Ideopathic Ventricular Tachycardia: The Usual and Unusual

Susan Blancher, MN, ARNP, CEPS
Virginia Mason Medical Center
Seattle, WA
Sustained Ventricular Tachycardias

- **Polymorphic VT**
  - Acute ischemia
  - Abnormalities of ion channels (long/short QT, Brugada, CPMVT)
  - Idiopathic ventricular fibrillation
  - Structural heart disease: (Hypertrophy, recent MI, Cardiomyopathy)

- **Monomorphc VT**
  - Scar-related reentry (Cardiomyopathies: idiopathic, viral, sarcoid, Chagas, aneurysms, ARVD; previous MI, surgical scar)
  - Purkinje Disease (BBRT, automaticity)
  - Idiopathic VT
    - Outflow Tract: RVOT, LVOT
    - Left fascicular VT (Belhassen’s VT)
Definition and Classification

- Ideopathic: “Cause Unknown”
- “VT in the absence of apparent structural HD”
- Multiple classification schemes (presentation, ECG, mechanism, response to meds, ventricular origin) has led to confusion
- Current use: MMVT, absence of structural heart disease, benign syndrome
- Excludes disorders associated with SCD
Ideopathic VT

I. Outflow Tract VT
   A. RVOT: Pulmonary Artery to His region
   B. LVOT: Aortic cusps, Aortomitral continuity, Epicardial, Mitral Annulus

II. LV Fascicular VT
   A. Posterior fascicular
   B. Anterior fascicular
   C. Upper septal
Distribution of VT

Total VT = 90% structural heart disease, 10% ideopathic

N = 122 undergoing ablation for IVT 1999-2003
• RVOT : 88 (72%)
• LVOT: 20 (16%) (9 aortic cusps, 11 basal LV endocardium)
• Fascicular VT : 14 (11%)

Dixit S, Lin D, Zado E., Heart Rhythm 1:S104m 2004
Outflow Tract VT
Symptoms and Presentation

- 70% RVOT are female; 70% LVOT are male
- Early age: 20-50 years
- Frequent PVC’s, Salvos of VT, or sustained VT
- Symptoms include palpitations and lightheadedness, dyspnea, CP, syncope
- Often provoked by exercise, stress, anxiety, stimulants, hormonal trigger in females, PVC’s and salvos can occur at rest or recovery from exercise, circadian pattern
- Lack of structural heart disease is the rule, subtle abnormalities may be present
Relationship of Heart Rate to Frequency of Ectopy From 3 Representative Patients

A

Heart rate (bpm)

# PVCs

B

Heart rate (bpm)

# PVCs

C

Heart rate (bpm)

# PVCs

# VT runs

Kim, RJ. et al. J Am Coll Cardiol 2007;49:2035-2043
Copyright ©2007 American College of Cardiology Foundation. Restrictions may apply.
Identical Morphologies of PVCs, NSVT, and Sustained VT From a Patient With Exercise-Induced VT

Kim, RJ. et al. J Am Coll Cardiol 2007;49:2035-2043

Copyright ©2007 American College of Cardiology Foundation. Restrictions may apply.
Mechanism of Outflow Tract VT

Delayed Afterdepolarizations
Anatomy of Cardiac Myocyte

The spiral arrangement of ventricular muscle allows ventricular contraction to squeeze the blood upward from the apex of the heart.

Intercalated disks contain desmosomes that transfer force from cell to cell and gap junctions that allow electrical signals to pass rapidly from cell to cell.

Role of Action Potential and Ca++ in Muscle Contraction

- Path of AP
- Surface of muscle fiber
- T tubule
- Myofibrils
- Sarcoplasmic reticulum
- Ca++
- Sarcomere
Calcium Overload causes DAD’s

1. Activation of beta-receptor (epi, isuprel)
2. G-protein stimulation
3. Adenylate cyclase stimulation
4. Increase of cAMP (via ATP)
5. PKA activation
6. Increase intracellular Calcium from SR and L channels
7. Increase transient Na influx via Na/CaX
8. Results in delayed afterdepolarization
9. If reaches threshold sets off AP and triggered arrhythmia

cAMP dependant Calcium Overload is Generated which leads to DAD via Na-CaX
Normal Action Potential
DAD’s and Triggered Beats

**AFTERDEPOLARIZATIONS**

- Early Afterdepolarization (during rapid repolarization)
- Delayed Afterdepolarization (following complete repolarization)

