Catheter ablation of post-infarct VT

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Background

EP tools for reentrant circuits

Ablation technique and results
Management of patients receiving implantable cardiac defibrillator shocks

Recommendations for acute and long-term patient management

Frieder Braunschweig (Chair)1*, Giuseppe Boriani (Co-chair)2, Alexander Bauer3, Robert Hatala4, Christoph Herrmann-Lingen5, Josef Kautzner6, Susanne S. Pedersen7, Steen Pehrson8, Renato Ricci9, and Martin J. Schalij10

VT + structural heart disease ➞ ICD

Class I, B (SFC / 2006)

Catheter ablation of ventricular tachycardia is recommended

1. For symptomatic sustained monomorphic VT necessitating frequent ICD therapies despite antiarrhythmic drug therapy or when antiarrhythmic drugs are not tolerated or not desired (especially when VT recurrences fulfil definition of ES).
2. For control of recurrent symptomatic or incessant monomorphic VT not suppressible by antiarrhythmic drug therapy, regardless whether VT is stable or unstable, or multiple VTs are present.
3. For bundle branch re-entrant or interfascicular VTs.
4. For recurrent sustained polymorphic VT and VF refractory to antiarrhythmic therapy when there is a suspected trigger that can be targeted by ablation.

There was consensus among the task force members that catheter ablation for VT should be considered early in the treatment of patients with recurrent VT.

Bundle branch reentrant VTs

Bundle branch reentrant VT

Although more frequent in patients with an underlying heart disease, BBR-VT may occur in patients without structural heart disease but an impaired HPS conduction.


Common type

Reverse type

## Ventricular Tachycardia

<table>
<thead>
<tr>
<th></th>
<th>Idiopathic VT</th>
<th>Structural Heart Disease VT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart disease</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Substrate</td>
<td>No Scar</td>
<td>Scar</td>
</tr>
<tr>
<td>Mechanism</td>
<td><strong>Focal</strong> &gt; 90%</td>
<td><strong>Reentrant</strong> &gt; 90%</td>
</tr>
<tr>
<td></td>
<td>Reentrant &lt; 10%</td>
<td>Focal &lt; 10%</td>
</tr>
<tr>
<td>Anatomical origin</td>
<td>- RVOT</td>
<td>- Endocardial &amp;/or Epicardial</td>
</tr>
<tr>
<td></td>
<td>- LVOT</td>
<td>- LV &amp;/or RV</td>
</tr>
<tr>
<td></td>
<td>- Left fascicles</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Papillary muscles</td>
<td></td>
</tr>
<tr>
<td>Guidelines</td>
<td>Usually no ICD</td>
<td>ICD implantation = Class IB</td>
</tr>
<tr>
<td></td>
<td>(only if VT is poorly tolerated and VT ablation is impossible or fails)</td>
<td>(AHA/ACC/HRS 2008 guidelines &amp; SFC 2006)</td>
</tr>
</tbody>
</table>
Slow conduction perpendicular to the fiber direction in infarcted myocardial tissue is caused by a "zigzag" course of activation at high speed. Activation proceeds along pathways lengthened by branching and merging bundles of surviving myocytes unsheathed by collagenous septa.

de Bakker JMT. et al. Circulation 1993;88:915-26
Background

*EP tools for reentrant circuits*

Ablation technique and results
Principles to rapidly localize the VT exit site
Typical RVOT Tachycardia
VT exit = RV infero-basal area ➔ ARVD

LBBB + left axis pattern ➔ not a normal heart!
VT exit = LV infero-apical area ➔ post-infarct VT
VT exit = LV infero-basal area ➔ post-infarct VT
Pace mapping to localize the exit of a reentrant VT

The 12-lead ECG morphologies during pacing and VT perfectly match.
Transient entrainment of a tachycardia

VT circuit
Propagation map

Pacing @ a faster rate @ a remote site

Resumption of the intrinsic rate of the tachycardia...

