Ventricular Tachycardia Substrate

For the ablationist

Stanley Tung, MD FRCPC
Arrhythmia Service/St Paul Hospital
University of British Columbia
Two Attitudes of Ventricular Tachycardia Ablation
Ventricular Tachycardia Substrate

- Mechanism of Ventricular Tachycardia (VT)
- Principals of scar related substrate (ischemic and non-ischemic)
- Disease specific VT substrate:
  Infiltrative Cardiomyopathy: ARVD/C, Sarcoidosis
  Post Valvular Surgery Cardiomyopathy
  Repaired Adult Congenital Heart Disease
Three Mechanisms of VT

Re-entry

Figure 2. Entrainment mapping — different responses at different sites. Schematic of a scar related reentry circuit. Grey areas represent scar. (a + c) represents the conduction time through the isthmus, (b) through the outer loop, (d) through a central bystander, and (e) to a remote bystander. Pacing is slightly faster as the VT cycle length resets (enters) the VT reentry circuit. Pacing at an exit site (1) results in a short stimulus to QRS (S-QRS) interval identical to the local electrogram to QRS (E-QRS) interval. The paced QRS complex is identical to the VT QRS complex (concealed fusion) and the PPI approximates the conduction time through the circuit which is the conduction time through the outer loop (b) and the isthmus (a + c). At a central isthmus site (2), the S-QRS is prolonged and equals the E-QRS with identical QRS morphology, resembling the conduction time (a). At an outlet loop site (3), the PPI is identical with the VTCL but the paced QRS complex differs from the VT QRS due to fusion. Pacing at a remote bystander (4) results in a different paced QRS complex. The PPI is prolonged by the propagation time (e) to and from the reentry circuit. Pacing at an adjacent bystander (5) results in concealed fusion with a S-QRS interval that is longer than the E-QRS interval, and a prolonged PPI interval due to the propagation time (d) to and from the circuit.
Three Mechanisms of VT

**Figure 6** Transmembrane potentials in a trabecula from a failing human heart during superfusion with modified Tyrode solution. The last four stimulated action potentials are marked by the filled circles. Upon cessation of stimulation a train of five triggered action potentials ensues, followed by a large delayed afterdepolarisation.

**Figure 7** Intracellular recording of transmembrane potentials obtained from a human trabecula. Automaticity develops after switch (arrow) from normal to modified Tyrode solution. A subthreshold depolarisation precedes the automatic rhythm.

Triggered Activity  Automaticity
Magnetic Resonance–Based Anatomical Analysis of Scar-Related Ventricular Tachycardia Implications for Catheter Ablation


Abstract—In catheter ablation of scar-related monomorphic ventricular tachycardia (VT), substrate voltage mapping is used to electrically define the scar during sinus rhythm. However, the electrically defined scar may not accurately reflect the anatomical scar. Magnetic resonance–based visualization of the scar may elucidate the 3D anatomical correlation between the fine structural details of the scar and scar-related VT circuits. We registered VT activation sequence with the 3D scar anatomy derived from high-resolution contrast-enhanced MRI in a swine model of chronic myocardial infarction using epicardial sock electrodes (n=6, epicardial group), which have direct contact with the myocardium where the electrical signal is recorded. In a separate group of animals (n=5, endocardial group), we also assessed the incidence of endocardial reentry in this model using endocardial basket catheters. Ten to 12 weeks after myocardial infarction, sustained monomorphic VT was reproducibly induced in all animals (n=11). In the epicardial group, 21 VT morphologies were induced, of which 4 (19.0%) showed epicardial reentry. The reentry isthmus was characterized by a relatively small volume of viable myocardium bound by the scar tissue at the infarct border zone or over the infarct. In the endocardial group (n=5), 6 VT morphologies were induced, of which 4 (66.7%) showed endocardial reentry. In conclusion, MRI revealed a scar with spatially complex structures, particularly at the isthmus, with substrate for multiple VT morphologies after a single ischemic episode. Magnetic resonance–based visualization of scar morphology would potentially contribute to preprocedural planning for catheter ablation of scar-related, unmappable VT. (Circ Res. 2007;101:939-947.)
Magnetic Resonance–Based Anatomical Analysis of Scar-Related Ventricular Tachycardia Implications for Catheter Ablation

