Epicardial Mapping and Ablation of VENTRICULAR TACHYCARDIA

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Epicardial Ablation of VT

Background
Electrocardiography
Anatomy
Access/ Epicardial Approach
Myocardial Anatomical Structure /Imaging
Advanced Mapping and Ablation/Outcomes
Complications of VT Ablation
Surgical Approach
Program/Lab Requirements
Lab and Case Management
VT Treatment – Milestones

- 1956 Zoll- external defibrillation
- 1959 First pacemaker implanted in Sweden
- 1962 Lown – synchronized DC cardioversion
- 1969 First His Bundle recording – start of “EP” era!
- 1987 First RF ablation procedure reported
- 1995 Mapping Systems introduced
- 1996 Sosa – described Epicardial Access for VT ablation in Chagas cardiomyopathy
- 2000s Mapping systems improve; Cooled tip catheters, ICE, Epicardial ablation expands
Epicardial Ablation of VT

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Willem Einthoven (Semarang, May 21, 1860 – Leiden, September 29, 1927)

The Nobel Prize in Physiology or Medicine 1924
QRS Morphology as Guide to VT Exit

- LBBB pattern in V1 suggest exit in RV or interventricular septum
- Dominant R waves in V1 indicate LV exit
- Superior axis => inferior wall exit
- Inferior axis => anterior wall
- Dominant S waves in V3,V4=>apical exit
- Dominant R waves in V3,V4=>basal exit

Electrocardiographic Recognition of the Epicardial Origin of Ventricular Tachycardias

Antonio Berruezo, MD; Lluis Mont, MD; Santiago Nava, MD; Enrique Chueca, MD; Eduardo Bartholomay, MD; Josep Brugada, MD, PhD

Background—Some ventricular tachycardias (VTs) originating from the epicardium are not suitable for endocardial radiofrequency ablation and require an epicardial approach. The aim of this study was to define the ECG characteristics that may identify an epicardial origin of VTs.

Brugada Criteria for Epicardial VT Origin

Pseudo delta >= 34 ms
Intrinsicsoid deflection > 85ms
Shortest RS complex > 121ms
Maximum Deflection Index (MDI) > 0.55

Predominantly ischemic cardiomyopathy patient group

Key Words: electrocardiography ■ pericardium ■ tachycardia, ventricular
MDI Maximum Deflection Index defined as the ratio of the interval from the onset of ventricular activation to the peak of QRS in a precordial lead to the QRS duration

IDT Intrinsicoid Defection Time is the interval from the onset of ventricular activation to the peak of the R wave in lead V2

Pseudodelta Interval from the onset of ventricular activation to the onset of the earliest rapid deflection in any precordial lead.
ECG Criteria to Identify Epicardial Ventricular Tachycardia in Nonischemic Cardiomyopathy

**Probable Epicardial Origin (based on interval criteria)**

1. Presence of inferior q waves?
   - NO
   - YES: No EPI VT
2. Pseudo-delta ≥ 75 ms?
   - NO
   - YES: EPI VT
3. MDI ≥ 0.59?
   - NO
   - YES: EPI VT
4. Presence of q wave in lead I?
   - NO
   - YES: EPI VT

**Sensitivity (SN) = 96%**
**Specificity (SP) = 93%**
Epicardial VT – from inferior LV surface
Multiple VT’s with basal exits

Hutchinson MD, Marchlinski FE: Epicardial Ablation of VT in patients with non ischemic LV cardiomyopathy: Cardiac Electrophysiology Clinics, 2(1);93-103. 2010
Epicardial Ablation of VT

Background
Electrocardiography

**Anatomy**
Access/ Epicardial Approach
Myocardial Anatomical Structure /Imaging
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Complications of VT Ablation

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Lab and Case Management
Pericardial sinuses
Epicardial Ablation of VT

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Lab and Case Management
Staining the pericardium
Confirming pericardial entry
Advance wire into pericardial space
LAO view
Oblique sinus
Sheath across the transverse sinus
Coronary arteriography
Epicardial Ablation of VT

Background
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Surgical Approach
Lab and Case Management
Peri-Procedural Imaging