...after abrupt cessation of pacing
The recognition of entrainment: entrainment criteria

- Constant fusion
- Progressive fusion
- Shorter conduction time associated with termination
- Fusion at the local electrograms
- Constant first post-pacing interval (pacing trains with different number of beats)

Waldo AL et al. Circulation 1977;56:737-45
Mc Lean WAH et al. PACE 1981;4:358-66
Waldo AL. Heart Rhythm 2004;1:94-106
Almendral JM et al. PACE 2013;36:508-532
Measurement of the first post pacing interval

Post pacing interval (PPI) = time interval (measured @ pacing site ++) from the last stimulus to the return cycle local activation

- PPI = St-Entrance delay + VT cycle length + Entrance-St delay
- PPI – VT cycle length = 0ms when pacing is in the circuit
Adapted from Stevenson WG et al. J Am Coll Cardiol 1997;29:1180-9

12-lead ECG = QRS fusion

St-QRS = 0 ms
EGM-QRS = 0 ms

12-lead ECG = concealed fusion

St-QRS = 70 ms
EGM-QRS = 70 ms

12-lead ECG = concealed fusion

St-QRS = 270 ms
EGM-QRS = 170 ms
VT entrainment → VT circuit mapping

**VT entrainment**

Concealed entrainment
S-QRS = 260ms

Entrainment with fusion
S-QRS = 20ms
EP characteristics of BBR-VT

- H and RB potentials preceding V with appropriate sequence according BBR-VT type (A,B or C)
- HV interval identical or 10-30 ms longer than during sinus rhythm (type B excepted)
- H-H variations preceding V-V variations
- Induction depending upon a critical delay in the HPS
- Termination by block in the HPS
- Short-long-short sequences frequently required to induce
- Non inducibility after RBB ablation
RV apical stimulation to measure PPI in suspected BBR-VT

Merino JL et al. Circulation 2001;103:1102-8
# Entrainment as a guide for VT ablation

<table>
<thead>
<tr>
<th></th>
<th>Remote site</th>
<th>Outer loop</th>
<th>Adjacent Bystander</th>
<th>Isthmus</th>
</tr>
</thead>
<tbody>
<tr>
<td>PPI - VT CL*</td>
<td>&gt; 30 ms</td>
<td>0-30 ms</td>
<td>&gt; 30 ms</td>
<td>0-30 ms</td>
</tr>
<tr>
<td>12-lead ECG</td>
<td>QRS fusion</td>
<td>QRS fusion</td>
<td>Concealed fusion</td>
<td>Concealed fusion</td>
</tr>
<tr>
<td>St-QRS – EGM-QRS</td>
<td>Variable</td>
<td>0-20 ms</td>
<td>&gt; 20 ms</td>
<td>0-20 ms</td>
</tr>
</tbody>
</table>

* Accurate measurement may be difficult, especially in case of a fractionated EGM
Some flexibility is needed since some conduction slowing may impact on the figure obtained
Value of entrainment as a guide for VT ablation

- PPI – VT cycle length ≤30ms → PPV = 20% for VT termination *
- Concealed entrainment → PPV = 54% for VT termination **
- Concealed entrainment + S-QRS criteria → PPV = 80% **

Limitation = width of the VT isthmus ! Actually the PPV of RF applications can only be evaluated in very narrow isthmuses...

* Stevenson WG et al. Circulation 1993;88:1647-70

- General limitations: failure to pace, failure to recognize fusion, challenging measurements in case of EGM with multiple components @ pacing site or when some part of the circuit shows decremental conduction properties...
3D (endocardial) mapping during VT
→ color-coded isochronal maps

- Isochronal steps = 5ms
- Isochronal steps = 40ms

**Focal VT pattern**

**Reentrant VT pattern**
What is the ablation target?

Focal VT pattern

Reentrant VT pattern

Isochronal steps = 5ms

Isochronal steps = 40ms
Protected isthmus ➔ ablation target ➔ linear lesions

Functional or anatomical fixed barriers in stable VT

Isthmus width = 16±8 mm (6 to 36)
Isthmus length = 31±7 mm (18 to 41)

