Unique Forms of Ventricular Tachycardia

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Absence of an RS complex in all precordials?

- **YES**
  - VT
  - R to S interval > 100 ms in 1 precordial ?
    - **YES**
      - VT
    - **NO**
  - More QRS complexes than P waves ?
    - **YES**
      - VT
    - **NO**
  - Morphology criteria for VT present in leads V1 and V6 ?
    - **YES**
      - VT
    - **NO**

**SVT with aberrant conduction**
64 male. HTN, ICM
Giddiness, papitation.
Can you see the Difference??
57 Y OLD MAN WITH HISTORY OF RECENT AVR AND PALPITATION CHST PAIN AND PRESYNCOPE
Patient was presented with this rhythm after AVR surgery
The “Re-Entry” Mechanism of Ectopic Beats & Rhythms.

Premature Beat Impulse

Repolarizing Tissue (long refractory period)

Fast Conduction Path
Slow Recovery

Cardiac Conduction Tissue

Slow Conduction Path
Fast Recovery

1. An arrhythmia is triggered by a premature beat

2. The beat cannot gain entry into the fast conducting pathway because of its long refractory period and therefore travels down the slow conducting pathway only
4. On arriving at the top of the fast pathway it finds the slow pathway has recovered and therefore the wave of excitation ‘re-enters’ the pathway and continues in a ‘circular’ movement. This creates the re-entry circuit.
The AV node crosses the central fibrous body to become the penetrating portion of bundle of His, and rests on the muscular ventricular septum, then enters the region of pars membranacea to become branching.
• Akhtar and Damato 1973
  – antecubital vein approach

  – Ventricular extra-stimulus with a critical V-H delay blocked in the right bundle and activated the His via the left bundle

  – A “V3” response conducted down via the right bundle with an H-V interval longer than that of sinus beat

  – Importantly – complete RBBB abolished the V3 response
- 5% of all sustained VT
- Non ischemic cardiomyopathy
- LBBB + 1º AVB (in NSR)
- Low LVEF
- EP: reentry → prog stim

Radiofrequency catheter ablation of ventricular tachycardia

Stevenson WG, Delacretaz E, Heart 2000
BBR VT

• Macro re-entrant (→Ventricular flutter) circuit employing...
  – Both bundle branches
  – Ramifications of the left bundle

• Hallmark: His-Purkinje system disease – functional or structural

• Ischemic (6%) vs. non-ischemic (40%) cardiomyopathy
• Close anatomical proximity of the HPS to the valvular annuli makes it vulnerable to surgical manipulations in this area.

• Correlates with historical literature on post-op peak sudden death time-course.

• In contrast to postoperative intramyocardial reentrant VT, BBR VT usually occurs in the early postoperative period (within first 2 weeks) and can be associated with preserved systolic left ventricular function.
**HPS – Site of Re-entrant Arrhythmias**

- **Bundle Branch Re-entry**
  - Macro re-entrant circuit between the left and right bundles
  - LBBB TYPE
  - RBBB TYPE
  - Inter-fascicular VT
Types of BBR Tachycardia

Table 1  Types of BBR-VT

<table>
<thead>
<tr>
<th>Type</th>
<th>Retrograde conduction</th>
<th>Anterograde conduction</th>
<th>VT morphology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type A</td>
<td>Left bundle</td>
<td>Right bundle</td>
<td>Left bundle branch block morphology</td>
</tr>
<tr>
<td>Type B</td>
<td>Fascicle of left bundle (Anterior or posterior)</td>
<td>Contra-lateral fascicle</td>
<td>Right bundle branch block morphology</td>
</tr>
<tr>
<td>Type C</td>
<td>Right bundle</td>
<td>Left bundle</td>
<td>Right bundle branch block morphology</td>
</tr>
</tbody>
</table>

Schematic diagram showing the circuits in 3 types of bundle branch reentry. LAF last anterior fascicle; LB left bundle-branch; LPF left posterior fascicle; RB right bundle-branch.
Clinical characteristics of patients

- BBR VT is usually seen in patients with an acquired SHD

- An important underlying cardiac pathology accounting for the majority of cases

- The relative contribution of BBR mechanism to inducible sustained monomorphic VT is significantly higher in non-ischemic (up to 40%) than in ischemic etiology (up to 6%)

- A resting ECG of these patients often reveal conduction abnormalities in the form of a prolonged PR interval, nonspecific intraventricular conduction delay, incomplete or complete BBB.

