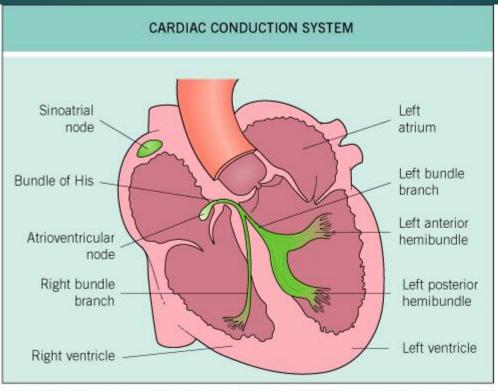
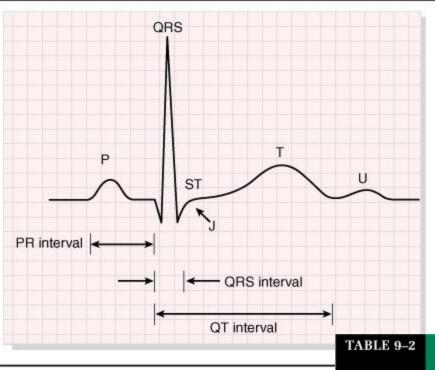
Fast & Slow Tachy & Brady Arrhythmias



DAVID STULTZ, MD, FACC KPN HEART & VASCULAR AUGUST 22, 2018



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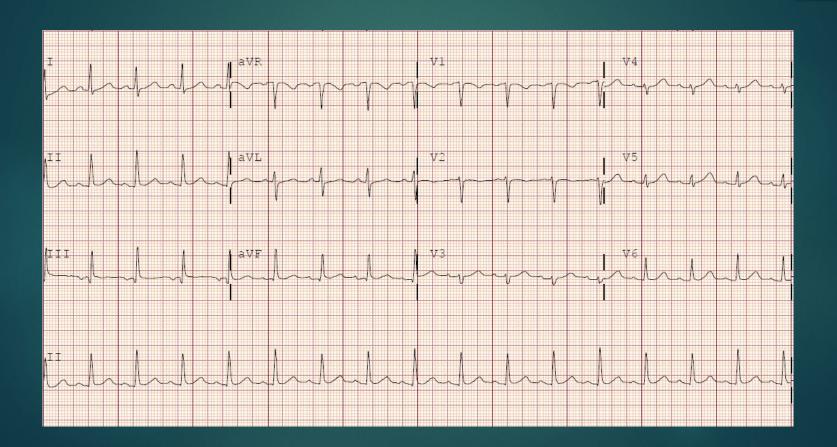
#### 2 Normal Values for Durations of Electrocardiographic Waves and Intervals in Adults

in Adults				
Wave/Interval	Duration (msec)			
P wave duration	<120			
PR interval	<120			
QRS duration	<110-120*			
QT interval (corrected)	≥440-460*			

<sup>\*</sup>See text for further discussion.