Scar VT Substrate

Figure 1. A. High-resolution contrast-enhanced MRI (spatial resolution, 0.39×0.39×0.36 mm). The region of high intensity (indicated by white arrows) is the infarct. The myocardium in the infarct region is substantially thinner than the remote myocardium, and a thin rim of the viable myocardium on both the septal and the endocardial edges of the infarct was observed. B through D, Basal (B), anterior (C), and posterior (D) views of the 3D infarct geometry reconstructed from MRI. The infarct regions are represented by dark gray, and the normal ventricular myocardium (RV and LV) regions are shown in pink. The scar exhibited variable wall thickness, with occasional branching of the infarct structure at the periphery. There are islands of infarct within the viable myocardium and also islands of viable myocardium within the infarct. The scar involved the RV in the apical region.
Scar VT Substrate

Figure 3. Epicardial reentry registered with MR-derived scar. A possible electrical propagation at the infarct border zone is indicated by dashed red arrows. A, VT no. 5. The isthmus (circumscribed by a broken red line) was located at the posteroapical segment of the infarct border zone. The scar geometry at the isthmus was characterized by scar tissue intercalated with multiple small areas of viable myocardium. Electrical activation initially spread longitudinally toward the base, split into 2 opposite lateral directions, and then returned longitudinally to the posteroapical region. B, VT no. 12. The isthmus (circumscribed by a broken red line) was located at the anterior irregular surface of the scar. This irregular surface consists of thin interwinding layers of viable myocardium (see the MR image on the right).
Scar VT Substrate

**Figure 4.** The border between the normal and infarcted/aneurysmal wall contains a stylized ventricular tachycardia (VT) circuit—predominantly endocardial, and partially intramural. The surgical procedures, subendocardial resection, and ventriculotomy are believed to either remove or transect critical endocardial portions of the VT circuit, respectively. After mapping the border zone using the electromagnetic mapping system, the putative exit site of the VT was identified with pace-mapping, and catheter-based linear lesions were placed. Although a single catheter-based linear lesion set along the scar border could potentially interrupt the circuit(s), unlike during surgery, one cannot ensure the lesion’s depth and continuity. Therefore, a second linear lesion set was placed proceeding into the scarred myocardium—with the hope that this empiric lesion set would transect critical portions of the VT circuit(s). LV = left ventricular. Figure reproduced and modified with permission, courtesy of J. Miller, MD, and Williams & Wilkins Inc. (19).
Scar VT Substrate

Normal myocardium
>1.5-1.8mV

Low Voltage region
0.5-1.5mV

Scar
<0.5mV

Francis E. Marchlinski, MD; David J. Callans, MD; *Circulation*, 2000;101:1288-1296.)
Figure 7. Outcome after linear lesions for unmappable VT. All patients had frequent VT during month before ablation. A total of 12 patients remained arrhythmia-free during follow-up. Isolated VT occurred at 3, 9, and 13 months in 3 patients, with frequent VT in only one patient.
Scar VT Substrate

Relationship Between Successful Ablation Sites and the Scar Border Zone Defined by Substrate Mapping for Ventricular Tachycardia Post-Myocardial Infarction

ATUL VERMA, M.D., NASSIR F. MARROUCHE, M.D., ROBERT A. SCHWEIKERT, M.D., WALID SALIBA, M.D., OUSSAMA WAZNI, M.D., JENNIFER CUMMINGS, M.D., AHMAD ABDUL-KARIM, M.D., MANDEEP BHARGAVA, M.D., J. DAVID BURKHARDT, M.D., FETHI KILICASLAN, M.D., DAVID O. MARTIN, M.D., and ANDREA NATALE, M.D.

From the Department of Cardiology, Section of Cardiac Pacing and Electrophysiology, Cleveland Clinic Foundation, Cleveland, Ohio, USA

Ventricular Tachycardia and the Scar Border Zone. Introduction: It is unknown if identification of scar border zones by electroanatomical mapping correlates with successful ablation sites determined from mapping during ventricular tachycardia (VT) post-myocardial infarction (MI). We sought to assess the relationship between successful ablation sites of hemodynamically stable post-MI VTs determined by mapping during VT with the scar border zone defined in sinus rhythm.