Background

EP tools for reentrant circuits

Ablation technique and results
Imaging the VT substrate
To identify the causes of cardiomyopathies


Myocardial infarction

DCM
HCM
Sub-endocardial or transmural MI
- Myocarditis
- Sarcoidosis
- Amyloidosis...
Arrhythmogenic substrate – post infarct VT

Voltage mapping
EGM < 1.5mV → scar
EGM < 0.5mV → ‘dense’ scar

Increased accuracy of the LV geometry reconstruction

Improved infarct border delineation especially in areas where catheter access and stability may be challenging or catheter contact may be poor

MATLAB (Mathworks, Natick, MA)

MRI & Arrhythmogenic substrate in VT

By courtesy of Dr Hubert Cochet
MRI & Arrhythmogenic substrate in VT

By courtesy of Dr Hubert Cochet
Special = Focal VT !!
Case Report #1

- 70 year-old man / Chronic AFib
- Inferior wall MI = 04/2000
- No acute revascularization
- CX occluded – EF=50%
- Syncopal VT ➔ ICD (03/2001)
- VT storm

- Post RF : no further VT
Case Report #2

- 52 year-old man
- Anterior wall MI = 12/2001
- No acute revascularization
- LAD occluded – EF=30%
- Severe HF 11/2002 : NHYA IV
- Repetitive incessant NSVT/SVT

- Post RF clinical improvement NYHA II
VT inducible and well-tolerated \( \rightarrow \) Mappable VT

3D mapping during VT to define the VT circuit
Step # 1 = 3D reconstruction of the LV $\rightarrow$ voltage mapping
Step #2 = VT induction and mapping $\rightarrow$ VT circuit reconstruction
Merging DCE-MRI into a 3D mapping system
EGM voltage within protected VT isthmuses

LV - inferior view

Table 3  Local electrogram amplitude for sites within the reentrant circuit

<table>
<thead>
<tr>
<th></th>
<th>Entrance</th>
<th>Central isthmus</th>
<th>Exit</th>
<th>Outer loop</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dense scar (&lt;0.5 mV)</td>
<td>17</td>
<td>30</td>
<td>18</td>
<td>6</td>
</tr>
<tr>
<td>Border zone (0.5–1.5 mV)</td>
<td>2</td>
<td>7</td>
<td>26</td>
<td>18</td>
</tr>
<tr>
<td>Normal (&gt;1.5 mV)</td>
<td>—</td>
<td>—</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>Total (136 sites)</td>
<td>19</td>
<td>37</td>
<td>48</td>
<td>32</td>
</tr>
</tbody>
</table>

Hsia HH et al. Heart Rhythm 2006;3:503-12
Post-infarct VT: peri-mitral reentrant circuit
Post-infarct VT: antero-apical reentrant circuit
Endocardial (figure-of-eight) reentrant VT circuit
Endocardial reentry > 90% of post-MI mappable VTs
Different VT morphologies & shared VT isthmus

Limitations of mapping and ablation during VT

- VT non inducible = 14%
- 12-lead ECG during VT non available = 30%
- VT non tolerated = 70%

Possibility to map at least one VT morphology in only 25% of patients

Pr Paolo Della Bella, Milan, Italy – ESC 2012
Non mappable VT
Post-infarct non mappable VT ablation
Possible empiric approaches

Where is the VT isthmus?

EGM-based ablation ➔ electrical targets

Linear ablation ➔ anatomical targets
Mapping potential VT channels during sinus rhythm
Voltage of the far-field signal may be higher as compared to the local component.
Variability of cut-off values…

Ablation target = site of entrance of a late potential channel

Elimination of local abnormal ventricular activities (LAVA)

Impact of local ablation on interconnected channels within ventricular scar

Tung R et al. Circ Arrhythm Electrophysiol 2013;6:1131-8
Defining a channel: not such an easy task!
Pace mapping during sinus rhythm to unmask VT isthmuses
Is pace mapping able to show up a VT isthmus?

