Fast & Slow
Tachy & Brady
Arrhythmias

DAVID STULTZ, MD, FACC
KPN HEART & VASCULAR
AUGUST 22, 2018
<table>
<thead>
<tr>
<th>Wave/Interval</th>
<th>Duration (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>P wave duration</td>
<td>&lt;120</td>
</tr>
<tr>
<td>PR interval</td>
<td>&lt;120</td>
</tr>
<tr>
<td>QRS duration</td>
<td>&lt;110-120*</td>
</tr>
<tr>
<td>QT interval (corrected)</td>
<td>≥440-460*</td>
</tr>
</tbody>
</table>

*See text for further discussion.
Normal(ish) EKG
EKG boxes

- Heart Rate
  - 1 big box = 200ms
  - 1 small box = 40ms

<table>
<thead>
<tr>
<th>Big Boxes Between QRS complexes</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart Rate (300/big boxes)</td>
<td>300</td>
<td>150</td>
<td>100</td>
<td>75</td>
<td>60</td>
<td>50</td>
<td>42</td>
</tr>
</tbody>
</table>
Case presentation

- 47 year old female presents to the emergency room with palpitations for 1 hour
- Mild lightheadedness, no syncope
- No significant past medical history
- No significant medications
After intervention…
General Mechanism of Nodal Dependent SVT

- Two Conduction Paths
  - Different conduction velocities
  - Different Refractory periods
- Faster conduction = longer refractory period
- AVNRT – two paths are within the AV node
- AVRT – one path is nodal, one is accessory
AVNRT

DUAL AV Nodal Pathways: Substrate for AVNRT

- Sinus
- Blocked PAC (P')
- Conducted PAC (P')
- AVNRT

P' conducts down slow pathway, reenters up fast, starting AVNRT

- Denotes point of block in AV node
- P: Sinus P wave
- P': PAC
- (P): Retrograde P wave due to reentry hidden in QRS

Goldberger
AV Node Reentry Tachycardia (AVNRT)

- 60% of all SVT's (most common)
- 70% are female
- Mostly patients age 30-40's
- 90% Typical (Slow-Fast)
  - Antegrade limb has slow conduction, retrograde is fast
- 10% Atypical
  - Fast-Slow
  - Slow-Slow
  - Fast-Fast
Typical AVNRT

- Starts with PAC
  - Fast path is refractory, so PAC is blocked
  - Slow path (short refractory period) is able to conduct
- PAC impulse conducted to ventricles by slow path
- PAC impulse simultaneously conducted up fast path (no longer refractory) in a retrograde fashion
- Atrial depolarization occurs simultaneous with Ventricular depolarization
EKG Features of AVNRT

- P waves either hidden in QRS or appear as part of QRS
  - Pseudo R in V1
  - Pseudo S in II, III, avF
  - P waves negative in inferior leads
AVNRT with pseudo S and pseudo R’ waves
My example may not have been the best for this phenomena...
Breaking a tachycardia

- Vagal Maneuvers (Valsalva, Carotid Massage)
- AV blocking drugs (Adenosine, Verapamil)
- AV node dependent tachycardias will break
  - If SVT terminates with a P wave then it is AVNRT or AVRT
  - If it terminates with a QRS, this is not discriminatory
- If it doesn’t break with above maneuvers it is most likely atrial tachycardia
Acute Management of SVT

- Vagal Maneuvers
  - Carotid Massage
  - Valsalva
  - Cold water immersion
  - Gag reflex
- Adenosine 6mg IV/12mg IV
- Verapamil 5-10mg IV / Diltiazem 10-20mg IV
  - Use digoxin 0.25-0.5mg IV instead if CHF is known
- Procainamide 1g IV / Amiodarone 150-300mg IV
- Synchronized cardioversion (start at 50J)
SVT Breaking with adenosine
Longterm Management of AVNRT

- No therapy if limited symptoms or infrequent episodes
- Lifestyle modification – avoid caffeine/stimulants
- Vagal maneuvers prn
- AV node dependent tachycardias (AVNRT)
  - Verapamil, Beta Blockers
  - Antiarrhythmics rarely used
- Ablation therapy
Another case...

