Basic Concept of Understanding Ventricular Tachycardias

Josef Kautzner
Department of Cardiology,
Institute for Clinical & Experimental Medicine,
Prague, Czech Republic

joka@medicon.cz
www.ikem.cz

www.escardio.org/EHRA
My Disclosures

• **Advisory Board member**
  – Biosense-Webster, Boston Scientific, GE Healthcare, Hansen Medical, Medtronic

• **Steering Committee member**
  – Biotronik, Medtronic, Boston Scientific, Sanofi Aventis

• **Clinical studies - PI**
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• **Speakers bureau**
  – Biosense-Webster, Biotronik, Boston Scientific, GE Healthcare, Medtronic, Hansen Medical, Siemens, St Jude Medical
EHRA/HRS Expert Consensus on Catheter Ablation of Ventricular Arrhythmias

Developed in a partnership with the European Heart Rhythm Association (EHRA), a Registered Branch of the European Society of Cardiology (ESC), and the Heart Rhythm Society (HRS); in collaboration with the American College of Cardiology (ACC) and the American Heart Association (AHA)

Etienne M. Aliot, MD, FESC, FHRS\(^1\), William G. Stevenson, MD, FHRS\(^2\), Jesus Ma Almendral-Garrote, MD, PhD\(^3\), Frank Bogun, MD\(^4\), C. Hugh Calkins, MD, FHRS\(^5\), Etienne Delacretaz, MD, FESC\(^6\), Paolo Della Bella, MD, PhD, FESC\(^7\), Gerhard Hindricks, MD, PhD\(^8\), Pierre Jaïs, MD, PhD\(^9\), Mark E. Josephson, MD\(^10\), Josef Kautzner, MD, PhD\(^11\), G. Neal Kay, MD\(^12\), Karl-Heinz Kuck, MD, PhD, FESC, FHRS\(^13\), Bruce B. Lerman, MD, FHRS\(^14\), Francis Marchlinski, MD, FHRS\(^15\), Vivek Reddy, MD\(^16\), Martin-Jan Schalij, MD, PhD\(^17\), Richard Schilling, MD\(^18\), Kyoko Soejima, MD\(^19\), and David Wilber, MD\(^20\)
Ventricular Tachycardia (VT) - a tachycardia (rate > 100/minute) with 3 or more consecutive ventricular beats independent of atrial or AV nodal conduction.

Monomorphic VT has a similar QRS configuration from beat to beat (some variability in QRS morphology at initiation is not uncommon).

Polymorphic VT has a continuously changing QRS configuration from beat to beat indicating a changing ventricular activation sequence.

Pleomorphic VT - more than one morphologically distinct QRS complex occurring during the same episode of VT, but the QRS is not continuously changing.

Ventricular Tachycardias

Polymorphic

- In structural heart disease
- Idiopathic

Monomorphomic

- In structural heart disease
- Idiopathic

MALIGNANT or POTENTIALLY MALIGNANT

BENIGN

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Ventricular Tachycardias

- Mechanism – focal vs reentry
- Pathophysiology of focal–triggered activity, automaticity, micro-reentry
Definitions

- **Sustained VT** - continuous VT for ≥ 30 seconds or that requires an intervention for termination (such as cardioversion).

- **Hemodynamically Unstable VT** causes hemodynamic compromise requiring prompt termination.

- **Nonsustained VT** terminates spontaneously within 30 seconds.

- **Repetitive monomorphic VT** - continuously repeating episodes of self terminating nonsustained VT.

Repetitive Monomorphic VT
Definitions

• **Clinical VT** - VT that has occurred spontaneously based on analysis of QRS morphology and rate on 12-lead ECG

• **Non-clinical VT** is a term used to indicate a VT induced by programmed ventricular stimulation or overdrive pacing that has not been documented previously (induced VTs with a QRS morphology that has not been previously observed should be called “undocumented VT morphology.”)

• **VT storm** is considered of 3 or more separate episodes of sustained VT within 24 hours, each requiring termination by an intervention\(^1,4\).

Electrical (VT) storm due to focally triggered polymorphic VT/VF
• Incessant VT is continuous sustained VT that recurs promptly despite repeated intervention for termination over several hours.

• Idioventricular rhythm is 3 or more consecutive beats at a rate < 100/minute that originate from the ventricles independent of atrial or AV nodal conduction.

