Ventricular Arrhythmias - Diagnosis & Treatment

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Topics

- Differentiating WCT
- Differentiating VT
- Acute management of specific VTs
  - Incessant VT
- VT with structural heart disease
  - CAD
    - Primary and Secondary SCD prevention
    - VT ablation
  - Dilated CM
  - HCM
  - RVCM (ARVD)
  - Myocarditis
Topics

• VT without structural heart disease
  – Long QT
  – Brugada Syndrome
  – CPVT
  – Short QT
  – Early repolarization
  – Idiopathic VF
Differentiating Wide Complex Tachycardia
Brugada algorithm

1. Absence of an RS complex in all precordial leads?
   - yes \rightarrow VT diagnosed
   - no

2. The longest R to S interval >100 ms in any precordial lead?
   - yes \rightarrow VT diagnosed
   - no

3. A-V dissociation?
   - yes \rightarrow VT diagnosed
   - no

4. Morphology criteria for VT present both in leads $V_{1,2}$ and $V_{6}$?
   - yes \rightarrow VT diagnosed
   - no \rightarrow SVT diagnosed
AV dissociation
Capture and Fusion Beats
Lead aVR Based Algorithm
New aVR algorithm

In lead aVR:

Step 1. Presence of an initial R wave?
  No  Yes
      ↓   ↓
      VT diagnosed VT diagnosed

Step 2. Presence of an initial r or q wave >40 ms?
  No  Yes
      ↓   ↓
      VT diagnosed VT diagnosed

Step 3. Presence of a notch on the descending limb of a negative onset and predominantly negative QRS?
  No  Yes
      ↓   ↓
      VT diagnosed VT diagnosed

Step 4. $v_i/v_t \leq 1$?
  No  Yes
      ↓   ↓
      SVT diagnosed VT diagnosed
New aVR Algorithm

\[ v_i = 0.4 \quad v_t = 0.2 \quad v_i/v_t > 1 \rightarrow SVT \]
# VT diagnosis

<table>
<thead>
<tr>
<th></th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brugada Criteria(^1)</td>
<td>89</td>
<td>73</td>
<td>92</td>
<td>67</td>
</tr>
<tr>
<td>New aVR Criteria(^2)</td>
<td>97</td>
<td>75</td>
<td>93</td>
<td>87</td>
</tr>
</tbody>
</table>


Vi/Vt=0.1/0.3<1 = VT

AV dissociation
Vi/Vt = 0.5/0.1 = 5 > SVT
Clinical Presentations of Patients with Ventricular Arrhythmias

- Asymptomatic individuals with or without electrocardiographic abnormalities
- Persons with symptoms potentially attributable to ventricular arrhythmias
  - Palpitations
  - Dyspnea
  - Chest pain
  - Syncope and presyncope
- Ventricular tachycardia that is hemodynamically stable
- Ventricular tachycardia that is not hemodynamically stable
- Cardiac arrest
  - Asystolic (sinus arrest, atrioventricular block)
  - Ventricular tachycardia
  - Ventricular fibrillation
  - Pulseless electrical activity
Acute Management of Specific Arrhythmia [ACC/AHA/ESC 2006]

Sustained Monomorphic VT

• Class I
  – Wide-QRS tachycardia should be presumed to be VT if the diagnosis is unclear.
  – Direct current cardioversion in patients with hemodynamic compromise.

• Class IIa
  – Intravenous procainamide is reasonable for stable patients
  – Intravenous amiodarone is reasonable in patients that are hemodynamically unstable, refractory to conversion with countershock, or recurrent despite procainamide or other agents.
  – Transvenous catheter pace termination can be useful to treat patients with VT that is refractory to cardioversion or is frequently recurrent despite antiarrhythmic medication
Acute Management of Specific Arrhythmia [ACC/AHA/ESC 2006]

Polymorphic VT

- Class I
  - Direct-current cardioversion for hemodynamic compromise
  - Intravenous **beta blockers** are useful especially if ischemia is suspected or cannot be excluded.
  - Intravenous loading with **amiodarone** is useful for patients in the **absence of abnormal repolarization** related to congenital or acquired LQTS.
  - Urgent angiography with a view to revascularization should be considered
Acute Management of Specific Arrhythmia [ACC/AHA/ESC 2006]

Torsades de Pointes

• Class I
  – Withdrawal of any offending drugs and correction of electrolyte abnormalities
  – Acute and long-term pacing is recommended for patients presenting with torsades de pointes due to heart block and symptomatic bradycardia.

