 to 2000 U per hour) throughout the procedure. Sedation was obtained with 10 mg IV nalbuphine, with incremental doses at 5 mg as necessary.

**3D Mapping Technique**

The CARTO nonfluoroscopic electrophysiological mapping and navigation system (Biosense, Johnson & Johnson) has been recently described: the ultralow magnetic field generated by a triangular location pad under the patient’s bed accurately determines the location of the tip of an 8F or 7F mapping/ablation catheter tip (NAVI-STAR, Cordis-Webster, Johnson & Johnson). The spatial reference is another NAVI-STAR catheter externally fixed to the back of the patient. The electrical reference was chosen as a morphologically stable and regular right ventricular electrogram that was obtained from either an endocardial or surface lead, with the choice determined by a QRS complex with a sharp apex and a strong positive (or negative) deflection during VT. The width of the window of interest varied from one VT map to another, inasmuch as it was correlated with the VT cycle length with the following formula: window of interest width = VT cycle length – 20 ms. The middle of the window of interest was selected to coincide with the electrical reference. The local activation time for each endocardial position under the mapping catheter was calculated as the interval between the electrical reference and the peak deflection of the mapping bipolar electrogram. In case of double potentials, the earliest peak deflection of the doublet was used. Long-duration fractionated electrograms were marked to the highest peaklet.

The left ventricle was plotted during the induced clinical VT by dragging the mapping catheter over the endocardium. The protocol was used to map the right ventricle when the left interventricular septum was close to the core of the VT circuit. Infarct regions were sought first, and data points were acquired around these areas. Refining the area under investigation relied on the usual clinical indicators, such as sinus rhythm analysis, echocardiography, and VT morphology on the 12-lead ECG. More data points were acquired in the zones defined as scarified, with low-amplitude potentials, with diastolic electrograms, or with double potentials (as defined below). These areas were probed because they are important for the identification of the reentrant circuit. The mapping procedure was terminated when a density of points was achieved that was sufficient enough to allow an understanding of the VT circuit. The resulting reentrant circuit was considered to be the spatially shortest route of unidirectional activation encompassing a full range of mapped activation times (>90% of the tachycardic cycle length) and returning to the site of earliest activation.

**RF Ablation**

Identification of the ablation site was based on analysis of the 3D map. RF current was delivered from a mapping catheter with a 4-mm or 8-mm tip. The anode was a 575-cm2 back plate placed under the patient’s left shoulder. RF ablation was performed with a 550-kHz RF Stockert-Cordis generator. The RF energy was delivered in a temperature-controlled mode for 60 to 120 seconds at each ablation site with a maximal temperature target of 65°C for 4-mm tips (55°C for 8-mm tips) and 75 W of maximum power delivered.

The procedural end point was the ablation of clinically well-tolerated VTs only. A successful ablation was defined as the termination of well-tolerated inducible VTs with the inability to re-induce any of them.

**Definitions**

Scar areas were defined from an electrophysiological standpoint as electrically silent areas that displayed neither distinguishable nor repetitive electrogram patterns with amplitudes <0.05 mV. These areas were presumed to be conductive.

Double-potential electrograms were defined by 2 distinct ventricular bipolar potentials with peaks separated by an interval of at least 50 ms and with a return to baseline between the 2 potentials.

During VT, an isthmus was defined as a conductive myocardial tissue delineated by nonconductive tissue. This nonconductive tissue could be either a line of double potentials or a scar area. It could also encompass an anatomic obstacle, such as the mitral valve.

On the basis of VT circuit analysis, an isthmus that the depolarization wavefront must cross to perpetuate the tachycardia was defined as the critical VT isthmus. As a consequence, ablation of the critical VT isthmus should interrupt the tachycardia and prevent its re-inducibility.

**Management After Ablation**

After ablation, patients were monitored for 72 hours by telemetry. Transesophageal echocardiography was performed within 2 days after ablation. Patients were then discharged and were followed on an outpatient basis with clinical evaluation and 24-hour Holter recordings performed regularly. When available, the ICD Holter function was permanently activated.

**Results**

**Mapping Results**

In the 21 patients, complete maps were obtained for 33 VT morphologies (mean VT cycle length 432±74 ms, ranging from 320 to 550 ms). Varying macroreentrant circuits were identified in all 21 patients. A mean of 144±69 points covering 93±4% of the arrhythmic cycle length was recorded.