Circulation 1997;96:4307-4313.
Preserved Systolic Left Ventricular Function

• BBR is an important mechanism of arrhythmia in patients with myotonic myocardial dystrophy.

• The disease is characterized by relatively selective and significant conduction system impairment.

• Conduction abnormalities due to sodium channel blockade with flecainide have been implicated in the development of BBR.

• The arrhythmia has also been reported in patients with idiopathic isolated conduction system disease and no apparent structural heart abnormalities.
Immediate hemodynamic collapse requiring CPR and direct cardioversion occurred in 13 patients (26%).

Syncope was present in 25 patients (51%)

Sustained palpitations was the only presenting symptom in 5 patients.

EP studies were performed to rule out proarrhythmia during antiarrhythmic therapy for AF in one patient, or for evaluation of rapid nonsustained VT in 4 patients.
Bundle Branch Re-entry VT DD

- Intra-myocardial re-entry VT

- *Inter*fascicular VT (form of BBRVT) – *RBBB* and *LPFB*

- Automatic fascicular tachycardia

- 1:1 Supraventricular tachycardia with aberrancy including (AV nodal reentrant tachycardia, junctional tachycardia, intrahisian reentry, atrial tachycardia with aberrant ventricular Conduction)

- Atrio-fascicular re-entry (*Mahaim*)
BBR VT – Pitt Falls

• Exclusion of SVT with aberrancy

• Need to prove A-V dissociation

• Need to prove active HPS participation in the VT mechanism – rather than passive participation
Management and prognosis

- Patients With preserved LV function who have no other risk markers for sudden death is less clear. Very limited data suggest that these patients may have a favorable long-term prognosis after successful ablation of bundle branch reentry.

- Patients with myotonic dystrophy may need prophylactic permanent pacemaker implantation because of the progressive nature of the conduction system disease. The recurrence of BBR VT after successful ablation would be extremely unlikely.

- Complete RBBB pattern on the ECG appears to be a good marker of long-term success after ablation of the RB.
• These 16 patients were followed for a mean duration of 22 ± 10 months (range 2-32 months)

• The success rate of radiofrequency ablation of the right bundle was 100%.

• In spite of this success rate and low incidence of short-term complications, the long-term outcome of these patients has been of major concern.

• The majority of these patients have poor left ventricular function and New York Heart Association Class III-IV symptomatology.

• Therefore, the long-term efficacy of the right bundle ablation in improving event-free survival in this patient population has been questioned
Surface 12 lead ECG during fascicular VT showing a right bundle branch block QRS morphology with left axis deviation, QRS duration is of 120 ms.
Idiopathic left ventricular tachycardia

• MC-ILVT is verapamil-sensitive tachycardia
• 1st - Zipes and colleagues in 1979
• Belhassen – demon verapamil sensitivity of the tachycardia
  (Response of recurrent sustained ventricular tachycardia to verapami, Br Heart J; 1981)

triad:
(1) induction with atrial pacing
(2) RBBB morphology + left axis deviation
(3) no structural heart disease
• Anatomic basis for ILVT is unclear

• Earliest site of activation - inferoposterior LV septum

• Originates from a false tendon - extending from the posteroinferior LV to the basal septum

• Localized reentry as the pred mech in verapamil-sensitive ILVT.
Fascicular VT - Circuit
Fascicular VT – Anatomy and Physiology

- Relatively narrow WCT
- 90% originate from left posterior fascicle
- Anatomic substrate: LV “false tendon” or postero-inferior fibromuscular band to basal septum
- Diagnostically – may require isoprenaline to facilitate induction

Purkinje Tissue running in false tendon
• 15-40 years old.

• Typical symptoms

  Palpitations, fatigue, and presyncope.

  Syncope and SCD are rare but described.

  Incessant-tachycardia-induced cardiomyopathy.

  Most episodes occur at rest (making exercise testing unreliable in assessment).
• baseline 12-lead ECG is normal in most patients.