**Delayed Afterdepolarization (DAD)**

- +10 mV
- -90 mV

**DAD**

- AP
- I_{NCX}
- CaT

Abnormal Ca^{2+} release

**Normal AP**

**Afterdepolarization**

**Single triggered AP**

**Sustained triggered activity**
RVOT VT Origin

- 70-80% septal
- 20-30% freewall
- Majority are septal, just below Pulmonary Valve, anterior
- Can be above Pulmonary valve
- Can be low, near HIS
Case Study: RVOT VT

- 37 year old female pediatrician
- Long history of asymptomatic but frequent PVC’s
- Recent near syncopal event while skiing (no family hx of syncope or sudden death)
- In ED very frequent unifocal PVC’s
- EF 50-55% normal echo, MRI r/o ARVC, negative stress test
- 24 hour holter : 27% PVC’s (27,000/24hr)
- ECG: PVC morphology LBBB, inferior axis. Otherwise normal.
- Trial of Beta Blockers, intolerant SE, wants ablation
LBBB, Inferior Axis

RVOT
Mapping and Ablation of RVOT VT

• Keep sedation light
• A pace to r/o preexcitation, SVT w/ aberrancy, and induce VT
• V pace to induce VT to r/o other VT/VF
• Isuprel infusion to induce VT
• Activation mapping: target earliest site: 10-60 ms before QRS onset
• Pace mapping: 12/12 lead perfect match (can be up to 2cm away)
• Unipolar: sharp QS
Mapping and Ablation of RVOT (cont)

- 4mm tip electrode, can be irrigated, avoid high temps, <50W
- 3-D electroanatomic mapping system, non-contact mapping system
- Non-reentrant mechanism, entrainment won’t work
- Terminates with adenosine (helps differentiate from ARVC VT)
- If no early focus, consider epicardial or LVOT origin
- Differentiate between RVOT and LVOT by ECG:
  - RVOT: transition V4,
  - LVOT: transition V1, V2
  - Either: transition V3
- Successful ablation >80%, failure usually due to inability to induce
Earliest Activation Site
RAO

ablation
LAO

ablation
Termination of VT During Ablation
RVOT Ablation Site
RVOT Ablation Site
Radiofrequency Catheter Ablation of Premature Ventricular Complexes From Right Ventricular Outflow Tract Improves Left Ventricular Dilation and Clinical Status in Patients Without Structural Heart Disease

Masao Takemoto, MD, Hitoshi Yoshimura, MD, Yurika Obha, MD, Yasuharu Matsumoto, MD, Umpei Yamamoto, MD, Masahiro Mohri, MD, Hideo Yamamoto, MD, Hideki Origuchi, MD

Kisakuya, Japan

OBJECTIVES The present study evaluated clinical benefits of radiofrequency catheter ablation (RFA) for premature ventricular complexes from right ventricular outflow tract (RVOT-PVC) in patients without structural heart disease.

BACKGROUND It is unknown whether PVC causes left ventricular (LV) dilation, which is a well-recognized precursor of LV dysfunction and heart failure, and whether eliminating PVC by RFA produces clinical benefits in patients with RVOT-PVC.

METHODS Frequency of PVC per total heart beats by 24-h Holter monitoring, left ventricular ejection fraction (LVEF), left ventricular end-diastolic internal dimension (LVIDd), normal segmental thickening (NSRT) by echocardiogram, cardiothoracic ratio (CTR) by chest radiogram, and New York Heart Association (NYHA) functional class of 40 patients with RVOT-PVC without

CONCLUSIONS These findings suggest that frequent (>20%) RVOT-PVC may be a possible cause of LV dysfunction and/or heart failure, and RFA produces clinical benefits in these patients. (J Am Coll Cardiol 2005;45:1259–65) © 2005 by the American College of Cardiology Foundation

CONCLUSIONS These findings suggest that frequent (>20%) RVOT-PVC may be a possible cause of LV dysfunction and/or heart failure, and RFA produces clinical benefits in these patients. (J Am Coll Cardiol 2005;45:1259–65) © 2005 by the American College of Cardiology Foundation

Isolated premature ventricular complexes (PVC) are the most common arrhythmias observed in patients without structural heart disease (1). It has been recently reported that frequent PVC caused left ventricular (LV) dysfunction that can be reversed by suppression of PVC with antiarrhythmic agents (2,3) or radiofrequency catheter ablation (RFA) (4,5) in patients with dilated cardiomyopathy. It is uncertain, however, whether frequent PVC causes LV dilation and dysfunction even in patients with no evidence of structural heart disease and, if so, whether suppression of PVC improves these changes. In recent years, RFA has proven to be a safe and successful therapy for arrhythmias (6,7). The purpose of this study was two-fold: 1) to examine whether frequent premature ventricular complexes from right ventricular outflow tract (RVOT-PVC) without structural heart disease may cause LV dilation, which is a well-recognized precursor of LV dysfunction and congestive heart failure (8); 2) to evaluate the role of ablating RVOT-PVC per RFA on cardiac function in patients with depressed cardiac function.

