Endocardial and Epicardial
Radiofrequency Ablation of Ventricular Tachycardia Associated With Dilated Cardiomyopathy
The Importance of Low-Voltage Scars
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OBJECTIVES
The purpose of this study was to evaluate the occurrence, locations, and relationship of ventricular tachycardia (VT) to low-voltage areas in dilated cardiomyopathy (DCM).

BACKGROUND
The substrate causing monomorphic VT after infarction is characterized by regions of low-voltage (<1.5 mV) scar on electroanatomic maps. The substrate causing VT associated with DCM is less well defined.

METHODS
A total of 28 patients were studied with endocardial (26 patients) and epicardial (8 patients) electroanatomic mapping. The VT circuits were defined by entrainment or pace mapping.

RESULTS
Ventricular tachycardia was due to focal VT in 5, bundle-branch re-entry in 2, and myocardial re-entry in 22 patients (both focal and re-entry VTs in 1 patient). All patients with myocardial re-entry had endocardial (20 of 20 patients) and/or epicardial (7 of 7 patients mapped) scar. Most (63%) endocardial scars were adjacent to a valve annulus. Of the 19 VT circuit isthmuses identified, 12 were associated with an endocardial scar and 7 with an epicardial scar. All myocardial re-entrant VTs were abolished in 12 of 22 patients, and inducible VT was modified in 4 patients. During follow-up of 334±280 days, 54% of patients with myocardial re-entry were free of VT despite frequent episodes before ablation.

CONCLUSIONS
The VTs in DCM are most commonly the result of myocardial re-entry associated with scar. Scars are often adjacent to a valve annulus, deep in the endocardium, and can be greater in extent on the epicardium than on the endocardium. The use of epicardial mapping and radiofrequency is likely to improve success. (J Am Coll Cardiol 2004;43:1834–42) © 2004 by the American College of Cardiology Foundation

Sustained monomorphic ventricular tachycardia (VT) associated with heart disease is often associated with areas of ventricular scar comprised of surviving myocytes and fibrotic tissue. After myocardial infarction, scar is typically evident involving the endocardium, and most re-entry circuits causing VT can be ablated from the endocardium (1). Sustained monomorphic VT also occurs, although less frequently in dilated cardiomyopathy (DCM) that are not associated with coronary artery disease. Re-entry within the myocardium is the most common cause, although bundle-branch re-entry and focal VT also occur (2–5). Catheter ablation for VT due to myocardial re-entry in DCM is generally thought to be more difficult than in patients with previous myocardial infarction. In some cardiomyopathies, such as Chagas disease, the presence of epicardial re-entry circuits that cannot be ablated with an endocardial approach contributes to this difficulty (6).

Recently, a method of plotting low-amplitude regions of scar on three-dimensional anatomic reconstructions of the ventricle has been successfully used to mark infarct regions and dense unexcitable scar that serves as a conduction block in infarct regions causing VT (7,8). The locations of low-amplitude bipolar electrograms correlate well with the location of infarct scars in animal models (9).

The purpose of this study was to evaluate the relationship of monomorphic VT to areas of low-amplitude scar by constructing three-dimensional electroanatomic ventricular maps of the endocardium and, in selected cases, of the epicardium in patients with DCM. The efficacy of catheter ablation was also evaluated.

METHODS
Studies were performed in 28 consecutive patients with DCM who were referred for catheter mapping and ablation of recurrent sustained monomorphic VT (24 male, 54±14 years, ejection fraction; 30±11%). The diagnosis of DCM was established based on a depressed left ventricular (LV) function (ejection fraction ≤0.50) known to have been present for a minimum of three months and on the absence of coronary artery disease according to coronary angiography. Patients with arrhythmogenic right ventricular (RV) dysplasia (10), cardiac sarcoidosis, or tachycardia-induced cardiomyopathy were excluded. Studies were performed in...
consenting patients referred for catheter ablation according to protocols approved by the Brigham and Women’s Hospital human subject protection committee.

**Mapping and ablation.** An electroanatomic mapping system (CARTO, Biosense Webster Inc., Diamond Bar, California) was used to identify the low-voltage area in the chamber of interest. Ventricular mapping was performed with 7-F steerable catheter with a 4-mm distal electrode (Cordis-Webster, Diamond Bar, California). Bipolar electrograms filtered at 30 to 400 Hz were recorded on the electroanatomic mapping system and on a separate digital system (Prucka Engineering Inc., Houston, Texas) with filtering at 30 to 500 Hz. For pace-mapping and entrainment mapping, unipolar pacing from the distal electrode of the mapping catheter was used with stimulus strength of 10 mA at a pulse width of 2 ms.