Methods and Results: Forty-six patients presenting with hemodynamically stable, mappable monomorphic VT post-MI and who had at least one such VT successfully ablated were prospectively included in the study. In each patient, VT was ablated by targeting regions during VT that exhibited early activation, ± isolated mid-diastolic potentials, and concealed entrainment suggesting a critical isthmus site. Prior to ablation, a detailed sinus-rhythm CARTO voltage map of the left ventricle was obtained. A voltage <0.5 mV defined dense scar. Successful VT ablation sites were registered on the sinus voltage map to assess their relationship to the scar border zone. Of the 86 VTs, 68% were successfully ablated at sites in the endocardial border zone. The remaining VTs had ablation sites within the scar in (18%), in normal myocardium (4%), and on the epicardial surface (10%). There were no significant differences in VT recurrence amongst the different groups.

Conclusion: Successful ablation sites of hemodynamically stable, monomorphic VTs post-MI are often located in the scar border zone as defined by substrate voltage mapping. However, in a sizable minority, ablation sites are located within endocardial scar, epicardially, and even in normal myocardium. (J Cardiovasc Electrophysiol, Vol. 16, pp. 665-671, May 2005)
Figure 2. Three-dimensional electroanatomical voltage map in sinus rhythm of a patient with successful VT ablation site in the scar border zone. The successful ablation site is indicated by the red dots in the figure (arrow). Note that the ablation site is located in the border zone adjacent to dense scar indicated by red.
Scar VT Substrate

Relationship Between Successful Ablation Sites and the Scar Border Zone Defined by Substrate Mapping for Ventricular Tachycardia

Verma et al
CAFCAA 2008
Post-Myocardial Infarction
VERMA et al
Scarf VT Substrate

Figure 1. Graphs depicting percentage of patients (white bars) and ventricular tachycardias (solid bars) for which a successful VT ablation site was found in a particular location (x-axis). The actual number of patients (out of 46) and the actual number of VTs (out of 89) is indicated above each bar for reference.
Scar VT Substrate

Antero-apical circuits

Figure-8
n=7

Figure-8
n=6

Single-loop
n=1

Septal circuits

Figure-8
n=1

Infero-lateral circuits

Figure-8
n=3

Figure-8
n=3

Figure-8
n=1

Single-loop
n=2

Single-loop
n=1

Perimtiral circuits

Figure-8
n=2

Figure-8
n=1

Single-loop
n=2

Single-loop
n=2

Christian de Chillou, MD, PhD; Dominique Lacroix, MD. Circulation. 2002;105:726-731.)
Scar VT Substrate

• Characteristics of Isthmus:
  – Average length 31mm ±7mm
  – Average width 16mm ±8mm (R 6-24mm)
  – Isolated potentials
  – IP often long delay during SR, and pre-systolic during VT
  – S-QRS=E-QRS < 70% VTCL
  – SR pacing often >40ms, but more specific if >80ms. Morphology adds no extra value
Scar VT Substrate

Figure 1. (A) Shown are recordings from surface leads I, aVF, and V1, and intracardiac recordings from the mapping catheter electrogram (EGM) and the right ventricular apex (RVA). The local EGM that is recorded by the mapping catheter is normal. The amplitude is 7.8 mV, and the width is 65 ms. (B) The tracings are analogous to the tracings in panel A. The local EGM recorded by the mapping catheter displays an isolated potential (oblique arrow) that is separated from the ventricular EGM by an isoelectric line of 210 ms. The EGM amplitude is 0.12 mV, and the EGM width is 357 ms. (C) The tracings are again analogous to the tracings in panel A. The recorded EGM is fractionated; the amplitude is 0.37 mV, and the width is 192 ms. (D) The tracings are again analogous to the tracings in panel A. The local EGM is abnormal. The amplitude is 0.7 mV, and the width of the EGM is 112 ms.
Scar VT Substrate
Scar VT Substrate

