EP

David Stultz
Cardiology fellow, PGY 5
April 28, 2005
Case #1

- 54 yo AAM admitted with chest pain, SOB
  - Multiple admissions for same over past several years
- ESRD, HD
- Hx CABG 2 years ago; recent EF 38%
  - Recent cath showed patent grafts
- Code Blue
  - VT, defibrillated, bradycardia
- CTSP following code
Baseline EKG
EKG following code
EKG next evening...
Fusion and Capture Beats

During the course of a tachycardia characterized by widespread, abnormal QRS complexes, the presence of fusion beats and capture beats provides maximum support for the diagnosis of VT

Braunwald
MAJOR FEATURES IN THE DIFFERENTIAL DIAGNOSIS OF WIDE QRS BEATS VERSUS TACHYCARDIA

SUPPORTS SVT

- Slowing or termination by vagal tone
- Onset with premature P wave
- RP interval ≤ 100 msec
- P and QRS rate and rhythm linked to suggest that ventricular activation depends on atrial discharge, e.g., 2:1 AV block
- rSR' V₁
- Long-short cycle sequence

SUPPORTS VT

- Fusion beats
- Capture beats
- AV dissociation
- P and QRS rate and rhythm linked to suggest that atrial activation depends on ventricular discharge, e.g., 2:1 VA block
- “Compensatory” pause
- Left axis deviation; QRS duration >140 msec
VT and SVT morphology

<table>
<thead>
<tr>
<th>Normal Conduction</th>
<th>VT</th>
</tr>
</thead>
<tbody>
<tr>
<td>LBBD</td>
<td>RBBB</td>
</tr>
<tr>
<td>SVT with Aberration</td>
<td></td>
</tr>
<tr>
<td>VT</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>LBBB-Type QRS</th>
<th>RBBB-Type QRS</th>
</tr>
</thead>
<tbody>
<tr>
<td>V1</td>
<td>V6</td>
</tr>
</tbody>
</table>

- Ventricular Tachycardia
- SVT with Aberration
- SVT with Preexcitation (WPW)
- SVT with Baseline Abnormal ECG
- SVT with Hyperkalemia
- Ventricular Pacing

Braunwald
Effect of carotid sinus pressure (CSP) on ventricular tachycardia. A, Wide QRS complex tachycardia with 1:1 retrograde conduction at left; this is followed by retrograde Wenckebach brought out by carotid sinus pressure (see aVF), clarifying the diagnosis of ventricular tachycardia. B, Termination of ventricular tachycardia by carotid sinus pressure, an unusual response. Shown are leads 1, 2, 3, V1, and V6 with intracardiac recordings from right ventricle (RV). T = time line. Asterisks denote dissociated P waves; lighter asterisks show location of P waves obscured by QRS.
Etiology of symptomatic recurrant VT

- Ischemic heart disease (>50%)
- Cardiomyopathy (both congestive and hypertrophic)
- Primary electrical disease
- Mitral valve prolapse
- Valvular heart disease
- Congenital heart disease
- Miscellaneous causes
Specific Forms of VT

- Arrhythmogenic Right Ventricular Dysplasia
- Tetralogy of Fallot
- Hypertrophic cardiomyopathy
- MVP
- Idiopathic VT
  - Idiopathic VF
  - Brugada
  - RVOT VT
  - Left septal/fascicular tachycardia
Arrhythmogenic Right Ventricular Dysplasia

- LBBB, Right axis, right precordial t wave inversions
- Epsilon waves
ARVD

- Familial form mapped to chromosomes 1 and 14q23–q24 and most recently chromosome 10
- Fatty infiltration of RV inflow, RV outflow and RV apex
- Visualize by MRI, echo
- Can involve LV
- Male predominence
- Nonspecific symptoms
- Surgery, Ablation, antiarrhythmics are not successful
- ICD is treatment
Tetralogy of Fallot

• Sustained VT can occur years after repair
• Often at site of previous surgery in RVOT
• Cured by ablation or surgical resection
Dilated Cardiomyopathy

- Noninvasive and invasive studies for VT have poor positive and negative predictive value
- ICD supported by multiple trials
  - MADIT 1 and 2
  - SCDHEFT
- Bundle branch VT more common in this population, potentially ablatable
Activation of the His-Purkinje system during bundle branch reentrant tachycardia with a right bundle branch block QRS pattern. Displayed from top to bottom are surface ECG leads I, II, and V1; intracardiac recordings from the high right atrium (HRA), proximal and distal His bundle (HBp and HBd), left bundle (LB), and right ventricle (RV); and time lines (T). During induced sustained bundle branch reentry with a right bundle branch block pattern, retrograde conduction occurs by the right bundle/His bundle axis, resulting in His bundle activation preceding that of the LB. A leftward QRS axis suggests that the antegrade limb of the reentry circuit is the posterior LB fascicle. Note that the underlying rhythm is atrial fibrillation.
Hypertrophic Cardiomyopathy

- Amiodarone useful for nonsustained VT in mildly symptomatic pts
- Unfortunately, no good way to risk stratify patients
- ICD indicated for high risk pts or pts with sustained VT
Mitral Valve Prolapse

- Increased risk of arrhythmia, although overall good prognosis
- But… Sudden death can occur
Idiopathic Ventricular Fibrillation