Incessant VT
Principles of Mapping and Ablation of Focal VTs
Activation Sequence Mapping & Pacemapping
Idiopathic Outflow Tract VTs

2 subtypes, different mapping strategies

- 1) focus in myocardium of outflow tracts (incl. close to cusps, accessible below ventriculoarterial junction) (presystolic activity 20–40, pacemap usually perfect match)
- 2) focus in myocardial extensions into cusps or great vessels (earlier prepotentials, same potentials recorded in sinus rhythm as late potentials, pacemapping could be misleading)
Idiopathic Outflow Tract VTs
Proximity of Semilunar Cusps to Ventricular Myocardium
70 pts w. idiopathic VT w. LBBB pattern
55 had earliest activation in RVOT
15 had earliest activation in ACS

Succesful ablation was at earliest activation site in all pts
Pacemap was best at this site only in 8 pts in ACS group (7 had best pacemap in RVOT)

Myocardial Extensions in PA or AO

95 human heart specimens Ventricular myo extensions in 21 (95%)
17% PA, 7% AO

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6pts w. LBBB VT morphology
RFA from PA

Note reversal of 2 components of PA EGM during ectopy (analogy to PVPs in AF patients)


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VTs from PA

12/276 pts (4%) – focus in PA
ECG similar to RVOT

RFA in RVOT – change in QRS morphology

Ablated from PA, mostly PL


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The Role of ICE to Guide the Procedure
Principles of Mapping and Ablation of Focally Triggered Polymorphic VT/VF
Focal Trigger for polymorphic VT/VF
Mapping and Ablation of Idiopathic Ventricular Fibrillation

Michel Haïssaguerre, MD; Morio Shoda, MD; Pierre Jaïs, MD; Akihiko Nogami, MD; Dipen C. Shah, MD; Josef Kautzner, MD; Thomas Arentz, MD; Dietrich Kalushe, MD; Dominique Lamaison, MD; Mike Griffith, MD; Fernando Cruz, MD; Angelo de Paola, MD; Fiorenzo Gaïta, MD; Mélèze Hocini, MD; Stéphane Garrigue, MD; Laurent Macle, MD; Rukshen Weerasooriya, MD; Jacques Clémenty, MD

Background—Ventricular fibrillation is the main mechanism of sudden cardiac death. The feasibility of eliminating recurrent episodes by catheter ablation has not been reported.

Methods and Results—Twenty-seven patients without known heart disease (13 men, 14 women, 41±14 years of age) were studied after being resuscitated from recurrent (10±12) episodes of primary idiopathic ventricular fibrillation; 23 had received a defibrillator. The first initiating beat of ventricular fibrillation had an identical electrocardiographic morphology and coupling interval (297±41 ms) to preceding isolated premature beats typically noted in the aftermath of resuscitation. These triggers were localized by mapping the earliest electrical activity and ablated by local radiofrequency delivery. Outcome was assessed by Holter and defibrillator memory interrogation. Premature beats were elicited from the Purkinje conducting system in 23 patients: from the left ventricular septum in 10, from the anterior right ventricle in 9, and from both in 4. The interval from the Purkinje potential to the following myocardial activation varied from 10 to 150 ms during premature beat but was 11±5 ms during sinus rhythm, indicating location at peripheral Purkinje arborization. The premature beats originated from the right ventricular outflow tract muscle in 4 patients. The accuracy of mapping was confirmed by acute elimination of premature beats during local radiofrequency delivery. During a follow-up of 24±28 months, 24 patients (89%) had no recurrence of ventricular fibrillation without drug.

Conclusions—Primary idiopathic ventricular fibrillation is a syndrome characterized by dominant triggers from the distal Purkinje system. These sources can be eliminated by focal energy delivery. (Circulation. 2002;106:962-967.)

Key Words: ablation ■ death, sudden ■ heart arrest ■ fibrillation ■ mapping
Electrical Storm Early after AMI

- 4 pts with drug-refractory repetitive VF, despite revascularization and Rx w. amiodarone and betablockers
- Short, HF. low-amplitude potentials (PLP) preceding PVCs (120-160ms)
- Purkinje potentials recorded at the site in SR 23-26 ms
- Site was close to the border zone of MI, after ablation - no recurrences of VT/VF for 33, 14, 6, and 5 months


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Pathogenesis?
Survival of Subendocardial Purkinje Fibers after MI

- dog model, two-stage ligation of the LAD
- bipolar EGMs from subendocardial layers of infarct demonstrated Purkinje fiber activity only

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How it Could Look Like?
3D Mapping = Tool for Annotation
Not Always the Narrowest is a Trigger..
Pragmatic Ablation Approach in Case of Absent Ectopy

First procedure

Procedure day after
 Awareness is important!!