Table 2. Electrogram Characteristics According to Electrogram Width

<table>
<thead>
<tr>
<th></th>
<th>&lt;133 ms</th>
<th>133–160 ms</th>
<th>160–200 ms</th>
<th>&gt;200 ms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of EGMs</td>
<td>1,532</td>
<td>273</td>
<td>171</td>
<td>187</td>
</tr>
<tr>
<td>Mean width (ms)</td>
<td>90 ± 25</td>
<td>145 ± 54</td>
<td>178 ± 12</td>
<td>270 ± 72</td>
</tr>
<tr>
<td>Amplitude (mV)</td>
<td>1.6 ± 2.0</td>
<td>0.41 ± 0.38</td>
<td>0.32 ± 0.3</td>
<td>0.28 ± 0.26</td>
</tr>
<tr>
<td>Normal EGMs</td>
<td>231/1,532 (15%)</td>
<td>1/273 (0.4%)</td>
<td>0/171 (0%)</td>
<td>0/187 (0%)</td>
</tr>
<tr>
<td>Abnormal EGM</td>
<td>879/1,532 (57%)</td>
<td>18/273 (7%)</td>
<td>5/171 (3%)</td>
<td>2/187 (1%)</td>
</tr>
<tr>
<td>Fractionated EGMs</td>
<td>381/1,532 (25%)</td>
<td>175/273 (64%)</td>
<td>89/171 (52%)</td>
<td>24/187 (13%)</td>
</tr>
<tr>
<td>IP EGMs</td>
<td>41/1,532 (3%)</td>
<td>79/273 (29%)</td>
<td>77/171 (45%)</td>
<td>161/187 (86%)</td>
</tr>
<tr>
<td>Isthmus sites</td>
<td>0/32</td>
<td>1/32</td>
<td>1/32</td>
<td>28/32</td>
</tr>
</tbody>
</table>

EGM = electrogram (at 2 of 32 isthmus sites the EGM width could not be determined); IP = isolated potential.
Scar VT Substrate

Christian de Chillou, MD, PhD; Dominique Lacroix, MD. Circulation. 2002;105:726-731.)
Scar VT Substrate
Scar VT Substrate
Scar VT Substrate
Scar VT Substrate

**Figure 2.** Typical contrast-enhanced images obtained by MRI. Scar involvement was most common in the basal myocardial slices. The myocardium has been divided into 12 sectors, starting from the posterior right ventricular insertion point (red line). A, Example of myocardium free of scar. B, Small region of hyperenhancement as an example of predominant scar distribution involving 1% to 25% of wall thickness. Sector numbering is clockwise, starting from the right ventricular insertion point, with sectors 3 to 5 having 4%, 20%, and 17% scar. C, An example of predominance of scar involving 26% to 75% of wall thickness. Sectors 1 to 8 have 50%, 56%, 53%, 61%, 62%, 71%, 40%, and 11% scar; sector 12 has 40% scar. D, The midwall area of hyperenhancement was visualized using a range of inversion times and in multiple planes. Example of scar involving 76% to 100% of wall thickness (sectors 11 and 12 with 85% and 78% scar).

**Figure 3.** Example of the relation between scar location on delayed enhancement images and morphology of ventricular tachycardia on 12-lead ECG. A, A 4-chamber image of the heart, with the right atrium and ventricle at the top of the image and left atrium and ventricle at the bottom. B, The left bundle branch-like configuration in lead V1 of the ventricular tachycardia ECG suggests an exit site in the right ventricle or interventricular septum and is compatible with the scar location in A.
### Scar VT Substrate

#### TABLE 2. MRI Parameters and Comparison After Stratification by Inducibility at Electrophysiological Study*

<table>
<thead>
<tr>
<th>Parameter</th>
<th>All Patients (n=26)</th>
<th>Inducible (n=5)</th>
<th>Noninducible (n=21)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left ventricular ejection fraction, %</td>
<td>27 (17–43)</td>
<td>16 (13–29)</td>
<td>29 (19–44)</td>
<td>0.18</td>
</tr>
<tr>
<td>Left ventricular end-diastolic volume, mL</td>
<td>201 (129–273)</td>
<td>159 (154–273)</td>
<td>205 (129–259)</td>
<td>0.72</td>
</tr>
<tr>
<td>Left ventricular mass, g</td>
<td>153 (113–192)</td>
<td>184 (160–197)</td>
<td>144 (113–199)</td>
<td>0.38</td>
</tr>
<tr>
<td>Patients with scar, n (%)</td>
<td>17 (65)</td>
<td>5 (100)</td>
<td>12 (57)</td>
<td>0.13</td>
</tr>
<tr>
<td>Scar volume, %</td>
<td>4.6 (0.8–7.2)</td>
<td>6.0 (5.7–7.2)</td>
<td>1.5 (0.7–6.3)</td>
<td>0.09</td>
</tr>
<tr>
<td>Predominant scar distribution involving 26%–75% of wall thickness, n (%)</td>
<td>6 (23)</td>
<td>5 (100)</td>
<td>1 (5)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

