Localization of ischemic VT

Seongwook Han, MD, PhD
Professor of Medicine, Keimyung University School of Medicine
Arrhythmia Service, Cardiology, Dongsan Medical Center
Mechanism of Scar Related VT

- **Reentry**: VT associated with healed or healing MI in > 95%
- 2 conditions
  - unidirectional block & circuit cycle length > refractory period
Reentry

- Originates from *surviving bundles of myocardium within the scar*, separated by connective tissue, fibrosis and disordered intercellular coupling.

- Substrate develops *gradually during the first 2 weeks following MI* and once established, *remains indefinitely*.

- Spontaneous VT occurs in the presence of *appropriate triggers* such as surges in *autonomic tone, electrolyte imbalance, acute ischemia, or acute heart failure decompensation*. 
Substrate of Scar related VT

A

Base
Normal
\[ \geq 1.50 \text{ mV} \]
Dense scar

\[ \leq 0.5 \text{ mV} \]

Septum
Apex

B

Infarct
RV
LV
Septum
Lateral
Figure of 8 reentry

Anatomical labyrinth circuit, created by strands of viable myocardium within the scar, with potential for multiple reentry circuits.
Enrolled 13 patients with healed MI & refractory VT

10 VTs in 8 patients were mappable with plunge needle electrodes (39 needles; 156 intramural recording sites)
5 of 10 VTs (50%) were *reentrant sustained VT*: initiated in subendocardium or epicardium by *intramural reentry*

- Functional and anatomic *block* were *prominent* in the *subendocardium & midmyocardium*

- *Multiple simultaneous circuits* can be present

---

**Keimyung University Dongsan Hospital**

Pogwizd SM et al. Circ 1992;86:1872
Reentrant and Focal Mechanisms Underlying Ventricular Tachycardia in the Human Heart

5 of 10 VTs (50%) were focal VT
Focal mechanism of ventricular tachycardia in coronary artery disease

- 46 patients with prior MI (male 89%, age 64.8 ± 10.2 years)
- 101 VTs were induced (91% macroreentrant VT, 9% focal VT)
ECG features suggesting VT related to old MI

- **Presence of Q waves** (qR, QR or Qr) in related leads
  QS implies an electrical impulse moving away from the recording site

- **Notched or wide QRS complexes**
  Electrical activation is initiated in the viable myocytes with slow conduction

- **Low QRS voltage**
  Larger scar with less viable myocardium

- **Multiple morphologies** of monomorphic VT

- Paroxysmal sustained episodes

Benito B & Josephson ME. Rev Esp Cardiol 2012;65:939
Factors affecting the QRS patterns of ischemic VT

- size & site of infarction
- degree of myocardial fibrosis
- shape of the heart (aneurysm) & attitudinal anatomy
- site and mechanism of VT
- Influence of nonuniform anisotropy in affecting propagation from the site of origin
- effects of acute ischemia and/or electrolyte abnormalities
- Integrity of His-Purkinje system
- structural abnormalities unrelated to the tachycardia mechanism
Localization of documented VT ECG allows for procedural planning, particularly regarding vascular access, and for guiding the initial mapping procedure.
Locate the Reentrant Circuit Exit

A

Lateral: aVR > aVL
Mid: aVR = aVL
Septal: aVR < aVL

B

Basal: No transition (+V1-V6)
Mid: Transition by V3-4
Apical: Transition by V1-2; CS V6
Things to consider in evaluating ischemic VT

- Always arise in LV & ventricular septum
- RBBB pattern (positive deflection as the latter half of lead V₁): from LV
- LBBB pattern (negative deflection as the latter half of lead V₁): from LV (septum or < 1cm paraseptal)
- QRS onset corresponds with the exit from a circuit
Things to consider in evaluating ischemic VT

- VT exit sites tend to occur within or at the periphery of infarct regions
- QS complex in V₄~V₆: from apex regardless of septal or lateral
- Accuracy of ECG localization: inferior MI >> anterior MI
- Impossible to discriminate the VT from apico-septum vs. apico-lateral wall in anterior MI-dependent VT
Relationship between the 12-lead ECG during VT & endocardial site of origin in patients with CAD