• Class IIa
  – Intravenous magnesium sulfate is reasonable for patients who present with LQTS. Magnesium is not likely to be effective in patients with a normal QT interval.
  – Acute and long-term pacing is reasonable for patients who present with recurrent pause-dependent torsades de pointes.
  – Beta blockade combined with pacing is reasonable acute therapy for patients who present with torsades de pointes and sinus bradycardia.
  – Isoproterenol is reasonable as temporary treatment in acute patients who present with recurrent pause-dependent torsades de pointes who do not have congenital LQTS.
VT Storm/ Incessant VT

Class I
- **Revascularization and beta blockade** followed by intravenous antiarrythmic drugs such as procainamide or amiodarone are recommended for patients with recurrent or incessant polymorphic VT due to acute myocardial ischemia. (Level of Evidence: C)

Class IIa
- **Intravenous amiodarone or procainamide** followed by **VT ablation** can be effective in the management of patients with frequently recurring or incessant monomorphic VT. (Level of Evidence: B)

Special Considerations:
- ICDs- Interrogation, reprogramming
- Ischemia – IABP, emergency cath,
- Pause dependent- Pacing, Isuprel
- Brugada- Isuprel, Quinidine
VT and CAD

• “Primary” and “early” VT/VF
  – How early is “early”?  
  – Impact on prognosis?

• VT associated with previous MI scar
  – Treatment
  – Risk stratification
  – Prevention of SCD
VT in patients with STEMI: Is early VT benign?

Drugs for VT/VF

Antiarrhythmic Drugs

♥ Beta Blockers: Effectively suppress ventricular ectopic beats & arrhythmias; reduce incidence of SCD

♥ Amiodarone: No definite survival benefit; some studies have shown reduction in SCD in patients with LV dysfunction especially when given in conjunction with BB.

♥ Sotalol: Suppresses ventricular arrhythmias; is more pro-arrhythmic than amiodarone, no survival benefit clearly shown

♥ Conclusions: Antiarrhythmic drugs (except for BB) should not be used as primary therapy of VA and the prevention of SCD
Incidence of Sudden Cardiac Death

- General population
- High-risk subgroups
- Any prior coronary event
- EF<30% or heart failure
- Cardiac arrest survivor
- Arrhythmia risk markers, post MI

**Incidence**

**Events**

Therapies for VA

ICDs: Results from Primary and Secondary Prevention Trials

<table>
<thead>
<tr>
<th>Trial Name, Pub Year</th>
<th>Hazard ratio</th>
<th>N</th>
<th>LVEF, other features</th>
</tr>
</thead>
<tbody>
<tr>
<td>MADIT-I 1996</td>
<td>0.46</td>
<td>196</td>
<td>0.35 or less, NSVT, EP positive</td>
</tr>
<tr>
<td>AVID 1997</td>
<td>0.62</td>
<td>1016</td>
<td>Aborted cardiac arrest</td>
</tr>
<tr>
<td>CABG-Patch 1997</td>
<td>0.83</td>
<td>191</td>
<td>0.35 or less, abnormal SAECG and scheduled for CABG</td>
</tr>
<tr>
<td>CASH* 2000</td>
<td>0.82</td>
<td>659</td>
<td>Aborted cardiac arrest</td>
</tr>
<tr>
<td>CIDS 2000</td>
<td>0.69</td>
<td>1232</td>
<td>Aborted cardiac arrest or syncope</td>
</tr>
<tr>
<td>MADIT-II 2002</td>
<td>0.65</td>
<td>458</td>
<td>0.30 or less, prior MI</td>
</tr>
<tr>
<td>DEFINITE 2004</td>
<td>0.65</td>
<td>674</td>
<td>0.35 or less, NICM and PVCs or NSVT</td>
</tr>
<tr>
<td>DINAMIT 2004</td>
<td>0.77</td>
<td>1676</td>
<td>0.35 or less, MI within 6 to 40 days and impaired cardiac autonomic function</td>
</tr>
<tr>
<td>SCD-HeFT 2005</td>
<td>0.83</td>
<td>196</td>
<td>0.35 or less, LVD due to prior MI and NICM</td>
</tr>
</tbody>
</table>