Programmed stimulation was performed from the RV apex and outflow tract with up to three extra stimuli at two different basic cycle lengths to induce VT and obtain the QRS morphology to compare with pace mapping. Ventricular tachycardia was terminated by pacing or cardioversion, and the ventricle was mapped during sinus rhythm to construct a voltage map displaying peak-to-peak electrogram amplitude with color range set for maximal voltage 1.5 mV, based on a previous study which found that 95% of electrograms recorded with this method in normal ventricles.

**Table 1.** Patient Characteristics

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**Abbreviations and Acronyms**
- DCM = dilated cardiomyopathy
- ICD = implantable cardioverter-defibrillator
- LV = left ventricle/ventricular
- RF = radiofrequency
- RV = right ventricle/ventricular
- S-QRS = stimulus to QRS interval
- VT = ventricular tachycardia

**Figure 1.** Flow diagram of the acute results of the endocardial and epicardial ablation. pts = patients; RF = radiofrequency.
Figure 2. (A) Voltage map of the endocardial surface of the left ventricle (LV) from Patient 7. An extensive low-voltage area is present in the LV outflow area. (B) Two morphologies of ventricular tachycardia (VT). Continued on next page.
had amplitudes exceeding this value (8). On the “voltage maps,” purple areas represent normal amplitude electrograms, and electrogram amplitude progressively diminishes as colors proceed to blue, green, yellow, and red.

If VT was stable, entrainment and activation sequence mapping was used to define a re-entry circuit isthmus for ablation. If VT was unstable as a result of hemodynamic intolerance or frequent change to another VT, pace mapping was used to define potential exit sites along the border of any low-voltage region, and limited entrainment mapping was performed (11,12). Radiofrequency (RF) ablation was initially attempted using a 7-F steerable catheter with a 4-mm distal electrode (Cordis-Webster). An internal irrigation catheter with a 4-mm distal electrode (Boston Scientific, Inc, Natick, Massachusetts) or 8-mm distal electrode catheter (EP Technologies, Sunnyvale, California) was used if the initial ablation lesions failed to render the region unexcitable as indicated by a pacing threshold >10 mA at pulse width 2 ms. Radiofrequency ablation lesions were applied to the target area until the pacing threshold exceeded 10 mA at 2 ms pulse width. After completion of the RF lesions, programmed stimulation was repeated to assess the acute result of ablation. The procedure ended when: 1) no monomorphic VT was inducible; 2) hemodynamically stable VT was inducible but no VT circuit sites could be found; or 3) only unstable VT that was faster than any of the previous VTs was inducible. After endocardial ablation, warfarin was administered if a line of RF lesions was placed on the LV endocardium; otherwise, patients received 325 mg of aspirin daily for ≥6 weeks.

**Epicardial mapping.** In eight patients who failed endocardial ablation, epicardial mapping and ablation were attempted at a subsequent session using a modification of the technique described by Sosa et al. (13). A 21-gauge micropuncture needle was inserted into the pericardial space for placement of a guide wire, followed by a small introducer that was then sized up to an 8-F introducer sheath. In two patients with difficult pericardial access owing to adhesions or previous cardiac surgery, the introducer and mapping catheters were inserted into the pericardial space under direct vision through a subxyphoid surgical incision.

**Types of VTs.** Sustained monomorphic VTs were divided into three groups on the basis of likely tachycardia mechanism (2): 1) focal VT—mapping shows spread of activation
away in all directions from the site of earliest activation relative to the QRS onset, and entrainment was not observed, suggesting automaticity as the likely mechanism; 2) bundle-branch re-entry—the His or right bundle potential is closely linked to the VT based on analysis of initiation and cycle length oscillations, and VT is abolished by ablation of the right or left bundle branch; and 3) myocardial re-entry—tachycardia can be entrained and does not meet the definitions for bundle-branch re-entry. For the purpose of this analysis, VT that is induced by programmed stimulation in a patient with other re-entrant VTs inducible was also considered to be due to re-entry.