12-lead ECG of triggering ectopic activity

Trigger is almost invariably localized within the Purkinje network, very subendocardially

“Bump“ into the focus region may transiently abolish ectopy (similarly LBBB caused by catheter manipulation)

If no ectopy is manifest during mapping, catheter ablation could target conduction system of the septum close to margin of the scar
Principles of Mapping of Reentrant VTs
Anatomical Substrates for Reentry

CAD – post MI

ARVC

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SMVT in Structural Heart Disease

Reentry is more frequent - figure 8 reentry

ECG
Conventional Mapping
Conventional techniques to guide catheter ablation

- ECG analysis of clinical VT
- sinus rhythm mapping/pacemapping
- activation sequence mapping in VT
- middiastolic potentials
- entrainment mapping

N.B. The use of last 3 strategies requires haemodynamic tolerability of VT
## ECG Analysis of Clinical VT

<table>
<thead>
<tr>
<th>V₁</th>
<th>Front. axis</th>
<th>V₄</th>
<th>LV location</th>
</tr>
</thead>
<tbody>
<tr>
<td>LB</td>
<td>L. superior</td>
<td>S or qS</td>
<td>Apical septum</td>
</tr>
<tr>
<td>LB</td>
<td>L. superior</td>
<td>R</td>
<td>Basal septum</td>
</tr>
<tr>
<td>LB</td>
<td>Inferior</td>
<td>-</td>
<td>Anterior septum</td>
</tr>
<tr>
<td>RB</td>
<td>L. superior</td>
<td>R or rS</td>
<td>Inferobasal</td>
</tr>
<tr>
<td>RB</td>
<td>L. superior</td>
<td>S</td>
<td>Apic. inf. / Apico-septal</td>
</tr>
<tr>
<td>RB</td>
<td>R. superior</td>
<td>S or rS</td>
<td>Apic. inf. / Apico-septal</td>
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<td>RB</td>
<td>R. superior</td>
<td>R</td>
<td>Inferobasal</td>
</tr>
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<td>Inferior</td>
<td>S</td>
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</tr>
<tr>
<td>RB</td>
<td>R. superior</td>
<td>R</td>
<td>Basal lateral</td>
</tr>
</tbody>
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Reentrant Ventricular Tachycardia

Sinus Rhythm Mapping

- identification of late potentials
- pace mapping
- assessment of slow conduction zones (S-QRS > 40 ms)
Pacing in SR (in LP Region)

S-QRS > 40 ms

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Reentrant Ventricular Tachycardia

Middiastolic Potentials

Map 1.2
Reentrant Ventricular Tachycardia

Entrainment Mapping

QRS

1. Entrance
2. Central Zone
3. Exit
4. Bystander
5. Outer Loops
6. Inner Loops

Schematic Arrhythmia Circuit
Middiastolic Potentials and Concealed Entrainment

Central zone of slow conduction

Concealed entrainment

Prolonged S-QRS

aVR
aVL
aVF
V1
V2
V3
V4
V5
V6

PPI=VT

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Ablation within the Central Zone
Substrate Mapping and Integrated Approach
VT in Structural Heart Disease

Bipolar voltage map
Normal LV myo 4.8±3.1 mV
Scar < 1.5 mV
Dense scar < 0.5 mV
Border zone = adjacent to dense scar

9 CAD pts and 7 CMP pts w. nonmappable VTs
RF sequential point lesions 1-2 min at 50-60C for linear lesions
81% free of VT, the rest but one improved

Marchlinsky et al, Circulation 2000;101:1288
Endocardial Conduction Channels

- 26 pts w uniform VT
- Electroanatomical mapping
- Entrainment mapping in 53 VTs
- Color scheme adjusted to identify conduction channels within the scar
  - 47/56 (86%) of entrance or isthmus sites located within dense scar (<0.5 mV)
  - 92% of exit sites located in border zone (0.5-1.5 mV)
  - Voltage range of channels ranged from 0.1-0.7 mV

Endocardial Conduction Channels

A
Right Ventricular Cardiomyopathy

B
Infarct-related Cardiomyopathy

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• 31 of 51 pts (30 men, mean age 64±11 years) w. documented VT after MI (13 anterior, 15 inferior, 2 both, 1 inferior and lateral)

• Primary untolerated VT in 16 pts (CL 335 ±52 ms) and both tolerated and untolerated VT in 15 pts (CL 464±85 ms)