METHODS

Study population and laboratory analysis. From 1994 to 2004, 45 consecutive patients (20 males and 25 females with mean age of 50 ± 2 years and body surface area of 1.57 ± 0.02 m2) with monomorphic RVOT-PVC and no evidence of underlying structural heart disease underwent RFA at our hospital. All patients had their history recorded, and underwent physical examination, laboratory analysis, chest radiogram, 12-lead electrocardiogram, 24-h Holter monitoring, M-mode, two-dimensional, and Doppler echocardiogram (SONOS 2000, Hewlett-Packard, San Diego, California, and SEQUOIA 512, Siemens, Erlangen, Germany) on admission or within at least 1 month before admission, and

Downloaded from content.onlinejacc.org by April 6, 2012
24 Hour Holter PVC’s

"lower group" <10% extrasystoles
"middle group" 10% to 20% extrasystoles
"upper group" > 20% extrasystoles

>20% (20,000 beats/day) associated with:
Enlarged LV dimension
Reduced LV EF
Increased MR
NYHA Functional Class decrease
Dramatic improvement to baseline after ablation

LVOT VT

- Aortic cusps
- Aortomitral continuity (AMC)
- Peri-Mitral region
- Epicardial
Aortic Sinus Cusps

ECG morphology:

- Broad R-wave in V1, V2
- Transition ≤ V2
- LCC = W or M in V1
- RCC = QS or QR in V1
Ablator in Left Aortic Cusp
Left Aortic Cusp Early Potential
Aortic-mitral Continuity
AMC VT

12/12 pace map  Earliest site
Ablation at AMC Site
Complications of Outflow Tract VT Ablation

- RVOT: perforation and tamponade, heart block
- LVOT: perforation and tamponade, heart block,
  - MI if ablate near coronary artery (aortic cusp VT), injury to aortic cusp
Left Fascicular VT

- Posterior fascicle
- Anterior fascicle
- Upper septal
Presentation and Symptoms

- Exercise related VT
- Verapamil sensitive
- Ages 15-40 years
- 60-80% males
- Usually paroxysmal, but can be incessant and lead to tachycardiomyopathy
- No structural abnormality
- RBBB, LAD in 90% (LPF VT), RBBB, RAD (LAF VT) 10%, upper septal <1%
- 10-25% can also have inducible SVT
- Palpitations, lightheadedness, may be asymptomatic
Mechanism of LV VT

• macroreentry
LPF VT Circuit

Ramprakash et al. Indian Pacing and Electrophysiology 2008
Successful Ablation Sites of Left Posterior Fascicular VT

Nakagawa: early Purkinje Potentials (P2) distal site

Tsuchiya: diastolic potentials (P1) proximal mid-septum

Nogami, PACE 2011
LPF and LAF VT Circuits

Antegrade limbs of LPF and LAF VT are abnormal midseptal Purkinje tissue

Nogami, PACE 2011
RBBB, LAD

Left posterior fascicular VT

RBBB, RAD

Left anterior fascicular VT

RAD, RBBB
Case Study LV VT

- 20 year old male presented to urgent care for evaluation of scabies, otherwise no complaints
- HR 108, ECG showed VT
- Entirely asymptomatic
- Echo: EF 36%, LV dilated
- Incessant VT for unknown duration
- VT morphology: RBBB, LAD
RB/ LAD: Left fascicular VT (posterior)
ECG of Case Study VT Rate 101 bpm
AV Dissociation in VT

P waves
Map Catheter in LV Against Apical Septum
Left Fascicular VT – Posterior Fascicle

ablate

LAO
His

RAO

ablate
Left Fasicular VT
Left fascicular VT ablation site
Linear Ablation of LV VT

Lin, D, et al. HR 2005
Upper Septal VT Circuit

Antegrade limbs are LAF and LPF
Retrograde limbs are abnormal midseptal purkinje tissue
Upper Septal Fascicular VT

Narrow QRS, Normal axis or RAD
Complications of LV VT Ablation

- AV Block
- LBBB
Summary

1. Outflow Tract VT: focal, DAD’s, adenosine sensitive, earliest site is target
   - RVOT VT - most common
   - LVOT VT - can be difficult to ablate, higher risk of complication

2. Left Fascicular VT: macroreentry, verapamil sensitive, ablate any P1, earliest P2, or linear ablation
   - LPF VT - most common
   - LAF VT - infrequent
   - Upper septal VT - rare