Data analysis. The area of a region of low amplitude, presumed scar, was calculated by approximating the area as a rectangle or ellipse, as appropriate. Continuous data are expressed as mean ± SD. A Wilcoxon test was used to compare the number of implantable cardioverter-defibrillators (ICD) firing before and after ablation.

RESULTS

The patient characteristics and results summary are shown in Table 1. Twenty patients had an ICD before RF ablation, and six patients received an ICD after ablation. Therapy before ablation included amiodarone in 12, sotalol in 7, and other antiarrhythmic agents in 9 patients. A total of 82 VTs were induced in 28 patients (average of 2.9 ± 1.7 VTs [range 1 to 7] per patient). Seven VTs had a focal origin, two were bundle-branch re-entry, and the remaining 73 VTs (89%) in 22 patients were due to myocardial re-entry. In one patient, both myocardial re-entry and focal origin VTs were observed. In nine patients all induced VTs were unstable. In 10 patients all inducible VTs were hemodynamically stable, allowing activation and entrainment mapping, and in the remaining nine patients both stable and unstable VTs were present.

Myocardial re-entrant VT (Fig. 1). ENDOCARDIAL MAPPING AND ABLATION. Electroanatomic mapping of the endocardium was performed in 20 of the 22 patients with myocardial re-entry VT; an average of 125 ± 82 endocardial sites was recorded per patient. In two patients, endocardial mapping was not repeated after endocardial ablation had failed at another institution. All 20 patients who had endocardial mapping had at least one low-amplitude (<1.5 mV) region consistent with scar (total 32 areas of scar, mean of 2.0 ± 0.7 mV per patient). More than half of the 32 scars (63%) extended to a valve annulus, including the mitral annulus (6 scars), aortic annulus (5 scars), tricuspid annulus (5 scars), and pulmonary valve annulus (4 scars). The total area of endocardial scar per patient was 16.5 ± 8.1 cm² (range 3.7 to 32.2 cm²).

An endocardial VT isthmus was identified by entrainment mapping in five patients. In an additional seven patients with unstable VTs, pace mapping identified regions of slow conduction with a stimulus to QRS interval (S-QRS) >40 ms and a pace-map match consistent with the exit region from an isthmus (Fig. 2) (7,12). Thus no isthmus regions were identified on the endocardium in 8 of 20 patients with myocardial re-entry (40%).

We attempted endocardial RF ablation in 18 patients, at the endocardial isthmus sites identified in 12 patients, and at the endocardial sites judged closest to the re-entry circuit based on entrainment or pace mapping in an additional six patients. An average of 17.7 ± 9.4 RF applications were made per patient. Standard catheters followed by internally irrigated RF catheters were used in nine patients, standard catheters followed by an 8-mm electrode catheter were used in one patient; only standard 4-mm electrode catheters were used in the remaining eight patients. In two patients, ablation lesions were not applied because the sites closest to the re-entry circuit were in regions of normal voltage and in close proximity to the His bundle. Endocardial RF abolished all inducible myocardial re-entrant VTs (a total of 21 VTs) in six patients. Ablation in one region often abolished more than one morphology of VT, such that ablation at the initial six regions abolished a total of 14 VTs and ablation in an additional region was required to abolish the remaining 7 VTs. In four patients, ablation abolished one or more VTs, but other VTs remained inducible. In eight patients, VT was not altered after the endocardial ablation. Ablation for VT arising from the region of the His bundle produced complete atrioventricular block in one patient, as anticipated. There were no other complications.

EPICARDIAL MAPPING AND ABLATION. In seven patients with myocardial re-entry and failed endocardial ablation, epicardial mapping was performed. An average of 82 ± 20 epicardial sites were recorded per patient. All had epicardial regions of low amplitude consistent with scar (Fig. 3). A total of 12 low-amplitude epicardial regions were detected; 5 over the base of the LV, 3 over the RV outflow tract, 2 at the basal RV, and 2 at the lateral LV. Average epicardial scar area was 37.5 ± 10.4 cm² (range 22 to 47.3 cm²). In the five patients who had both epicardial and endocardial mapping, the area of scar was larger on the epicardial surface than on the endocardial surface (Fig. 4).