- 1% of out of hospital arrests, mostly middle aged males
- Monomorphic VT rarely inducible at EP study
Brugada Syndrome

- RBBB and ST elevation in precordial leads without structural heart disease
- Probably 40-60% of Vfib
- Sudden death (esp nocturnal) in healthy Southeast Asians
- SCN5A sodium channel mutation
- ICD is only prevention
Brugada Syndrome
Idiopathic VT

• Right Ventricular outflow tract VT
  – LBBB and Right Axis
  – Adenosine terminates VT
  – Verapamil and Beta blockers suppress VT
  – Paroxysmal form – exercise/stress induced
  – Repetitive form – occurs at rest
  – Similar VT rarely from RV inflow, RV apex or LV
RVOT VT
Idiopathic VT

• Left septal VT
  – Arises in left posterior septum
  – Sometimes called fasicular tachycardia
  – Verapamil (IV>oral) or diltiazem suppress
  – Adenosine not effective
  – Initiates with exercise, rapid pacing, or isuprel
Left Septal VT (Posterior)
Sudden infant death syndrome

• Unknown cause
• Potential cardiac etiologies:
  – Long QT
  – Brugada
Case #2

- 29 yo M with 10 year history of frequent PVC’s
- Bigeminal pattern
- Asymptomatic
- Recent echo with EF 35%
Baseline EKG
EKG during EP
Pace mapping of PVC
Pace Mapping PVC
Post Ablation (8 burns)
Localizing PVC origin

- Lateral (vs septal)
  - QRS $\geq 140$ ms
  - Inferior notching
  - R/S $< 1$ in V3
- Anterior (vs posterior)
  - Negative or isoelectric QRS in 1
- Inferior (vs superior)
  - Positive or isoelectric QRS in aVL

- QRS transition in lateral leads (first positive R)
  - V1: LVOT
  - V2: deep LVOT or RVOT
  - V3: just under pulmonary valve
  - V4: RV inflow
## Origin of VT

<table>
<thead>
<tr>
<th>Focal VT</th>
<th>Mechanism</th>
<th>QRS</th>
<th>Ablation Site</th>
<th>Success</th>
</tr>
</thead>
<tbody>
<tr>
<td>RV outflow VT</td>
<td>A</td>
<td>LBBB, inferior axis</td>
<td>RVOT</td>
<td>&gt;90%</td>
</tr>
<tr>
<td>LV outflow VT</td>
<td>A, R</td>
<td>inferior axis, V1=R or RS, V2-V6=R</td>
<td>Near aortic valve</td>
<td>Unknown</td>
</tr>
<tr>
<td>Mitral annular VT</td>
<td>A, R</td>
<td>RBBB, inferior axis</td>
<td>Near mitral valve</td>
<td>High</td>
</tr>
<tr>
<td>LV verapamil sensitive</td>
<td>R</td>
<td>RBBB, superior left or right axis</td>
<td>Inferoseptal mid-LV</td>
<td>&gt;90%</td>
</tr>
<tr>
<td>Bundle branch re-entry</td>
<td>R</td>
<td>RBBB or LBBB</td>
<td>Left or right bundle</td>
<td>&gt;95%</td>
</tr>
<tr>
<td>Prior MI</td>
<td>R&gt;&gt; A</td>
<td>Variable</td>
<td>MI border zone</td>
<td>60-70%</td>
</tr>
<tr>
<td>RV dysplasia</td>
<td>R</td>
<td>LBBB</td>
<td>RVOT, near tricuspid annulus</td>
<td>Palliative</td>
</tr>
<tr>
<td>Chagas</td>
<td>R</td>
<td>Variable</td>
<td>Near LV scar, epicardial</td>
<td>Unknown</td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>R</td>
<td>LBBB or RBBB</td>
<td>RVOT or septum</td>
<td>High</td>
</tr>
<tr>
<td>Sarcoid</td>
<td>R</td>
<td>Variable</td>
<td>?</td>
<td>Unknown</td>
</tr>
</tbody>
</table>
Expanded CMS ICD coverage

- Coronary artery disease with a documented myocardial infarction and left ventricular ejection fraction (LVEF) \( \leq 0.35 \) with inducible VT or VF at EP study (performed at least 4 weeks after MI)
- Documented prior MI and a measured LVEF \( \leq 0.30 \). Patients must not have:
  - New York Heart Association (NYHC) classification IV
  - Cardiogenic shock or symptomatic hypotension while in a stable baseline rhythm
  - Had a coronary artery bypass graft (CABG) or percutaneous transluminal coronary angioplasty (PTCA) within past 3 months
  - Had an MI within past 40 day
  - Clinical symptoms or findings that would make them a candidate for coronary revascularization
  - Any disease, other than cardiac disease (e.g., cancer, uremia, liver failure), associated with a likelihood of survival less than 1 year.
- Patients with ischemic dilated cardiomyopathy (IDCM), documented prior MI, NYHA Class II and III heart failure, and measured LVEF \( \leq 35\% \)
- Patients with non-ischemic dilated cardiomyopathy (NIDCM) > 9 months, NYHA Class II and III heart failure, and measured LVEF \( \leq 35\% \)
- Patients who meet all current Centers for Medicare & Medicaid Services (CMS) coverage requirements for a cardiac resynchronization therapy (CRT) device and have NYHA Class IV heart failure