*Data are expressed as median (interquartile range) or absolute numbers (column percentage).
Figure 1. Estimation of the abnormal LV endocardium in a patient with nonischemic cardiomyopathy. Endocardial 3D voltage maps were constructed using electroanatomical mapping and displayed in RAO and posterior projections. Purple-colored areas represent normal endocardium (≥1.8 mV) with dense scar depicted as red (<0.5 mV). Border zone (0.5 to 1.8 mV) is defined as areas with the color gradient between red and purple. Endocardial surface area was estimated by using a computer algorithm. A. Extent of abnormal endocardium was estimated by measuring contiguous areas of abnormal electrogram recordings represented by the non-purple-colored area. The area was first outlined and then divided into multiple, nonoverlapping triangular segments. Overall extent of abnormal endocardium (<1.8 mV) can be determined as the sum of all abnormal segments. In this example, the abnormal LV endocardium has an area of 81 cm². B. Entire LV endocardial surface area can also be calculated. Valvular annuli were outlined and excluded from analysis. Remaining LV endocardial surface was divided into nonoverlapping triangular segments. Total LV surface area was estimated as the sum of all segments. In the example shown, the total LV endocardial surface area was calculated to be 218 cm².

19 patients, 20% of the endocardium bipolar EGM<1.8mV

Characterization of Endocardial Electrophysiological Substrate in Patients With Nonischemic Cardiomyopathy and Monomorphic Ventricular Tachycardia Henry H. Hsia et al.
Scar VT Substrate

Figure 3. Localization and characterization of the VT circuit. Entrainment responses coupled with the endocardial voltage map are shown. Map color gradient is described as in Figure 1. Pacing during sustained monomorphic VT resulted in a return cycle length equal to the VT cycle length. A, Entrainment with minimal fusion is observed near the exit with a short stimulus-QRS interval. Voltage characteristics at the exit site is consistent with border zone. B, Entrance site is identified with an entrainment response and concealed fusion. Long stimulus-QRS matches the electrogram-QRS interval with voltage characteristics consistent with dense scar. Entrance and exit sites for this VT are located near the LV base in the area of abnormal endocardium as depicted by the voltage map.

Table 3. Comparison of VT Site-of-Origin to the Location of Abnormal Endocardial Electrogram Distribution

<table>
<thead>
<tr>
<th>Region</th>
<th>Abnormal Endocardial Electrogram Location (19 patients)</th>
<th>VT Site-of-Origin (57 VTs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basal</td>
<td>19/19 (100%)</td>
<td>50/57 (88%)</td>
</tr>
<tr>
<td>Nonbasal</td>
<td>1/19 (5%)</td>
<td>7/57 (12%)</td>
</tr>
</tbody>
</table>

Characterization of Endocardial Electrophysiological Substrate in Patients With Nonischemic Cardiomyopathy and Monomorphic Ventricular Tachycardia Henry H. Hsia et al.
Scar VT Substrate

≈ 5% Ischemic cardiomyopathy

≈ 20-40% Non-ischemic cardiomyopathy

Baseline IVCD and HV prolongation (>75ms)

Stable HV during VT and HV interval ≥ SR
Figure 1. Termination of bundle branch reentrant ventricular tachycardia during application of radiofrequency energy. Tracings in each panel, from top to bottom, are surface electrocardiographic leads 1, 2, and V1, right atrium (RA), proximal and distal recordings from the ablating catheter (RFp and RFd), right ventricular electrogram (RV), and time lines (T). Pertinent intervals are labeled and all intervals are in milliseconds. In panel A, activation of the right bundle branch (RB) is recorded in the distal bipolar of the ablating catheter during sinus rhythm. Panel B displays BBB tachycardia with a left bundle branch block morphology and a cycle length of 310 msec. Activation of the right bundle branch during tachycardia is recorded in the ablating catheter. Ablation of the right bundle branch and termination of BBB tachycardia occur within 6 seconds of energy application (panel C). Radiofrequency energy was delivered from the distal electrode of the ablating catheter to a skin patch. Note the expected right bundle branch block morphology of the first sinus beat after termination of the tachycardia.
Scar VT Substrate