ICD better
VT treatment in post MI patients

Class I

• Treat HF
• Reduce Ischemia
• ICDs
  – patients who are survivors of cardiac arrest due to ventricular fibrillation or hemodynamically unstable sustained VT after evaluation to define the cause of the event and to exclude any completely reversible causes
VT treatment in post MI patients

Class I- cont

• ICDs
  – in patients with structural heart disease and spontaneous sustained VT, whether hemodynamically stable or unstable
  – in patients with syncope of undetermined origin with clinically relevant, hemodynamically significant sustained VT or VF induced at electrophysiological study
VT treatment in post MI patients

Class I- cont

- ICDs
  - in patients with LVEF less than or equal to 35% due to prior MI who are at least 40 days post-MI and are in NYHA functional Class II or III
  - in patients with LV dysfunction due to prior MI who are at least 40 days post-MI, have an LVEF less than or equal to 30%, and are in NYHA functional Class I.
  - in patients with nonsustained VT due to prior MI, LVEF less than or equal to 40%, and inducible VF or sustained VT at electrophysiological study
Catheter ablation of VT

• VT mapping
  – Pace
  – Activation
  – Entrainment

• Substrate modification
  – Unstable VTs
  – 3-D voltage mapping
Activation Mapping
Substrate and Pace Mapping
## SMASH-VT: Clinical end points

<table>
<thead>
<tr>
<th>End point</th>
<th>Ablation group (n=64), n (%)</th>
<th>Control group (n=64), n (%)</th>
<th>Hazard ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICD events</td>
<td>8 (12)</td>
<td>21 (33)</td>
<td>0.35 (0.15–0.78)</td>
</tr>
<tr>
<td>ICD shocks</td>
<td>6 (9)</td>
<td>20 (31)</td>
<td>0.27 (0.11–0.67)</td>
</tr>
<tr>
<td>ICD storm</td>
<td>4 (6)</td>
<td>12 (19)</td>
<td>0.30 (0.09–1.00)</td>
</tr>
<tr>
<td>Death</td>
<td>6 (9)</td>
<td>11 (17)</td>
<td>0.59 (0.22–1.59)</td>
</tr>
</tbody>
</table>

SMASH-VT: Survival Free from ICD Therapy

Catheter ablation of VT is recommended

1. for symptomatic sustained monomorphic VT (SMVT), including VT terminated by an ICD, that recurs despite antiarrhythmic drug therapy or when antiarrhythmic drugs are not tolerated or not desired;

2. for control of incessant SMVT or VT storm that is not due to a transient reversible cause;

3. for patients with frequent PVCs, NSVTs, or VT that is presumed to cause ventricular dysfunction;

4. for bundle branch reentrant or interfascicular VTs;

5. for recurrent sustained polymorphic VT and VF that is refractory to antiarrhythmic therapy when there is a suspected trigger that can be targeted for ablation.

EHRA/HRS Expert Consensus on Catheter Ablation of Ventricular Arrhythmias 2009
A previously healthy 35 y.o. man was admitted after a collapse at his office. 
EMS recorded VF that was successfully defibrillated into NSR.
Due to rapid CPR, he regained full consciousness
His ECG:
A case

- A bedside Echo study: mild LV dysfunction
- The next step?
  1. Immediate cath
  2. IV Amiodarone
  3. IV Mg
  4. EP consult
- Pt. had nonsignificant CAD, LV function normalized
Differential Dx- VF in a “structurally normal” heart

• Primary electrical disease
  – Long QT
  – Brugada syndrome
  – CPVT
  – Short QT
  – Early repolarization

• Other
  – Myocarditis
  – ARVD
  – Coronary spasm

• Idiopathic VF
LQT?