Transthoracic Echo
baseline EF, wall motion, …

MRI/CT scan
Confirm/predict scar location

Transesophageal Echocardiogram (TEE)
Exclude intracardiac thrombus

Intracardiac Echocardiography (ICE)
Guide transseptal and monitor for pericardial effusion
Evaluation of Viability and Myocardial Perfusion with Contrast Enhanced MRI

Gadolinium contrast injection

Normal Myocardium

Infarcted Myocardium

First-Pass

Delayed Enhancement

time
Detection of Myocardial Infarction in Patients with Ischemic, Nonischemic and Mixed Cardiomyopathy by Contrast-Enhanced MRI: Bello D, Shivkumar K; Magnetic Resonance Imaging 2010
FROM SCAR TO VENTRICULAR ARRHYTHMIAS
Epicardial Ablation of VT

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Myocardial Anatomical Structure /Imaging
Advanced Mapping and Ablation/Outcomes
Complications of VT Ablation
Surgical Approach
Program/Lab Requirements
Lab and Case Management
Influence of Hemodynamic Status

- **Stable**
  - Mapping Techniques
    - Activation
    - Pace
    - Entrainment
    - Substrate

- **Unstable**
  - Mapping Techniques
    - Pace
    - Substrate

Hemodynamic support - IABP, Ventricular Assist Devices
Theoretical reentry circuits related to an inferior wall infarct scar are shown.
A: Electrograms types recorded from 2 mm bipolar electrodes with a 5-10 mm interelectrode distance filtered at 30-500 Hz.

B: Example of isolated late split potential recorded during V pacing.

Value of high-density endocardial and epicardial mapping for catheter ablation of hemodynamically unstable ventricular tachycardia

David A. Cesario, MD, PhD,* Marmar Vaseghi, MD,* Noel G. Boyle, MD, PhD,* Michael C. Fishbein, MD,† Miguel Valderrábano, MD,* Calambur Narasimhan, MD,‡ Isaac Wiener, MD,* Kalyanam Shivkumar, MD, PhD*

*From the UCLA Cardiac Arrhythmia Center, Division of Cardiology, Department of Medicine, David Geffen School of Medicine at UCLA, Los Angeles, California,
†Division of Anatomic Pathology, David Geffen School of Medicine at UCLA, Los Angeles, California, and
‡CARE Cardiovascular Institute, Hyderabad, India.

Cesario D et al Heart Rhythm 2006 ;3:1-10
## Patient Characteristics

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Antiarrhythmic drugs</th>
<th>Etiology</th>
<th>Previous ablation</th>
<th>Ejection fraction (%)</th>
<th>Endocardial points</th>
<th>Epicardial points</th>
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<td>1</td>
<td>Male</td>
<td>76</td>
<td>Amio + Mex</td>
<td>Ischemic</td>
<td>1</td>
<td>20</td>
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<td>Amio</td>
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<td>254</td>
<td>154</td>
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<td>Amio + Mex + Lido</td>
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<td>0</td>
<td>35</td>
<td>293</td>
<td>165</td>
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<td>Amio + Mex</td>
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<td>25</td>
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<td>52*</td>
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<td>78</td>
<td>Amio + Lido</td>
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<td>15</td>
<td>356</td>
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<td>386</td>
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<tr>
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<td>Amio</td>
<td>Valvular/dilated</td>
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<td>273</td>
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<td>56</td>
<td>Amio</td>
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<td>296</td>
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<td>286</td>
<td>200</td>
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<tr>
<td>14</td>
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<td>75</td>
<td>Amio</td>
<td>Ischemic</td>
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<td>352</td>
<td>62</td>
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<td>37</td>
<td>Amio</td>
<td>Dilated (nonischemic)</td>
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<td>45</td>
<td>358</td>
<td>320</td>
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<tr>
<td>16</td>
<td>Male</td>
<td>79</td>
<td>Amio + Mex</td>
<td>Ischemic</td>
<td>0</td>
<td>25</td>
<td>286</td>
<td>143</td>
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<tr>
<td>17</td>
<td>Male</td>
<td>60</td>
<td>Amio + Mex</td>
<td>Dilated (nonischemic)</td>
<td>1</td>
<td>25</td>
<td>361</td>
<td>289</td>
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<tr>
<td>18</td>
<td>Male</td>
<td>56</td>
<td>Amio + Sotalol</td>
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<tr>
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<td>Male</td>
<td>69</td>
<td>Amio + Lido</td>
<td>Ischemic</td>
<td>1</td>
<td>35</td>
<td>285</td>
<td>132</td>
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<tr>
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<td>52</td>
<td>Amio + Mex</td>
<td>Ischemic</td>
<td>1</td>
<td>15</td>
<td>327</td>
<td>96</td>
</tr>
</tbody>
</table>