Epicardial substrate

More common in non-ischemic cardiomyopathy and in inferior MI

PsΔ ≥ 34 ms
Sens 83
Spec 95
ID time ≥ 85 ms
Sens 87
Spec 90
RS ≥ 121 ms
Sens 76
Spec 85

Antonio Berruezo, MD; Lluis Mont, MD; Circulation. 2004;109:1842-1847.)
Scar VT Substrate

- Inferior, basal and apical, epicardial: Initial Q’s II, III, aVF
- Anterior, apical, epicardial: Q wave in lead I
- Anterior, basal, epicardial: Q wave in lead I, absent of initial Q’s II, III, aVF

Victor Bazan, MD, Edward P. Gerstenfeld, MD, Heart Rhythm 2007;4:1403–1410
Scar VT Substrate

William G. Stevenson, MD; Kyoko Soejima, MD Circulation. 2007;115:2750-2760
Scar VT Substrate

William G. Stevenson, MD; Kyoko Soejima, MD
Circulation. 2007;115:2750-2760
ARVC/D VT Substrate

Around TA and outflow tract usually re-entry

RV apex could be re-entry or automaticity

Figure 1. Schema of the right ventricular endocardium. This illustrates a representation of the double or fractionated potential sites and electrical silent (scar) areas in the 17 patients. The right ventricle is shown with the free wall, dissected down, thus exposing the septum, inferior wall, and tricuspid annulus. The sites of the DPs and fractionated electrograms are represented by circles (●), and electrical silent areas by open circles (○). Star symbols (★) indicate the sites where radiofrequency ablation terminated the ventricular tachycardia (VT). Abnormal areas and VT termination sites are circumscribed around the tricuspid annulus and in the right ventricular outflow tract. AO = aorta; DP = double potential; ESA = electrical silent area; FRE = fractionated electrogram; PA = pulmonary artery; RA = right atrium; RV = right ventricle; SVC = superior vena cava.
ARVC/D VT Substrate

Atul Verma, MD; Felhi Kilicastan, MD. Circulation, 2005;111:3209-3216
ARVC/D VT Substrate
ARVC/D VT Substrate

Atul Verma, MD; Felhi Kilcastan, MD. *Circulation*, 2005;111:3209-3216
ARVC/D VT Substrate

TABLE 3. Patient Electrophysiological Characteristics

<table>
<thead>
<tr>
<th>Patient</th>
<th>VT Cycle</th>
<th>Scar Location(s)</th>
<th>Linear Lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>290-440</td>
<td>VEO, NS</td>
<td>EL</td>
</tr>
<tr>
<td>2</td>
<td>350-440</td>
<td>VEO, NS</td>
<td>LC (enlarge)</td>
</tr>
<tr>
<td>3</td>
<td>320-400</td>
<td>Post-pect</td>
<td>EL</td>
</tr>
<tr>
<td>4</td>
<td>310-380</td>
<td>Rvsl post-lat</td>
<td>LC (enlarge)</td>
</tr>
<tr>
<td>5</td>
<td>320-410</td>
<td>Ant and ant-optal</td>
<td>EL, LC (sever)</td>
</tr>
<tr>
<td>6</td>
<td>250-300</td>
<td>Inf-lat, inf-optal</td>
<td>EL</td>
</tr>
<tr>
<td>7</td>
<td>450</td>
<td>NS</td>
<td>EL</td>
</tr>
<tr>
<td>8</td>
<td>270-300</td>
<td>Rvsl mid-lat</td>
<td>LC (enlarge)</td>
</tr>
<tr>
<td>9</td>
<td>270-300</td>
<td>NS</td>
<td>EL, LC (enlarge)</td>
</tr>
<tr>
<td>10</td>
<td>370-400</td>
<td>NS</td>
<td>EL</td>
</tr>
<tr>
<td>11</td>
<td>350-500</td>
<td>NS, MCM</td>
<td>EL, LC (enlarge)</td>
</tr>
<tr>
<td>12</td>
<td>390-580</td>
<td>Basal lat, proximal outflow tract</td>
<td>EL, LC (sever)</td>
</tr>
<tr>
<td>13</td>
<td>240-280</td>
<td>Ant-lst, proximal outflow tract</td>
<td>EL, LC (enlarge)</td>
</tr>
<tr>
<td>14</td>
<td>330-510</td>
<td>Basal post-lat, basol inf</td>
<td>LC (enlarge)</td>
</tr>
<tr>
<td>15</td>
<td>315-370</td>
<td>NS</td>
<td>LC (sever)</td>
</tr>
<tr>
<td>16</td>
<td>290-420</td>
<td>Basal lat, proximal outflow tract</td>
<td>EL, LC (enlarge)</td>
</tr>
<tr>
<td>17</td>
<td>250</td>
<td>NS</td>
<td>Ant-optal</td>
</tr>
<tr>
<td>18</td>
<td>350-405</td>
<td>NS, MCM</td>
<td>LC (enlarge)</td>
</tr>
<tr>
<td>19</td>
<td>355-450</td>
<td>NS, MCM</td>
<td>LC (enlarge)</td>
</tr>
<tr>
<td>20</td>
<td>230</td>
<td>NS</td>
<td>Inf-optal</td>
</tr>
<tr>
<td>21</td>
<td>230</td>
<td>NS</td>
<td>Basal post-lat</td>
</tr>
<tr>
<td>22</td>
<td>340-380</td>
<td>NS, MCM</td>
<td>Mid-optal, post sept</td>
</tr>
</tbody>
</table>