An epicardial VT isthmus was identified in six of the seven patients; by entrainment mapping in three patients, and in three patients by pace mapping that showed slow conduction with an S-QRS >40 ms and match to the VT morphology. Ablation with a 4-mm electrode catheter was successful in one patient. In five patients an internally irrigated catheter was used to apply RF with average power of 35 ± 11 W, impedance of 82 ± 10 ohms, temperature of 41 ± 8°C, after initial RF lesions with a standard 4-mm electrode catheter achieved heating to 60°C with low power (<15 W) and failed to render the site unexcitable to pacing at 10 mA 2 ms. All six of these VTs were successfully ablated after an average of 10.9 ± 8.0 epicardial RF applications per patient. In two patients, RF application was limited owing to the proximity to a coronary artery or to the phrenic nerve such that the area of a circuit isthmus was not
Figure 3. (A) An endocardial activation sequence map of the left ventricle (LV) from Patient 22. A large area of earliest endocardial activation at the mid-anterior LV (red) suggests the mechanism of the tachycardia being of focal origin; however, the ventricular tachycardia (VT) could be entrained. Radiofrequency (RF) applications at the earliest endocardial sites failed to abolish VT. (B) An epicardial activation sequence map of the same VT as in (A). The activation sequence is consistent with a large re-entry circuit. Sites with green tags are close to the phrenic nerve, as the pacing at these sites produced phrenic nerve stimulation. (C) The epicardial voltage map. Normal voltage (>1.5 mV) tissue is purple. A large area of low-voltage scar is shown. Radiofrequency ablation in the isthmus created by the dense, unexcitable scars (tagged as gray) was not performed owing to proximity to the phrenic nerve (green tags). A series of RF applications along the inner loop sites over the right ventricle did not terminate VT. Additional RF applications from the dense scar to the normal voltage area near the exit over the LV terminated VT. A series of RF applications near the exit was made from the gray area to the normal voltage area, which terminated and abolished inducible VT, but was limited by proximity to the left anterior descending coronary artery (LAD). Ventricular tachycardia recurred 27 days later, likely as a result of the healing of these lesions.
rendered unexcitable to pacing. One patient developed symptomatic pericarditis after the epicardial ablation that resolved with administration of a nonsteroidal anti-inflammatory agent. No other complication was observed.

FOLLOW-UP OF MYOCARDIAL RE-ENTRANT VT PATIENTS. In 16 patients, antiarrhythmic agents were discontinued, and in 6 of the patients who had inducible VT at the end of the procedure, antiarrhythmic agents (3 amiodarone, 3 sotalol) were continued. The 15 patients who had endocardial RF ablation alone for myocardial re-entrant VT were followed for a mean of 348 ± 345 days (range 49 to 1,083 days) during which time 7 patients had recurrent VT after 121 ± 92 days (range 35 to 274 days) (Table 1). Two patients who had endocardial ablation underwent only heart transplant. One patient had VT recurrence before the transplant, and the other patient had no recurrence. One patient died of heart failure after 76 days.

The seven patients who underwent epicardial RF ablation were followed for a mean of 115 ± 89 days during which time three patients had recurrent VT (range 27 to 246 days).

Other mechanisms of VT. Ablation of focal VTs or bundle-branch re-entry was successful in six of seven patients; none of these VTs recurred during a mean follow-up of 307 ± 97 days (range 110 to 361 days). One patient who had both myocardial re-entrant and focal VT had successful ablation of both VTs. Ablation was not attempted in one patient with a focal VT adjacent to the His bundle region. Endocardial mapping was more limited in these patients (97 ± 87 sites/patient). Only the LV was mapped in four, only the LV in one, and both ventricles were mapped in two patients. All had areas of scar, but the low-voltage region was relatively small (average of 7.5 ± 6.1 cm²) compared with that observed in patients with myocardial re-entry. All focal VTs originated in low-voltage regions. One patient underwent epicardial mapping, which showed no scar area. In five patients, antiarrhythmic agents were discontinued, and one patient received amiodarone, as the VT was not ablated.

Effect of ablation on VT frequency. For the total of 28 patients, the number of VT episodes/month decreased markedly after RF ablation, as shown in Figure 5.