*Amio = amiodarone; HCM = hypertrophic cardiomyopathy (late-stage presentation as dilated cardiomyopathy); Lido = lidocaine; Mex = mexiletine.

*The average number of therapies (antitachycardia pacing or shocks) was 10 ± 3 in the week prior to ablation (data available for 11/20 patients).

Heart Rhythm, 2006;3:1-10
Epicardial Map: Non-Ischemic Cardiomyopathy

TARGETED VT

EPICARDIAL VOLTAGE MAP

Cesario D et al *Heart Rhythm* 2006;3:1-10
Ischemic Cardiomyopathy: Ablation Strategy – Endocardial Maps

A

B

C

Posterior Scar

D

E

Lateral Scar

F

Scar

Ablation line

AV valve annulus

Combined Endocardial and Epicardial ablation for VT - Patient Population

20 patients with hemodynamically unstable VT
15M, 5F; mean age 63 ± 11 years
12 ischemic, 8 nonischemic CM; Mean EF 28 ± 10%

Average ICD therapies: 10 ± 3 in week pre ablation
Epicardial ablation (in addition to endocardial) was required in 8/20 (40%) of the patients

Table 3 Epicardial ablation

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Etiology</th>
<th>Scar location</th>
<th>Targeted ventricular tachycardia cycle length CL (ms)</th>
<th>Epicardial ablation strategy</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Male</td>
<td>76</td>
<td>Ischemic</td>
<td>Posterolateral</td>
<td>320</td>
<td>Substrate mapping</td>
</tr>
<tr>
<td>3</td>
<td>Male</td>
<td>70</td>
<td>Ischemic</td>
<td>Posterolateral</td>
<td>280</td>
<td>Substrate mapping</td>
</tr>
<tr>
<td>5</td>
<td>Female</td>
<td>61</td>
<td>Ischemic</td>
<td>Inferomedial</td>
<td>420</td>
<td>Substrate mapping</td>
</tr>
<tr>
<td>8</td>
<td>Male</td>
<td>63</td>
<td>Ischemic</td>
<td>Inferomedial</td>
<td>342</td>
<td>Substrate mapping</td>
</tr>
<tr>
<td>13</td>
<td>Male</td>
<td>65</td>
<td>Dilated (nonischemic)</td>
<td>Inferior</td>
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<td>Substrate mapping</td>
</tr>
<tr>
<td>16</td>
<td>Male</td>
<td>79</td>
<td>Ischemic</td>
<td>Posterolateral</td>
<td>242, 284</td>
<td>Substrate mapping</td>
</tr>
<tr>
<td>17</td>
<td>Male</td>
<td>60</td>
<td>Dilated (nonischemic)</td>
<td>Inferior</td>
<td>292</td>
<td>Substrate mapping</td>
</tr>
<tr>
<td>20</td>
<td>Male</td>
<td>52</td>
<td>Ischemic</td>
<td>Posterolateral</td>
<td>364</td>
<td>Substrate mapping</td>
</tr>
</tbody>
</table>

Cesario D et al Heart Rhythm 2006;3:1-10
Ablation Results for Combined Endocardial and Epicardial VT Ablation

70% 1 year event free survival from VT

n=20

Cesario D et al *Heart Rhythm* 2006;3:1-10
Mapping Techniques Have Greatly Improved

Characterization of the Arrhythmogenic Substrate in Ischemic and Nonischemic Cardiomyopathy

Implications for Catheter Ablation of Hemodynamically Unstable Ventricular Tachycardia

Shiro Nakahara, MD, PhD, Roderick Tung, MD, Rafael J. Ramirez, PhD, Yoav Michowitz, MD, Marmar Vaseghi, MD, Eric Buch, MD, Jean Gima, RN, MN, NP, Isaac Wiener, MD, Aman Mahajan, MD, PhD, Noel G. Boyle, MD, PhD, Kalyanam Shivkumar, MD, PhD