**Scar Areas and Linear Zones of Block**

Scar areas were noted in 8 patients. These electrically silent areas had surfaces ranging from 4.1 to 24.2 cm2. Thirty-seven zones of block (mean 1.8±0.9 per patient) were identified. Each zone corresponded to a continuous, 32±9-mm line of double potentials. Five (14%) of these 37 lines of block (LOBs) were recorded close (within 20 mm) to the mitral valve annulus (MVA) and were oriented parallel to the MVA plane. The remaining 31 LOBs were recorded >20 mm from the MVA and exhibited an orientation grossly perpendicular to the MVA plane.

On the basis of echocardiographic data, the scar areas and the LOBs coincided with an infarct zone. (Examples of LOBs and scar areas are given in Figures 1, 2, and 4. The topography of the different LOBs and scar areas is given in Figure 3).
Characteristics of VT Circuits

A single-loop reentrant circuit (Figure 1) was identified in 8 (24%) of 33 VTs. It was rotating (left anterior oblique view) around the MVA annulus counterclockwise in 2 cases and clockwise in 2 other cases and was bounded by an LOB (n=3) or a scar area (n=1) defining an isthmus. In the remaining 4 VTs, the circuits rotated around a scar area (n=1) or around an LOB (n=3) and were bounded by another scar area (n=3) or another LOB (n=1) defining an isthmus located in different regions of the left ventricle.

Twenty-five (76%) of 33 VTs exhibited a double-loop figure-8 reentrant circuit (Figure 2), with 2 loops rotating in opposite directions around barriers delineating an isthmus. The approximately parallel barriers consisted of at least 1 LOB, with either another LOB (n=20), a scar area (n=2), or the MVA (n=3) as an anatomic barrier. Three of the 25 double-loop VTs were perimital, with 1 loop rotating around the mitral annulus and the second loop rotating around an LOB (Figure 2). The core center of the remaining 22 VTs was located in different regions of the left ventricle. Figure 3 summarizes the different circuits identified in relation to their topography.

Characteristics of the Critical VT Isthmi

A total of 26 critical VT isthmi were identified for the 33 VTs mapped. A given isthmus was associated with a single VT morphology in 19 patients, but the same critical isthmus was shared by 2 to 4 different VT morphologies in 6 patients (Figure 4). On average, isthmi were 31±7 mm (ranging from 18 to 41 mm) long and 16±8 mm (ranging from 6 to 36 mm) wide.

The critical VT isthmus encompassed diastolic potentials only. The entrance of the isthmus showed early-diastolic electrograms, progressing toward middiastolic and exiting with late-diastolic electrograms (Figure 2). Critical isthmi corresponded to a slow conduction zone, with 57% to 81% of the VT cycle lengths being recorded in these paths. Conduction velocity in the critical isthmus ranged from 6.9 to 19.5 cm/s (mean=12.9±3.5 cm/s) and was not correlated with isthmus width.

A double-potential line was at least one of the boundaries of the isthmi in the great majority of the cases (31 of 33 VTs). Therefore, the isthmus axis had the same orientation as the double-potential lines, ie, parallel to the MVA plane in perimital circuits and perpendicular to the MVA plane in anteropical, inferolateral, and septal circuits.

Ablation Results

One session (16 patients), 2 sessions (3 patients), 3 sessions (1 patient), or 4 sessions (1 patient) were required (mean 1.4±0.8, median=1). The mean cumulative procedure and fluoroscopy durations were 265±87 (range 120 to 540) minutes and 32±17 (range 5 to 66) minutes per patient.

A mean number of 7.9±8.9 RF applications per isthmus (range 1 to 35, median 4) resulted in the interruption and no further inducibility in 32 (97%) of the 33 VTs mapped. Multiple RF lesions were required in the great majority of cases. Indeed, of the 32 successfully ablated VTs, only 6

Figure 1. Illustration of single-loop VT circuit (VT cycle length 500 ms). Top left, VT activation sequence map as shown by CARTO. Right, Propagation map (A to D) during VT showing clockwise progression of depolarization wave front (red) around double-potential barrier (yellow line). Bottom left, ECG strip showing VT interruption during single RF pulse delivery.

Figure 2. Illustration of figure-8 VT circuit (VT cycle length 540 ms). Top left, Documentation of early-diastolic electrograms, progressing toward mid-diastolic and exiting with late-diastolic electrograms within VT isthmus. Top right, VT activation sequence map as shown by CARTO. Bottom, ECG strip showing VT interruption during single RF pulse delivery.
could be terminated with a single RF pulse. VTs could be terminated after a mean number of 6.1 ± 6.4 RF pulses, with additional applications being required to prevent re-inducibility. The 7 perimitral VT circuits were ablated by an RF line connecting the MVA to the second boundary of the critical isthmus, ie, a scar area in 2 cases and an LOB in 5 cases (see Figure 3C). The remaining 25 VTs were ablated by an RF line transecting the critical VT isthmus. One patient with an inferolateral figure-8 VT circuit (extreme left schematic representation in Figure 3D) unsuccessfully underwent an RF transection of the identified critical isthmus.