- 25 year old male with palpitations
- 1 episode of syncope in teens
- No other significant past medical history
- No medications
Wide complex tachycardia
After Intervention
AV Reentrant Tachycardia
AVRT

- Second most common SVT
- Uses accessory path of Myocardial tissue connecting atrium and ventricle
  - >50% left free wall
  - 20-30% posteroseptal
  - 10-20% right free wall
  - 5-10% anteroseptal
- Paths most commonly conduct bidirectionally but may be solely antegrade or retrograde
- Accessory paths are usually fast conduction
Accessory Pathways

- Antegrade conduction path
  - In normal conduction, ventricles activated 1\textsuperscript{st} by accessory path and 2\textsuperscript{nd} by normal AV-His conduction
    - Preexcited ventricle, short P-R interval, delta wave
    - Variable degree of preexcitation amongst individuals
    - Preexcitation can be modulated by antiarrhythmics, autonomic tone

- Retrograde conduction path (25%)
  - Concealed pathways
  - Not apparent on normal EKG

- Large electrical loop
- Slower rates than AVNRT

Chauhan, Goldberg
Types of AVRT

- SVT initiated by PAC or PVC
- Orthodromic AVRT
  - Uses AV node as antegrade limb, accessory path conducts retrograde
  - Common
  - EKG shows no delta wave
    - (Typically Narrow Complex)
- Antidromic AVRT
  - Accessory path is antegrade, AV node retrograde
  - Uncommon
  - EKG shows preexcitation (Wide Complex)
  - May involve multiple bypass tracts (rare)
Antidromic AVRT

Antegrade conduction from left paraseptal bypass tract, retrograde conduction through AV node
Acute management of WPW

- If narrow complex, regular tachycardia, management identical to AVNRT
- If wide complex and regular
  - Consider VT
  - Avoid calcium channel blockers (verapamil)
  - Vagal maneuvers, adenosine, beta blockers, cardioversion
17 yo male with palpitations and lightheadedness after playing soccer
Acute management of WPW

- If narrow complex, regular tachycardia, management identical to AVNRT
- If wide complex and regular
  - Consider VT
  - Avoid calcium channel blockers (verapamil)
  - Vagal maneuvers, adenosine, beta blockers, cardioversion
- If wide complex and irregular (Atrial fibrillation with WPW)
  - Procainamide
  - Cardioversion
  - Avoid all negative chronotropes!!
Board Question

21 year old male with EKG showing a Delta wave.

- What do you do if asymptomatic?
- What if he is symptomatic?
Therapy for WPW

- Catheter ablation of the accessory pathway for symptomatic patients
- Asymptomatic patients with delta wave
  - No palpitations, syncope, family history of sudden death
  - No specific therapy unless symptoms develop
  - Exception may be for airline pilots, police officers, and firefighters, high level competitive athletes; may prefer catheter ablation
Atrial flutter
Ventricular tachycardia

- Wide complex, regular tachycardia
- May be “stable” or unstable
- Differential for wide complex tachycardias
  - For any regular, wide complex tachycardia, assume VT until proven otherwise!
  - Look for old Bundle Branch Block
  - Consider “SVT with aberrency”
  - WPW?
Etiology of symptomatic recurrent VT

- Ischemic heart disease (>50%)
- Cardiomyopathy (both congestive and hypertrophic)
- Primary electrical disease
  - hypo/hyperkalemia
  - hypomagnesemia
- Mitral valve prolapse
- Valvular heart disease
- Congenital heart disease
- Miscellaneous causes
Case VT

- 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...
Rhythm Strip
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.
Acute management of VT

- **Pulseless**
  - ACLS protocol
    - 360J unsynchronized shock
    - Amiodarone
    - Epinephrine
- **Hypotensive/unstable (but with pulse)**
  - 50J synchronized shock
- **Stable (No VT is really stable)**
  - Amiodarone or lidocaine or other antiarrhythmic
  - 50J synchronized shock
52 year old female with 1.5 hours of chest pressure, palpitations, shortness of breath, lightheadedness, +/- diaphoresis
No significant past medical history, no significant medications, nonsmoker, no DM or HTN
No family history of atherosclerosis or sudden cardiac death
drinks about 4 glasses of wine daily, under a lot of stress recently; denies illicits/OTC's
Typically walks several miles, no dyspnea, no chest pain; never had syncope or symptoms like this
Initial cardiac enzymes negative, no other labs available
Cardiac Catheterization Normal
Echocardiogram Normal
Diagnosed with idiopathic Left Ventricular Outflow Tract VT
(Right bundle branch block + Left Axis Deviation)