ICM: n=16
12mth success: 82%

NICM: n=17
12mth success: 50%
Knowledge Gained about VT Circuits: ICM vs NICM

- MMVT Circuits Are Deep Within Scar: **ICM > NICM**
- Scar Border Zones Are Critical: **NICM**
- Late potentials an ‘electrical footprint’: **ICM > NICM**
- Clinical Imaging Is Providing New Insights
- Epicardial vs Endocardial Scar: **NICM > ICM**
- Interplay Between Structural VT And Functional Components: **NICM possibly > ICM**
Epicardial Ablation of VT

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Program/Lab Requirements
Lab and Case Management
Complications with Epicardial Approach

- Pericardial inflammation/infection
- **Coronary arterial damage**
- Hemopericardium
- Phrenic nerve damage
- Liver laceration
- Peritoneal bleeding secondary to diaphragmatic arterial laceration
Complications with Epicardial Approach

- Pericardial inflammation/infection
- Coronary arterial damage
- Hemopericardium
- Phrenic nerve damage
- Liver laceration
- Peritoneal bleeding secondary to diaphragmatic arterial laceration
Targeted VT

LMCA

Epi

Endo
Intraoperative Cryoablation
Ablation Site: Pacing via ablation catheter

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Epicardial VT Originating from LV base in a patient with a VAD
Epicardial VT Originating from LV base in a patient with a VAD
Surgical Access to LV base via lateral thoracotomy and Epicardial ablation with closed irrigated catheter and surgical ablation pen
Post-ablation Images of Epicardial Aspect of LV

UCLA Cardiac Arrhythmia Center
Epicardial Ablation of VT

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- Surgical Approach
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- Lab and Case Management
Requirements for VT Ablation Program

**Institutional/Lab**
- Biplane EP lab
- Digital pulsed fluoroscopy
- EP recording and Electroanatomic mapping systems
- RF and cooled tip ablation equipment
- TEE, Intracardiac echo

**Personnel**
- EP Physicians with adequate technical skills and volume of experience
- Epicardial access skills
- Adequately trained laboratory staff
- Anesthesia Support
- Imaging, Heart Failure, Interventional and CT Surgery support

VT ABLATION CHECKLIST – EP TEAM

PRE-PROCEDURE CHECKLIST (TO BE FILLED OUT AND FAXED TO CATH LAB DAY BEFORE CASE)

Date of procedure:__________ Device: NONE PM ICD BIV BSC BTK MDT SJM ELA

Allergies (reaction):__________ Research study patient: YES NO

On Coumadin? YES NO If YES, held for ____ days TEE done already? YES NO

On Plavix? YES NO If YES, held for ____ days Images to download: NONE MR CT

Mapping: NAVX CARTO Epicardial prep: YES NO

ABLAITION CHECKLIST (TO BE USED DURING PROCEDURE)

Patient preparation
☐ MD aware of allergies: ____________ INR ________ Plts ________ K+ ________ Preg ________ (<60 yrs)

☐ Imaging: MRI/CT burned to disk for NavX

☐ Consents done: Cardiac Catheterization, EP study, Catheter Ablation, Transseptal, TEE, (Epicardial access if planned)

☐ Wrist restraints, NavX patch, defib patches, grounding pads, Bovie set to cutting 45W

☐ Foley catheter (only after completion of TEE, page GUI if ANY difficulty)

☐ TEE to be performed or results of recent (<48 hrs) test available

☐ ICD/PM device programmer in room, ICD therapies disabled (do not turn off therapy for any open-chest subxiphoid case)

Anesthesia
☐ TEE to be performed or results of recent (<48 hrs) test available

☐ General anesthesia with endotracheal intubation, no skeletal muscle relaxants

☐ MAC is preferred for focal VT (RVOT, LVOT, fascicular)

☐ IV filters for all infusion lines

Equipment to have ready (not opened until MD in room)

☐ Sheaths: LFV (10F duo, 8F or 11F for ICE), RFV (8F, 8F), RIJ (8F locking), RFA (4F)