3D activation map during SR

3D activation map during VT

VT isthmus = ablation target
Pace mapping to target the origin of RVOT tachycardia
Pace mapping to localize the exit of a reentrant VT

The 12-lead ECG morphologies during pacing and VT perfectly match
Pace-mapping with computerized real-time comparison matching with a reference

Real-time analysis of the QRS morphology of each QRS complex on the 12-lead ECG ➔ comparison with a QRS morphology reference

BARD™ template matching

\[
\text{CORR} = \frac{\sum_{\text{Lead } 1}^{12} \left[ \sum_{i=1}^{n} (X_i - \bar{X}) \times (Y_i - \bar{Y}) \right]}{\sqrt{\sum_{\text{Lead } 1}^{12} \left( \sum_{i=1}^{n} (X_i - \bar{X})^2 \times \sum_{i=1}^{n} (Y_i - \bar{Y})^2 \right)}}
\]

“PM-map” of a focal VT originating from the RVOT

Pace mapping and activation mapping are highly correlated

Color-coding = value of PM correlation (%)

Red is > 90% - Purple is poor…
Relationship between VT circuit mapping & pace mapping results

This is a consistent finding in all patients!
Pacing at VT isthmus exit

Pace mapping

VT entrainment

Average correlation = 100%

Concealed entrainment
S-QRS = 70ms
Pacing at VT isthmus entrance

Pace mapping

VT entrainment

Average correlation = 10%

Concealed entrainment
S-QRS = 260ms
3D LV geometry during SR ➔ voltage map

Scar = 65cm²
(29% of LV surface)
Perimeter = 38cm
3D LV geometry during SR ➔ local activation time map
3D LV geometry during SR ➔ LAT propagation map
3D LV geometry during SR ➔ pace mapping map
Inferior wall infarct $\rightarrow$ pace mapping map

Clinical VT induced

A few millimeters separate perfect pace-mapping sites from very poor pace-mapping sites!!

Explanation: how can PM allow the identification of VT isthmuses?
Pacing at VT isthmus exit

Pace mapping

VT entrainment

Average correlation = 100%

Concealed entrainment
S-QRS = 70ms
Pacing at VT isthmus exit

VT mapping

VT entrainment

12-lead ECGs look alike
Pacing at VT isthmus exit

VT mapping

Pacing during SR

12-lead ECGs look alike

Activation map 100ms after pacing stimulus
Pacing at VT isthmus entrance

Pace mapping

VT entrainment

Average correlation = 10%

Concealed entrainment
S-QRS = 260ms
Pacing at VT isthmus entrance

VT mapping

VT entrainment

12-lead ECGs look alike
Pacing at VT isthmus entrance

Activation map 100ms after pacing stimulus

12-lead ECGs look very different!

VT mapping

Pacing during SR
One step forward...
Pacing within the VT isthmus

Pacing during SR immediately after the mid-isthmus limit

Pacing during SR immediately before the mid-isthmus limit
Pacing within the VT isthmus / functional barriers

12-lead ECGs should look very similar!

Pacing during SR immediately after the mid-isthmus limit

Pacing during SR immediately before the mid-isthmus limit
Inferior wall infarct ➔ pace mapping map

Clinical VT induced (CL=384ms)
BP = 55/25mmHg

ATP to restore sinus rhythm 90 seconds after VT induction
Inferior wall infarct ➔ pace mapping map

Clinical VT

Average 98%

Average -29%

98% QRS Correlation -36%

Isochronal Steps

Main Map Viewer

Step 10

Mitral Annulus

Aorta

36mm X 28mm

-32%

67%

97%

10mm X 10mm
Inferior wall infarct ➔ pace mapping map

Clinical VT induced (CL=384ms)
BP = 55/25mmHg

98% QRS Correlation -36%

Isochronal Steps
Main Map Viewer

Step 10
Final PES to test VT inducibility
How to prove conduction block across the RF line?
How to prove conduction block across the RF line?