Atul Verma, MD; Fetih Kilicaslan, MD. *Circulation*. 2005;111:3209-3216
Post Corrective Valve Surgery VT Substrate

20 patients with 17 patients inducible, 42 VT induced. All reentry.

Figure 1. Endocardial exit sites of induced monomorphic VT associated with surgical valvular disease. Of the 14 patients with inducible macroreentrant VT, the area of interest was found to be in proximity to the annulus in 9 patients (64.3%). Sites labeled by patient number as given in the Table. Those underlined indicate the patient had prior aortic valve surgery; those italicized indicate the patient had prior mitral valve surgery. Ao indicates aorta; LA, left atrium; RV, right ventricle; and LV, left ventricle.

Robert E. Eckart, DO; Tomasz W. Hruczkowski, MD; Circulation. 2007;116:2005-2011.)
Post Corrective Valve Surgery VT Substrate

Figure 2. Bipolar endocardial voltage map as seen from the anterior and inferior views of the left ventricle (LV) in a 41-year-old patient 16 years after aortic valve replacement, with an ejection fraction of 30%. Evidence of anatomic low-voltage scar with adjacent points within a territory with bipolar voltage <1.5 mV throughout mid to distal anterolateral wall (LV anterior), as well as diffusely low voltage from base to apex along the inferior wall. Brown tags indicate sites of radiofrequency lesions placed at reentry circuit sites. Three different VTs were inducible originating from the anteroapical and inferoapical regions of low voltage.

41/42 VT was successfully ablated.

Late recurrent of VT post TOF, VSD, or RVOT patch repair ≈12%
SCD rate was 9% over 35 years of follow up
All VTs were reentrant

Katja Zeppenfeld, MD; Martin J. Schalij, MD; *Circulation*. 2007;116:2241-2252.)
Post Corrective CHD Surgery VT Substrate

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Conclusions

- Scar resulting in reentry is the most common cause of VT
- Isthmus mostly present along scar border zone
- Isthmus length and width are variable
- Characteristic of the isthmus can often identify during substrate mapping
- Isolate potential is crucial in targeting isthmus site for successful ablation
Conclusions cont’d

- Ischemic scar closely locates to previous infarct territory
- Non-ischemic scar often locates around valvular apparatus without following coronary perfusion landmarks
- BB reentrant VT is far more common with non-ischemic VTs
• Epicardial VT substrate is more common with inferior MI and non-ischemic cardiomyopathy
• ARVC/D often with scar around TA, RVOT, and RVA with VT mostly due to reentry
• Ablation often successful with ablation line connecting the scar border to the vulvular annulus
Conclusions cont’d

• Reentrant VT Post valvular surgery is not infrequently presents late after surgery
• Critical isthmus leading to VT exit often around the valvular apparatus with successful ablation along the region
Conclusions cont’d

• Post repair surgery for congenital heart disease is not infrequently presents late with reentrant VT
• Most experience are with repair of TOF, VSD and RVOT patch
• Critical isthmus often borders by the valve annulus with either the ventriculotomy scar, or with the patch.
• Ablation is highly successful with ablation lines connecting either the scar of the patch to the annulus
Linear Ablation Lesions for Control of Unmappable Ventricular Tachycardia in Patients With Ischemic and Nonischemic Cardiomyopathy

Francis E. Marchlinski, MD; David J. Callans, MD; Charles D. Gottlieb, MD; Erica Zado, PA-C

Background—Conventional activation mapping is difficult without inducible, stable ventricular tachycardia (VT).