• ILVT-RBBB, left sup axis + rel narrow QRS duration(≤140 ms).

• exit site near the area of the left posterior fascicle
## Three Subtypes

<table>
<thead>
<tr>
<th>QRS Morphology</th>
<th>Anatomic Origin</th>
<th>QRS Axis</th>
</tr>
</thead>
<tbody>
<tr>
<td>LAHB</td>
<td>RBBB</td>
<td>Rightward</td>
</tr>
<tr>
<td>LPHB</td>
<td>RBBB</td>
<td>Leftward</td>
</tr>
<tr>
<td>Upper Left</td>
<td></td>
<td>Normal</td>
</tr>
<tr>
<td>Septal</td>
<td></td>
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</tbody>
</table>
A 12 lead ECG showing broad complex tachycardia with right bundle branch block morphology and left axis deviation.

Rate 146  Tachycardia, ? origin, rate 146  Absent P waves, rate >= 130
PR 0  Bifascicular block: RBBB & LAFB  RBBB with left axis deviation
QSD -122  Probable LVH with ST-T abnormalities  LVH voltage, ST-T negative
QT 322  Anterolateral ST-T abnormalities  ST-T negative in I, aVL, V2-V6
QTc 502  Also c/w ischemia &/or subendo. injury  ST > -.30 mV, T > -.10 mV
• Long term prognosis good

• A/c-iv verapamil, c/c-oral verapamil

• RFA- severe symptoms, resistant or intolerant to med

• Long-term success after catheter ablation is >than 90%
Outflow Tract Tachycardias

- outflow tract region
  - RV region between the pulm and tricuspid valves,
  - Basal left ventricle- outflow tract under the AoV, the aortic cusps, and the basal LV epicardium.

- A) repetitive monomorphic VT
- B) paroxysmal sustained VT
RVOT VT

- Normal structural heart
- LBBB + INF axis
- Repetitive bursts → NSVT
- Triggered automaticity
- EP: Provoked by adrenergic stim
- Ablation 85% success

Stevenson WG, Delacretaz E, Heart 2000
- RVOT VTs -75%
  - LBBB+ INF AXIS
  - QRS transition from $V_3 \backslash V_4$ (late)

- Jadonath RL et al, localizing the origin of right ventricular outflow tract tachycardia. Am Heart J 1995
  RVOT - 9 regions and used QRS morphology in leads I and aVL + R wave transition to diff ant from post RVOT sites

- Anterior sites- Q wave (Q or qR) in lead I and a QS in lead aVL.
- Posterior site- R wave in lead I and an early precordial transition( R to S) in V3
- Septal- taller, narrower monophasic R-inferior leads
- Free wall RVOT VTs-notchi inf leads and later transition (>V3)
- RVOT VT-Posterior- +ve in V1, ant- -ve in V1
RVOT VT
• Outflow tract VT - good prognosis- benign course
• Polymorphic VT- unusually short–coupled RVOT VPCs- respond well to successful VPC ablation

• **Management of outflow tract VTs**
  medical therapy or catheter ablation.

• Adenosine, verapamil, b-blockers, and carotid sinus massage - acutely
• b-blockers and CCBs-c/c supp therapy (efficacy 67% typical RVOT VT)
• Pats- breakthrough tachy on b-blocker or CCB, class I or class III antiarrhythmic therapy
• Catheter ablation - high success rate (>80%) in treating these arrhythmias

• Long-term cure rates after a successful initial ablation are high

• overall recurrence rate is approximately 10%
CASE

- 35 year old gentleman
- 1 episode of syncope while waiting in line at the bank
- Echo normal LV function
- EP study negative for inducible VT
- EKG as shown
UNUSUAL VT

ARRHYTHMOGENIC RIGHT VENTRICULAR DYSPLASIA VT

- Fatty/fibrous tissue in RV and/or LV
- LBBB in V1 with frontal axis directed inferiorly
- Success depends on LV extension
Surface ECG of repetitive monomorphic right ventricular outflow tract tachycardia. Repetitive bursts of ventricular tachycardia are present, with occasional sinus complexes.
Surface ECG of sustained right ventricular outflow tract tachycardia.
Thank you!