According to the procedural end point, the ablation was successful in 20 (95%) of 21 patients. A fast, poorly tolerated VT could be induced at the end of the procedure in 8 of these 20 patients (6 of them were ICD bearers). The fast-induced VT was nonclinical in the 2 patients not equipped with an ICD at admission.

Postprocedural ICD Implantation
In total, 3 patients received an ICD after the ablation procedure. This was due to a procedural failure in 1 patient and to a successful ablation followed by an inducible, nonclinical, poorly tolerated VT in 2 patients. Eight patients were discharged without ICD. Postprocedural PES could not induce any VT in those patients.

Follow-Up
At a mean follow-up of 16 ± 8 (range 4 to 34) months, 17 patients remained free of any episode of VT/ventricular fibrillation, which was confirmed by ICD interrogation in 9 of the patients; the remaining 8 patients were not equipped with an ICD. Two other ICD bearers presented only syncopal fast VT episodes during the follow-up period. In the remaining 2 patients (1 successfully and 1 unsuccessfully ablated), recurrence of the mapped and ablated VT was documented respectively 6 and 2 months after the procedure. Two patients died during the follow-up period; these deaths were documented in hospital as being attributed to nonarrhythmic heart failure.

Discussion
The present study describes the characteristics of post-MI isthmus-related monomorphic VTs. The critical VT isthmus was bounded by 2 approximately parallel conduction barriers that consisted of a line of double potentials, a scar area, or the MVA. The endocardial reentrant VT loops had rotated around the isthmus boundaries and were propagated slowly through the critical isthmus, which harbored diastolic potentials and measured 31 mm long by 16 mm wide on average. The axis of a critical isthmus was oriented parallel to the MVA plane in perimitral circuits and perpendicular to the MVA plane in all other circuits.

The ablation tactic was used to transect the critical isthmus in the most convenient area, targeting the narrowest portion of the isthmus when allowed by catheter positioning and stability. This strategy was acutely successful in 32 (97%) of 33 VTs, with a median number of 4 RF pulses delivered per
isthmus. To join the isthmus boundaries, RF lines were perpendicular to the MVA plane in perimitral circuits and parallel to the MVA plane in all other circuits.

Literature in the late 1970s and early 1980s had suggested that post-MI VTs were based on a reentry mechanism involving mainly endocardial regions on the border of infarct tissue. In the 1990s, computer modeling and animal and human studies have demonstrated that such circuits contain a narrow isthmus of viable tissue and slow conduction that forms a critical part of the VT reentrant circuit and is bounded by infarcted tissue or anatomic barriers, such as the MVA. Our mapping observations are consistent with all these reports in the literature. In addition, the present study is the first to describe the characteristics of the critical isthmus, especially in terms of dimensions and orientation in relation to left ventricular anatomy.

Localization of vulnerable regions of post-MI VT circuits can be guided during endocardial catheter ablation by fractionated electrograms, middiastolic electrograms, and entrainment with concealed fusion. Despite improvement in so-called conventional electrophysiological techniques, many VTs cannot be ablated successfully, and clinical success remains limited compared with that obtained for other arrhythmias. Among factors that may explain this limited clinical success, one could hypothesize that a focal RF ablation may not be as appropriate as a linear RF ablation in postinfarct VTs. Indeed, Friedman et al first reported on the use of an electroanatomically guided RF linear lesion to eliminate a postinfarct VT by transecting its critical isthmus in a patient who had previously undergone an unsuccessful focal ablation. Our mapping and ablation data are consistent with this case report of a perimitral VT circuit as depicted in Figure 3C.

Study Limitations

In the present study, mapping was performed during VT only. Therefore, it is not possible to know whether a given line of double potentials corresponds to a functional or to an anatomic conduction block.

The creation of block across a VT isthmus was not assessed by any method at the end of the ablation procedure. As a consequence, whether the different RF lines created a continuous LOB across the isthmus cannot be confirmed. Entrainment mapping was not systematically performed in the present study. Therefore, the correlation between electroanatomic data and entrainment mapping is not available.

The characteristics of the critical isthmus depicted in the present study are drawn from mappable VTs that had cycle lengths ranging from 320 to 550 ms. No extrapolation to faster VTs can be drawn from these data.

Conclusions

Detailed 3D electroanatomic mapping is helpful in reconstructing postinfarct VT circuits and in defining the characteristics of their related protected isthmuses. The wide range (6 to 36 mm) of isthmus width values supports the need of linear RF lesions to eliminate the reentrant substrate of postinfarct VTs.

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References


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