A

B

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**QT scale.**

<table>
<thead>
<tr>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>Very long QT.</td>
<td>480</td>
</tr>
<tr>
<td>LQTS even if asymptomatic. Exclude II^0 causes</td>
<td></td>
</tr>
<tr>
<td>Long QT.</td>
<td>460</td>
</tr>
<tr>
<td>LQTS when supported by symptoms, family history or additional tests.*</td>
<td></td>
</tr>
<tr>
<td>Long QT possible.</td>
<td>460</td>
</tr>
<tr>
<td>Additional tests when indicated:* Repeated ECG, Holter, T-wave morphology, exercise, epinephrine-challenge, adenosine-challenge.</td>
<td></td>
</tr>
<tr>
<td>Normal QT.</td>
<td>370</td>
</tr>
<tr>
<td>Short QT.</td>
<td>340</td>
</tr>
<tr>
<td>SQTS when supported by symptoms or family history. Additional tests: Repeated ECG, Holter, T-wave morphology (?), electrophysiologic studies (?)</td>
<td></td>
</tr>
<tr>
<td>Very short QT.</td>
<td>340</td>
</tr>
<tr>
<td>SQTS even if asymptomatic. Exclude II^0 causes</td>
<td></td>
</tr>
</tbody>
</table>
Adrenaline infusion unmasks LQT and types it. Shimizu et al JACC 2001, Heart Rhythm 2004
ΔQT > 30 ms with Adrenaline Infusion Predicts LQT1

Vyas, H. et al. Circulation 2006;113:1385-1392

Sens=93%
Spec=86%
PPV=76%
NPV=96%
Brugada Syndrome
Flecainide test
J-Point Elevation and R-Wave Slurring

Rosso, R. et al. J Am Coll Cardiol 2008;52:1231-1238
Systematic Assessment of Patients With Unexplained Cardiac Arrest.
Krahn et al. Heart Rhythm 2009

- Cardiac Arrest
- Electrocardiography
  - Resting ECG and SAECG
  - Telemetry
- Imaging
  - Echocardiogram, CT
  - Cardiac MRI, Coronary Angiogram
- Provocation
  - Exercise Test
  - Adrenaline/Procainamide Challenge
- Discretionary
  - EP Study and Voltage Map
  - Ventricular Biopsy

Phenotype Directed Genetic Testing
Diagnosed 35 of 63 Cases
Sustained Ventricular Tachycardias

Polymorphic VT
- acute myocardial ischemia
- abnormalities of ion channels
  - acquired long QT syndrome
  - genetic arrhythmia syndromes
    - Long QT, Short QT, Brugada,
    - Familial catecholaminergic polymorphic VT
- Idiopathic ventricular fibrillation
- Structural disease: hypertrophy, recent infarction, cardiomyopathy

Monomorphic VT
- Scar-related reentry
  - prior infarction
  - cardiomyopathies
    - predominant LV
      - idiopathic, familial, post viral
      - inflammatory: sarcoid, Chagas
      - idiopathic aneurysms
    - predominant RV
      - arrhythmogenic RV dysplasia
      - sarcoid, idiopathic
      - surgical incisions: ventriculotomy, repaired Tetralogy of Fallot

Purkinje disease
- Bundle branch reentry
- Automaticity
Idiopathic VT

Outflow type VTs with focal origin
ECG: inferior axis

RVOT, pulmonary artery
ECG: LBBB, transition V3-V4

LVOT, Aortic sinus, epicardial
ECG: prominent r or R in V1 or V2

Focal mitral annulus VT
ECG: RBBB, prominent R or r in V2-V6

Verapamil sensitive fascicular reentry
ECG: RBBB L or R axis
TODA RABA