☐ Catheters: hex, CRD2, 7F CSL, 4mm irrigated-tip Ablation catheter

☐ ICE catheter (8F or 10F), Acuson or Vivid 7 machine, NavX or Carto

☐ Double transseptal equipment: 8F Mullins (2), pressure transducer, heparinized flush with Pall filter (2), BRK-1 needle, Toray wire

(Ef epicardial access planned)

☐ Prepare subxiphoid skin and administer 1g Ancef (or Vanco for PCN allergy)

☐ Equipment: new 22G needle and scalpel, long and short Tuohy needles, 5F Cook dilator, short guidewire, additional SL0 8F sheath, Meditech XXL balloon

☐ Left heart kit with manifold ready for coronary injection

☐ Solumedrol 125 mg mixed with 10cc normal saline

☐ Type and Cross 2 units pRBCs

End of case

☐ Cine of both hemidiaphragms recorded moving spontaneously if epicardial ablation

☐ ICE clips stored to document absence of pericardial effusion

☐ ICD therapy turned back on
Catheter Ablation of Ventricular Tachycardia

Roderick Tung, MD; Noel G. Boyle, MD, PhD; Kalyanam Shivkumar, MD, PhD

Ventricular tachycardia (VT) is an abnormal rapid heart rhythm originating from the lower pumping chambers of the heart (ventricles). The normal heart usually beats between 60 and 100 times per minute, with the atria contracting first, followed by the ventricles in a synchronized fashion. In VT, the ventricles beat at a rapid rate, typically from 120 to 300 beats per minute, and are no longer coordinated with the atria. The controlled contraction of the ventricles is important for the heart to pump blood to the brain and the rest of the body and to maintain a normal blood pressure. Abnormal and fast rhythms from the ventricle may impair the ability of the pump to supply blood to the brain and the rest of the body as a result of the rapid rate and weak contractions. This may result in palpitations (a feeling of rapid or abnormal heart beat), dizziness, lightheadedness, or syncope (loss of consciousness). If the heart rate increases to more than 300 beats per minute and becomes totally uncoordinated, this is usually called ventricular fibrillation (VF), which will cause sudden cardiac death.

VT occurs most commonly in patients with weakened heart muscle (cardiomyopathy) or when scar tissue develops in the heart. In patients with coronary artery disease (blockage of blood vessels on the surface of the heart), this scar is the result of a prior heart attack (myocardial infarction) when the muscle dies as a result of a blockage in blood flow. Scar, or fibrosis, can interfere with the normal electrical impulse in the heart, leading to a short-circuiting of the rhythm, called reentry. VT can also occur in patients with normal hearts by a different mechanism whereby the electrical conduction is overtly excitable, like a muscle twitch.

Sudden cardiac death causes about 450,000 fatalities each year in the United States alone. It is most commonly caused by VT deteriorating into VF, which is fatal within a few minutes if not defibrillated (shocked) back to a normal rhythm. (Figure 1A) Defibrillation may be accomplished by an automated external defibrillator or an implantable cardioverter-defibrillator (ICD). It is important to distinguish VT and VF, which are electric problems of the heart, from a heart attack, which is due to the sudden blockage of an artery. Heart attacks are treated with clot-busting drugs, balloon angioplasty, or stents. Sometimes, VT and VF are seen in that setting and are treated with electric shocks and drugs. The treatment of abnormal rhythms is discussed below.

Treatment Options

There are 3 treatment options for VT, although many patients require a combination: an ICD, antiarrhythmic medications, or catheter ablation. Many patients at risk for VT are treated with an ICD. This is the most effective method of treating potentially life-threatening rhythm such as VT or VF back to a normal rhythm. However, an ICD does nothing to prevent the heart from going into VT. The ICD is a "safety net" and is like having an ambulance crew accompany you 24 hours a day.

Antiarrhythmic medications that modify the conduction of the electric impulse of the heart can be effective in suppressing VT. These medications can reduce the risk of recurrence by 75% but have potential side effects that include proarrhythmia, or worsening of the heart rhythm. For this reason, initiation of antiarrhythmic agents often requires close monitoring. Amiodarone, the most effective drug, has many side effects, which can involve toxicity to the vital organs such as the liver, thyroid, lungs, eyes, and skin. Because of the discomfort associated with frequent ICD shocks and the side effects of antiarrhythmic drugs, catheter ablation is an additional treatment option for many patients already using these therapies.