- Endocardial mapping: 182 VTs from 108 patients
- Cather:surgical:both = 154:85:57 VTs
- ECG, characterized by 4 features
  1. Location of infarction
  2. BBB pattern
  3. Axis: four quadrants
  4. R wave progression pattern (RWP)
- Validation cohort: 110 VTs in 63 patients
  - 93% of the 65 VTs (59% of the total number) to which the algorithm could be applied
Relationship between the **12-lead ECG** during VT & endocardial site of origin in patients with **CAD**

**Precordial R Wave Patterns**

<table>
<thead>
<tr>
<th>R wave Pattern</th>
<th>V1</th>
<th>V2</th>
<th>V3</th>
<th>V4</th>
<th>V5</th>
<th>V6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increasing</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
</tr>
<tr>
<td>None/Late</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
</tr>
<tr>
<td>Loss/Increase (rS)</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
</tr>
<tr>
<td>Loss/Increase (QS)</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
</tr>
<tr>
<td>Dominant</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
</tr>
<tr>
<td>Abrupt Loss</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
</tr>
<tr>
<td>Early Reverse</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
</tr>
<tr>
<td>Late Reverse</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
<td>↘</td>
</tr>
</tbody>
</table>
**Inferior infarction-dependent VT**

**LB VT**

- Left superior
- Right superior
- Left inferior
- Right inferior

Increasing

R wave Pattern

| $V_1$ | $V_2$ | $V_3$ | $V_4$ | $V_5$ | $V_6$ |

Inferobasal septum
Inferior infarction-dependent VT

**RB VT**

- Left superior
- Right superior
- Left inferior
- Right inferior

<table>
<thead>
<tr>
<th>Reverse (either)</th>
</tr>
</thead>
<tbody>
<tr>
<td>R wave Pattern</td>
</tr>
<tr>
<td>Early Reverse</td>
</tr>
<tr>
<td>Late Reverse</td>
</tr>
</tbody>
</table>

Inferobasal free wall

<table>
<thead>
<tr>
<th>Reverse (late)</th>
</tr>
</thead>
<tbody>
<tr>
<td>R wave Pattern</td>
</tr>
<tr>
<td>Late Reverse</td>
</tr>
</tbody>
</table>

Inferobasal lateral wall
Anterior infarction-dependent VT

**LB VT**

- Left superior
- Right superior
- Left inferior
- Right inferior

**R wave Pattern**

- None / Late
- \( V_1 \), \( V_2 \), \( V_3 \), \( V_4 \), \( V_5 \), \( V_6 \)

**Inferoapical septum**
- Any pattern
  - Anteroapical septum

Keimyung University Dongsan Hospital

Miller JM et al. Circulation 1988;77:759
Anterior infarction-dependent VT

RB VT

- Left superior
- Right superior
- Left inferior
- Right inferior

Dominant or abrupt loss

R wave Pattern

<table>
<thead>
<tr>
<th></th>
<th>$V_1$</th>
<th>$V_2$</th>
<th>$V_3$</th>
<th>$V_4$</th>
<th>$V_5$</th>
<th>$V_6$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dominant</td>
<td>♠</td>
<td>♠</td>
<td>♠</td>
<td>♠</td>
<td>♠</td>
<td>♠</td>
</tr>
<tr>
<td>Abrupt Loss</td>
<td>~</td>
<td>~</td>
<td>~</td>
<td>~</td>
<td>~</td>
<td>~</td>
</tr>
</tbody>
</table>

Anteroapical septum
the 12-lead ECG during VT contains adequate information to specify a region of the LV endocardium that is likely to contain the VT site of origin in approximately half of all VTs in patients with a single prior infarction
A Novel Algorithm for Determining Endocardial VT Exit Site from 12-Lead Surface ECG Characteristics in Human, Infarct-Related Ventricular Tachycardia

noncontact activation mapping in 121 VTs from 51 post-MI patients
Mechanism for spontaneous changes in QRS morphology ~ During Reentrant VT in a Canine Infarct Model

- Small *changes in conduction velocity* in the segment of the circuit, which *modified the length of the functional lines of block* resulted in a *shift of the exit*
  → QRS morphology changes
Summary

In order to regionalize the origin of the ischemic VT

- Predictive power of surface ECG: ~ 75%
- QRS patterns can be categorized depending on the site of infarction
- Changes of QRS pattern can occur spontaneously
- Site & size of infarction, attitudinal relationship, general concept of surface ECG should be taken into consideration
- VT related with ischemic CM may be focal
Thank You for Your Attention!