Methods and Results—We evaluated 16 patients with drug refractory, unimorphic, unmappable VT. Nine patients had ischemic and 7 had nonischemic cardiomyopathy. All patients had implantable defibrillators and had experienced 6 to 55 VT episodes during the month before treatment. Patients underwent bipolar catheter mapping during baseline rhythm. The amount of endocardium with an abnormal electrogram amplitude was estimated using fluoroscopy in 3 patients and a magnetic mapping system (CARTO) in 13 patients. For the magnetic mapping, normal endocardium was defined by an amplitude >1.5 mV; this measurement was based on sinus rhythm maps in 6 patients who did not have structural heart disease. Radiofrequency point lesions extended linearly from the “dense scar,” which had a voltage amplitude <0.5 mV, to anatomic boundaries or normal endocardium. To limit radiofrequency applications, 12-lead ECG during VT and pacemapping guided placement of linear lesions. No new antiarrhythmic drug therapy was added. The amount of endocardium demonstrating an abnormal electrogram amplitude ranged from 25 to 127 cm². A total of 8 to 87 radiofrequency lesions (mean, 55) produced a median of 4 linear lesions that had an average length of 3.9 cm (range, 1.4 to 9.4 cm). Twelve patients (75%) have been free of VT during 3 to 36 months of follow-up (median, 8 months); 4 patients had VT episodes at 1, 3, 9, and 13 months, respectively. Only one of these patient had frequent VT.

Conclusions—Radiofrequency linear endocardial lesions extending from the dense scar to the normal myocardium or anatomic boundary seem effective in controlling unmappable VT. (Circulation. 2000;101:1288-1296.)
Figure 2. Epicardial sock data during reentry VT. Left, An isochronal map. The black dots indicate the location of electrodes. Right (A through I), Signals from bipolar electrograms at respective locations. This figure shows progression of electrical activation from point A (blue, 1 ms) to point I (yellow, 147 ms) in alphabetical order.
Three Mechanisms of Tachycardia

Re-entry
Ischemic VT Substrate

Figure 4. Epicardial reentry registered with MR-derived scar (continued). A possible electrical propagation at the infarct border zone is indicated by dashed red arrows. A, VT no. 18. The isthmus (circumscribed by a broken red line) was located at the anterior right ventricular insertion, where a small volume of viable myocardium bound by the infarct tissue is protruding into the RV. B, VT no. 19. The isthmus (circumscribed by a broken red line) was located at a relatively large volume of the viable myocardium surrounded by the infarct tissue at the anterolateral infarct border. Note A and B are 2 distinct epicardial reentry circuits in the same heart with different isthmus locations.
Ischemic VT Substrate

Figure 6. Epicardial centrifugal pattern of VT registered with MR-derived scar (continued). A possible electrical propagation is indicated by dashed red arrows. A, VT no. 7. The breakthrough points were located in the viable myocardium in the RV, and the electrical wave propagated to the latest activation site (circumscribed by a dashed red line) in the viable myocardium in the anterior LV. B, VT no. 17. The breakthrough was located in the viable myocardium in the lateral LV, and the electrical wave propagated to the latest activation site (circumscribed by a broken red line) in the infarct border zone.
Scar VT Substrate
Figure 8. Schema for suggested mechanism of linear lesion technique contrasted with surgical subendocardial resection. Bipolar electrogram voltage map identifies dense scar (consistent with aneurysm formation), border zone, and normal endocardium. Subendocardial linear lesions extend from dense scar to normal endocardium. Linear lesions guided by pacemapping may interrupt potential for reentry, much like subendocardial resection.
Disease Specific VT Substrate

Normal Structure Heart:
- Outflow Tract VTs: Right, Left
- Great Vessels VTs: Pulmonary, Aortic Cusp
- LV Fascicular VT
- Mitral Annulus VT
- Polymorphic VT/VF: Long QT Syndrome
  Brugada Syndrome