Catheter Ablation Therapy

Catheter ablation has been used to treat heart rhythm disorders for more than 25 years. This procedure targets the origin of the VT by placing a long, thin wire or catheter into the heart chambers through the veins of the leg. The catheter is inserted through intravenous ports, or sheaths, placed in the veins in the groin and sometimes through a vein on the side of the neck. To access the left ventricle, a needle may be used to create a small puncture in the wall between the right and left sides of the heart under ultrasound guidance (called transesophageal catheterization). Alternatively, a catheter can be inserted into the heart through an artery in the groin (similar to heart catheterization procedures). The ablation catheter is moved around the ventricle, and a virtual 3-dimensional image of the heart is created with a computer mapping system that acts like a navigation system (Figure 2). The location of the catheter is determined by use of fluoroscopy (x-ray) and this mapping system. Typically, the procedure lasts from 3 to 6 hours.

In some instances, the physician determines that the VT may originate from a circuit on the outer surface of the heart, or epicardium. If this is the case, a puncture to the sac, or pericardium, around the heart is performed just beneath the breastbone. This enables the ablation catheter to be inserted and maneuvered through the pericardium to determine whether the VT originates there. If a patient has a previous history of open heart surgery, a small surgical incision may be necessary to access the pericardium because of the presence of scar tissue, which can make the pericardium stick to the heart. These procedures to access the epicardium are usually performed at highly experienced centers.

Afterward, the catheters are removed, but the sheaths are left in until the blood thinner wears off. Typically, this occurs about 7 to 10 days. The patient is usually discharged from the hospital about 1 week after the procedure.
Epicardial Ablation of VT

Background
Electrocardiography
Anatomy
Access/ Epicardial Approach
Myocardial Anatomical Structure /Imaging
Advanced Mapping and Ablation/Outcomes
Complications of VT Ablation
Surgical Approach
Program/Lab Requirements
Lab and Case Management
Flow Chart: Suggested Approach to Epicardial Access/Ablation

1. ECG suggest Epicardial VT exit site
   - NO
   - YES

2. Prior unsuccessful Endocardial Ablation
   - NO
   - YES

3. Define SCAR location with CE imaging: subendocardial or mid-myocardial scar
   - NO
   - YES

4. Consider likelihood of Epicardial circuit for Underlying Substrate:
   - LOW
   - HIGH

Perform endocardial mapping and ablation first

A
ECG Criteria (Ref 27)
1) pseudo-delta >34 ms
2) intrinsicoid deflection time (v2) >85 ms
3) Shortest RS complex >121 ms
4) ORS duration >211 ms

B
ECG Criteria for NICM (ref 29)
1) Absence of inferior Q wave
2) pseudo-delta ≥75 ms
3) MDI >0.59
4) Presence of Q wave in lead I

C
Probability of Epicardial Focus (ref 7)
- Normal 6%
- ICM 16%
- NICM 35%
- ARVD 41%
- Other CM 18%
UCLA Epicardial VT Ablation Playbook

Hour 1  Prep, Anesthesia, TEE, NIPS
Hour 2  Venous Access, Catheter placement, Epicardial Access, (Coronary Angiography) Transseptal(s)
Hour 3  Endocardial Mapping, Epicardial mapping
Hour 4  Assess induction and mapping data; develop ablation plan and endpoints
Hour 5  Ablation progressing ……..
Hour 6  Reassess ablation plan/endpoints.
Hour 6+ Wrap up !
Time Zones for VT Ablation Procedures

Hour 1-3  Zone of Entrainment !
Hour 4   Zone of Entertainment !
( Show VT ECGs and voltage maps to visitors !)
Hour 5-6  Ablation Zone
Hour 7  Fatigue zone
Hour 8  Frustration zone
Hour 9  Futility zone
Time Zones for VT Ablation Procedures

Hour 1-3  Zone of Entrainment!
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          (Show VT ECGs and voltage maps to visitors!)
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Hour 7    Fatigue zone
Hour 8    Frustration zone
Hour 9    Futility zone
Hour 10+  Martyrdom zone
Epicardial Interventions in Electrophysiology

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