Managed on Metropolol 25mg po bid
Had Treadmill stress test 2 weeks later without arrhythmia
Torsades de Pointes

- Twisting of Points
- Management similar to monomorphic VT
- More often associated with Long Q-T syndrome
  - Medication induced or congenital
  - Think Tikosyn (dofetilide)
- Remember hypokalemia/hypomagnesemia as causes!
Initiation of polymorphic VT
Long-short-long cycle of QRS with R on T
Another Torsades...
Acute treatment of Torsades

- Acquired Long QT (ie medication induced)
  - IV Magnesium
  - Temporary pacing (high rate)
  - Isoproterenol (to increase heart rate)
  - IV Lidocaine
  - Mexiletine
  - Phenytoin
- Congenital Long QT
  - Beta Blocker
  - Pacemaker/ICD
You are called from 5S...

Pseudo-Ventricular Tachycardia (artifact)
And now to Slow it down....
1\textsuperscript{st} Degree AV Block

- >200 ms from onset of P wave to onset of QRS
2\textsuperscript{nd} Degree AV Block
Type 1 - Wenkebach

- P-R interval prolongs until QRS is dropped
2\textsuperscript{nd} Degree AV Block
Type 1 - Wenkebach
2\textsuperscript{nd} Degree Heart Block
Type 2

- PR interval remains constant, QRS drops unexpectedly
2nd Degree Heart Block
Type 2
3rd degree Heart Block

- P rate faster than QRS rate
- No correlation between P's and QRS
Bundle Branch Blocks

- Right Bundle Branch Block
- QRS duration >120ms (3 small boxes)
- rsR’ in V1
- ‘Rabbit Ears’
Bundle Branch Blocks

- Left Bundle Branch Block
- QRS duration >120ms (3 small boxes)
- R in V6
<table>
<thead>
<tr>
<th></th>
<th>$V_1$</th>
<th>$V_6$</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Normal</strong></td>
<td><img src="image1.png" alt="Normal V1" /></td>
<td><img src="image2.png" alt="Normal V6" /></td>
</tr>
<tr>
<td><strong>RBBB</strong></td>
<td><img src="image3.png" alt="RBBB V1" /></td>
<td><img src="image4.png" alt="RBBB V6" /></td>
</tr>
<tr>
<td></td>
<td>R’</td>
<td>R</td>
</tr>
<tr>
<td></td>
<td>S</td>
<td>q</td>
</tr>
<tr>
<td><strong>LBBB</strong></td>
<td><img src="image5.png" alt="LBBB V1" /></td>
<td><img src="image6.png" alt="LBBB V6" /></td>
</tr>
<tr>
<td></td>
<td>T↓</td>
<td>T↓</td>
</tr>
</tbody>
</table>
**Bundle Branch Block Criteria**

**TABLE 9-7 Common Diagnostic Criteria for Bundle Branch Blocks**

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Graphs</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Complete left bundle branch block</strong></td>
<td><img src="image" alt="Graphs" /></td>
</tr>
<tr>
<td>QRS duration ≥120 msec</td>
<td><img src="image" alt="Graphs" /></td>
</tr>
<tr>
<td>Broad, notched R waves in lateral precordial leads (V₅ and usually leads I and aV₁)</td>
<td><img src="image" alt="Graphs" /></td>
</tr>
<tr>
<td>Small or absent initial r waves in right precordial leads (V₂) followed by deep S waves</td>
<td><img src="image" alt="Graphs" /></td>
</tr>
<tr>
<td>Absent septal q waves in left-sided leads</td>
<td><img src="image" alt="Graphs" /></td>
</tr>
<tr>
<td>Prolonged intrinsicoid deflection (&gt;60 msec) in V₅ and V₆</td>
<td><img src="image" alt="Graphs" /></td>
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**Complete right bundle branch block**