• 104 VTs (34 stable, 70 unstable) induced (mean of 3.4±1.4 VT/patient)

• Clinical VT abolished in 30/31 (96.7 %)

• Noninducibility of any VT - 19/31 (61 %)

An Example of Integrated Approach

65-year-old man w. a history of remote anterior MI

3 years after ICD implant for monomorphic VT

Admitted for multiple ICD therapies
Documented VTs

Inducibility of multiple VT morphologies
Mapping in Sinus Rhythm
Delineation of arrhythmogenic substrate

• Creation of voltage map during sinus rhythm
• Annotation of
  – low amplitude
  – fragmented signals
  – late potentials
• Pacemapping
  – Allocation of exit for inducible VTs
  – Scar tag in case of noncapture at 10V
Mapping in Sinus Rhythm
Pacemapping I

Clinical VT

Pacing with very long S-QRS interval

Pacing site in central zone
Mapping in Sinus Rhythm
Pacemapping II

Clinical VT

Pacing with shorter long S-QRS interval

Pacing site near exit
Central zone – concealed entrainment, PPI=CL, long S-QRS interval
Visualization of the Scar

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Subendocardial Substrate

Contact Force Map

Adjusted Bipolar Voltage Map (LPs)
Specific Situation in Congenital Heart Disease Patients
ToF- Anatomical Isthmuses


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"No Channel = No Reentry"

- 15 VTs (mean cycle length, 276±78 ms; 73% poorly tolerated)
- reentry circuit isthmuses of all VTs located in an anatomic isthmus
- 11 of 15 VTs had reentry isthmus in anatomic isthmus 1
- Transecting the anatomic isthmuses by ablation abolished all VTs
- FU: 30.4±29.3 months, 91% pts free of VT

Typical Isthmuses


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Another Example
Case 1

- 79-year-old male with CAD, after anterior wall MI, systolic LV dysfunction EF 20-25%
- 1999 implantation of PM (DDD)
- 2005 upgrade to BiV ICD for documented VT – defibrillator lead Medtronic 6949-65cm
- 2008 repeated paroxysms of VT with CL 300 ms, nonsustained
BiV Pacing
LV Pacing
1. Focal mechanism plays presumably role with focus in vicinity of the RV lead
2. VT is presumably of reentrant origin
3. VT originates from the left ventricle
4. VT is most probably epicardial
Case 1

Acceleration during ablation in RV
Case 2

- 65-year-old man
- History of HCMP w/o obstruction
- Admitted for heart failure
- Incessant monomorphic VT (135 bpm)
- Failed catheter ablation for similar VT in the past
Case 2

65-year-old patient with a history of HCMP w/o obstruction, presenting with heart failure and incessant VT (HR 135 bpm), failed previous endocardial ablation.
Q1

1. VT originates from the right ventricle
2. VT originates from the left ventricular apex
3. VT is most probably epicardial from left lateral wall
4. VT is most likely bundle branch reentry
Epicardial electroanatomical map
Case 3

- 62-years old female with a history of rheumatic heart disease
- Eight years after mitral and tricuspidal valve replacement with mechanical prothesis
- Presented with ventricular tachycardia requiring electrical cardioversion
Case 3

- VTs of two morphologies inducible during EPS
Q1

• The exit zone of VTs is:
  1. Basal, inferolateral LV
  2. Basal, posteroseptal LV
  3. Apical, inferolateral LV
  4. Apical, posteroseptal LV
  5. RV origin is likely
Case 3

• Concealed entrainment
Case 3
Case 4

- 36-year-old man with a history of aortic valve disease (10 years)
- Admitted to hospital after syncope preceded with palpitations
- ECG documented tachycardia with broad QRS (185/min)
- Cardioverted by amiodarone i.v.
- Early recurrence requiring DC shock
- Bicuspid aortic valve w. AR 4/5, LVEDD 56 mm with normal LVEF, dilation of asc aorta to 58 mm
Clinical arrhythmia and SR
Q1

• Clinical arrhythmia is most probably?
  a) Atrial tachycardia with aberrant conduction
  b) Antidromic AV reentry
  c) VT originating from conduction system
  d) VT due to intramyocardial reentry
  e) VT of epicardial origin
VA dissociation
Spontaneous change
Entrainment v oblasti levého zadní fasciklu LK
Clinical tachycardia is most probably?
1. Idiopathic fascicular VT
2. Bundle branch reentry
3. Intramyocardial reentry with critical isthmus close to mitral annulus
4. Focal VT from MP papillary muscle
Intracardiac signals
RB ablation