Clinical VT

Average  
-29%  96%

98%  QRS Correlation -36%

Isochronal Steps
Main Map Viewer

Step

10mm X 10mm
• Pace mapping is able to unmask post-infarct VT isthmuses…
• …because VT isthmuses borders are not functional barriers
• Pace mapping can be used as a tool to validate the presence of a conduction block through post-infarct VT isthmuses following RF applications
• Perspective: unmasking VT isthmuses in post-infarct patients with known stable VT (ICD data) regardless the availability of a 12-lead ECG during VT
Epicardial approach for VT ablation


<10% of post-infarct VT
VT ablation in patients with a structural heart disease

Literature review (case reports excluded)
Results for both ablation of stable and unstable VTs are shown

<table>
<thead>
<tr>
<th>Heart disease</th>
<th>Studies</th>
<th>Years</th>
<th>Patients</th>
<th>Acute success</th>
<th>Mean FU (mo)</th>
<th>Recurrences*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Post-MI</td>
<td>29</td>
<td>1993 - 2007</td>
<td>1093</td>
<td>76%</td>
<td>19.2</td>
<td>30%</td>
</tr>
<tr>
<td>ARVD/C</td>
<td>11</td>
<td>1998 - 2007</td>
<td>211</td>
<td>72%</td>
<td>31.5</td>
<td>33%</td>
</tr>
<tr>
<td>Idiopathic DCM</td>
<td>6</td>
<td>1995 - 2006</td>
<td>77</td>
<td>73%</td>
<td>13</td>
<td>41%</td>
</tr>
</tbody>
</table>

* Global recurrence rate, mixing patients with a successful ablation and those with a failed one
Long-term results and predictors of recurrence after ablation of post-infarct VT

95 consecutive patients with an ICD and an electrical (VT) storm
72 with an ischemic heart disease
1 to 3 procedures
Median FU = 22 months

Long-term results and predictors of recurrence after ablation of post-infarct VT

A Stepwise Approach to the Management of Postinfarct Ventricular Tachycardia Using Catheter Ablation as the First-Line Treatment: A Single-Center Experience

Maheshwar Pauriah, Gabriel Cismaru, Isabelle Magnin-Poull, Marius Andronache, Jean-Marc Sellal, Jérôme Schwartz, Béatrice Brembilla-Perrot, Nicolas Sadoul, Etienne Aliot and Christian de Chillou

Circ Arrhythm Electrophysiol 2013;6;351-356; originally published online March 19, 2013;
DOI: 10.1161/CIRCEP.113.000261

Median FU = 4.5 yrs
## Complications of VT Ablation in Patients with Structural Heart Disease

<table>
<thead>
<tr>
<th>Literature data</th>
<th>Acute complications = 7.4% !!</th>
</tr>
</thead>
<tbody>
<tr>
<td>Post-infarct VT</td>
<td>Death = 0.9%</td>
</tr>
<tr>
<td></td>
<td>Transient Ischemic Attack or Stroke = 0.9%</td>
</tr>
<tr>
<td></td>
<td>Pericardial Effusion or Tamponade = 1.8%</td>
</tr>
</tbody>
</table>
Conclusions

• In patients with no heart disease most VTs are ‘focal’, as opposed to VTs in patients with a remote myocardial infarction which are mostly related to a reentrant circuit.

• Whatever the mechanism, most VTs can be successfully treated by catheter ablation.

• VT ablation should be offered early, especially in post-infarct patients.
Prophylactic catheter ablation for the prevention of defibrillator therapy
(Substrate Mapping and Ablation in Sinus Rhythm to Halt Ventricular Tachycardia = SMASH-VT trial)

128 adult patients: previous MI (>1 month) + spontaneous VT or VF or syncope (and inducible VT)
Randomly allocated ➔ ICD alone / catheter ablation + ICD

Catheter ablation of stable VT before ICD implantation in patients with coronary heart disease (VTACH): a multicentre randomised controlled trial

16 European centres
110 adult patients: VT + previous MI + LVEF≤50%
Randomly allocated → ICD alone / catheter ablation + ICD