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<tr>
<td>QRS duration ≥120 msec</td>
<td><img src="image" alt="Graphs" /></td>
</tr>
<tr>
<td>Broad, notched R waves (rsr’, rsR’, or rSR’ patterns) in anterior precordial leads (V₁ and V₂)</td>
<td><img src="image" alt="Graphs" /></td>
</tr>
<tr>
<td>Wide and deep S waves in left precordial leads (V₃ and V₄)</td>
<td><img src="image" alt="Graphs" /></td>
</tr>
</tbody>
</table>

*Criterion required by some authors.
Axis
Left Anterior Fascicular Block

- Frontal Axis -45 to -90 degrees
- QRS <120ms
- rS pattern in II, II, aVF (inferior leads)
LAFB + RBBB
Left Posterior Fascicular Block

- Frontal Axis +/-120 degrees (typically right axis deviation)
- QRS <120ms
- RS pattern I
- qR pattern in II, II, aVF (inferior leads)
Fascicular Blocks

QRS Duration <120ms

LAHB (LAFB)
Severe LAD without explanation
• Deep S waves in II, III, aVF
• Frontal Axis < -45 to -60 degrees
• Positive in I, Negative in aVF
• Not explained by LBBB, LVH, inferior infarct

LPHB (LPFB)
Opposite of LAFB, Rare
• Usually Right Axis deviation
• Negative in I, Positive in aVF
• Positive in II, III, aVF
• Not explained by RVH, anterolateral infarct

Schedit, S. Basic Electrocardiography. CIBA-GEIGY Pharmaceuticals, USA, p 49.
**Fascicular Block Criteria**

<table>
<thead>
<tr>
<th>TABLE 9–6</th>
<th>Common Diagnostic Criteria for Unifascicular Blocks</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Left anterior fascicular block</strong></td>
<td></td>
</tr>
<tr>
<td>Frontal plane mean QRS axis of −45 to −90 degrees with rS patterns in leads II, III, and aV_{1} and a qR pattern in lead aV_{1}</td>
<td></td>
</tr>
<tr>
<td>QRS duration less than 120 msec</td>
<td></td>
</tr>
<tr>
<td><strong>Left posterior fascicular block</strong></td>
<td></td>
</tr>
<tr>
<td>Frontal plane mean QRS axis of ±120 degrees</td>
<td></td>
</tr>
<tr>
<td>RS pattern in leads I and aV_{1} with qR patterns in inferior leads</td>
<td></td>
</tr>
<tr>
<td>QRS duration of less than 120 msec</td>
<td></td>
</tr>
<tr>
<td>Exclusion of other factors causing right axis deviation (e.g., right ventricular overload patterns, lateral infarction)</td>
<td></td>
</tr>
</tbody>
</table>

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| COMMON CAUSES OF ATRIOVENTRICULAR AND INTRAVENTRICULAR CONDUCTION DISTURBANCE |
|---------------------------------|------------------|
| **Intrinsic causes**            | **Extrinsic causes**              |
| Congenital                      | Drugs                           |
| Sclerodegenerative              | Autonomic disorders             |
| Ischemia                        | Hypothyroidism                  |
| Trauma (surgical)               |                                |
| Connective tissue disorders     |                                |
| Tumors                          |                                |
| Sarcoidosis                     |                                |
Case Presentation

- 50ish year old white female
- No cardiac history
- Admitted 2 weeks ago at outside hospital for syncope
- Watched for 2 days, diagnosed with possible seizures, had “negative” echo
- Recurrent syncope, admitted to KMC
ECG Network (635) Kettering/sycamore Hosp (93560)

Device: Speed: 25 mm/sec Limb: 10 mm/mV Chest: 10 mm/mV

Previous ECG: 21-Apr-2007 21:29:56 - Abnormal Confirmed

Confirmed By: Salem Ahmad, M.D. 22-Apr-2007 14:56:11

Requested By: XERG

Standard 12
Later that night....
Board Pearls for Heart Block

- Think of potential causes of heart block
  - Lyme disease
  - Sarcoidosis
  - Drug overdose
  - Hyperkalemia
  - Hypothyroidism
Sometimes heart blocks don’t easily fit into a defined category...
2:1 AV Block
Another case...

- 75 year old male admitted with syncope
- No significant past medical history or medications
- Nothing on telemetry overnight...
NSR → 20 second asystole
Atrial fibrillation → Asystole
References

- Goldberger AL, Goldberger ZD, Shvilkin A. Goldberger's Clinical Electrocardiography, Chapter 14, 130-143