DISCUSSION

This study further defines the substrate causing VT in patients with DCM and supports myocardial fibrosis as an important factor. As we observed in a previous series, myocardial re-entry was the most common cause (2). In the present study, the re-entry circuits identified were associated with regions of low-amplitude electrograms, consistent with scar, and in agreement with the findings of Hsia et al. (4,14). In studies of explanted hearts, de Bakker et al. (15) found unexcitable fibrosis creating regions of conduction block and surviving myocardium creating potential re-entry circuit paths after infarction and in DCM. Slow conduction through muscle bundles separated by interstitial fibrosis can cause a zigzag path producing slow conduction that promotes re-entry. The cause of fibrosis in cardiomyopathy is not well defined. Scattered regions of replacement fibrosis are commonly seen at autopsy, but confluent regions of scar are not common (16,17). In some cases, coronary embolism from left atrial or ventricular thrombus might cause a confluent infarct. Interestingly we also observed low-voltage areas in patients with focal VT and bundle-branch re-entry, although the scar areas appeared to be smaller. These findings should be interpreted with caution because mapping was less extensive in these patients and the number of patients studied was small. It is tempting to postulate, however, that as for patients with myocardial infarction, larger areas of scar are associated with a greater chance of developing monomorphic VT.

We observed several similarities of the arrhythmia substrate in patients with myocardial re-entry VT and DCM to that in patients with previous infarction. Low-voltage areas were observed in all patients. The regions of scar were frequently adjacent to a valve annulus, as is often the case in VT after inferior wall infarction (18,19). The annulus often seems to form a border for an isthmus in the re-entry path. It is interesting to speculate that the formation of a long...
channel, or isthmus along an annulus contributes to the formation of re-entry circuits that can support sustained monomorphic VT. Pace mapping demonstrated slow conduction in these regions with long S-QRS delays during pace mapping and entrainment.

The success of endocardial ablation was lower than that which we have observed for post-infarct VT, although similar to that observed in a previous series from our center (2). Re-entry circuits deep to the endocardium and in the epicardium appear to be a likely explanation. Epicardial mapping led to successful ablation in more than half of the patients in whom it was attempted. The successful ablation sites were again associated with low-amplitude regions. Pacing in these regions also showed evidence of slow conduction. Interestingly, the region of low amplitude was strikingly larger in the epicardium than at the endocardium in five of seven patients.

The importance of epicardial re-entry circuits in cardiomyopathy was demonstrated by Sosa et al. (6) for patients with Chagas disease, in whom approximately 70% of VTs were epicardial in origin. Recently, Hsia et al. (14) used limited epicardial mapping via the coronary venous system to demonstrate epicardial involvement in re-entry circuits in 3 of 19 patients with cardiomyopathy unrelated to Chagas disease. In the present study of nonischemic cardiomyopathy not due to Chagas disease, involvement of the epicardium was also common. In addition, ablation of VT could be achieved from the epicardium guided by entrainment mapping and pace mapping with unipolar pacing. Interestingly, when VT was identified on the epicardium, the area of low voltage was larger on the epicardial surface compared with the endocardial surface.

We did not observe any serious complications after epicardial mapping and ablation. Safe epicardial ablation has been reported by others (6,20). Precautions to avoid coronary artery and phrenic nerve injury are prudent. We performed coronary angiography while the ablation catheter was on a target site to assess the distance to the coronary artery and used unipolar pacing to detect proximity to the left phrenic nerve.

The relationship of re-entry circuits to regions of scar supports the feasibility of a substrate mapping approach, targeting the abnormal area based on mapping during sinus rhythm and pace mapping, to guide ablation of unstable VT, similar to that described for patients with previous infarctions (8,12). Unstable VTs that did not allow mapping during the VT were present in seven of the eight patients who underwent epicardial mapping and were ablated in six patients. Thus our study also suggests that a substrate mapping approach can be used for epicardial re-entry circuits.

CONCLUSIONS

Sustained monomorphic VTs in DCM are usually due to re-entry associated with low-voltage areas consistent with scar. Scars are often adjacent to a valve annulus, extend deep to the endocardium, and can be transmural or greater in extent on the epicardium than on the endocardium. Combined endocardial and epicardial mapping approaches are likely to improve the success of ablation. Because it is desirable to achieve pericardial access before systemic anticoagulation for endocardial LV mapping, performing epicardial mapping before LV endocardial mapping in DCM is a reasonable consideration. This approach must be balanced, however, by anticipated risks and the experience of the team with the epicardial approach, because many VTs can be ablated from the endocardium.

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