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## Normal(ish) EKG



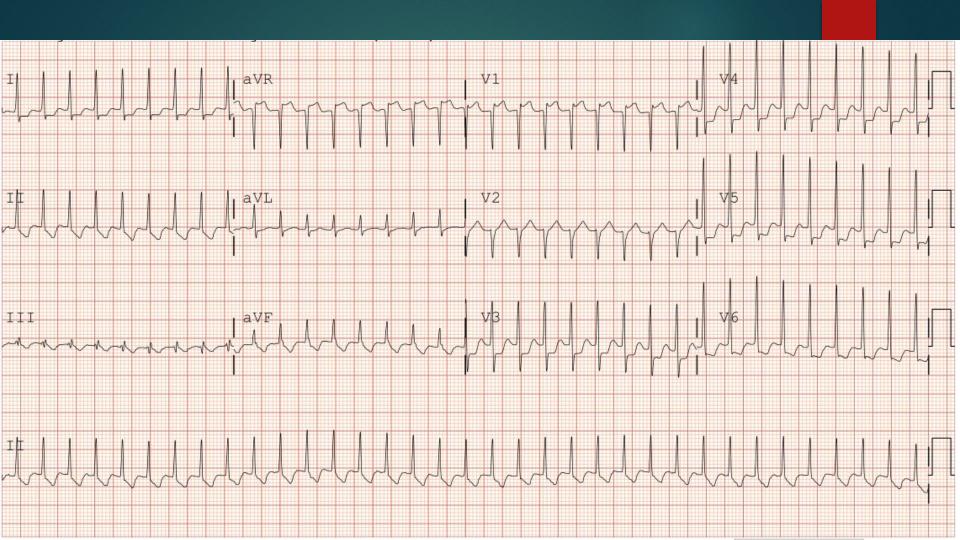
## EKG boxes

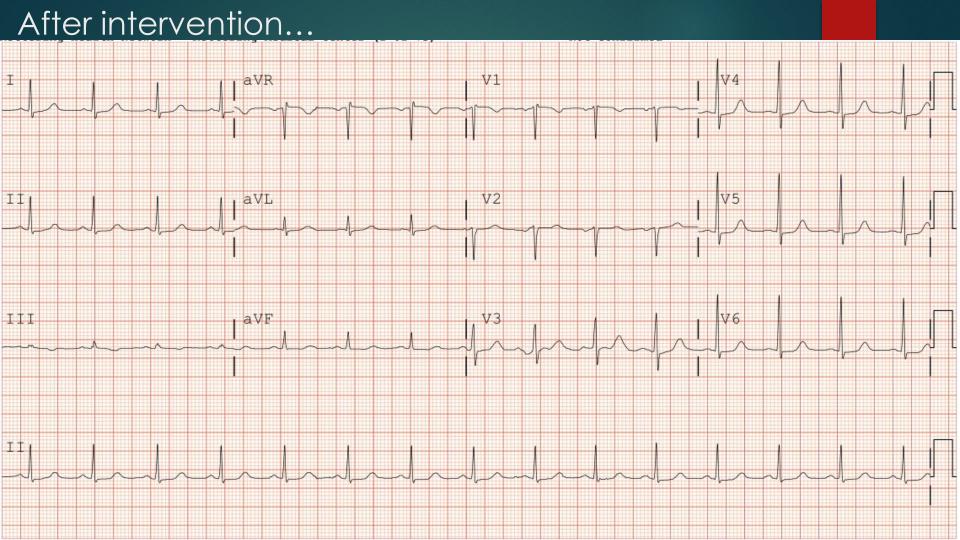
- ▶ Heart Rate
  - ▶ 1 big box = 200ms
  - $\triangleright$  1 small box = 40ms

Big Boxes  Between QRS complexes	1	2	3	4	5	6	7
Heart Rate (300/big boxes)	300	150	100	75	60	50	42

## 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



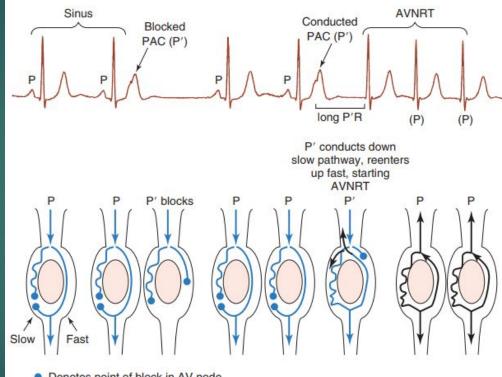


# General Mechanism of Nodal Dependent SVT

- 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**



Denotes point of block in AV node

P: Sinus P wave

P': PAC

(P): Retrograde P wave due to reentry hidden in QRS

## AV Node Reentrent Tachycardia AVNRT

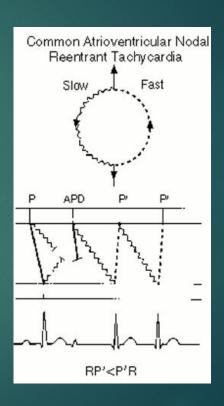
- 60% of all SVT's (most common)
- ▶ 70% are female
- Mostly patients age 30-40's
- ▶ 90% Typical (Slow-Fast)
  - Antegrade limb has slow conduction, retrograde is fast
- 10% Atypical
  - Fast-Slow
  - Slow-Slow
  - Fast-Fast

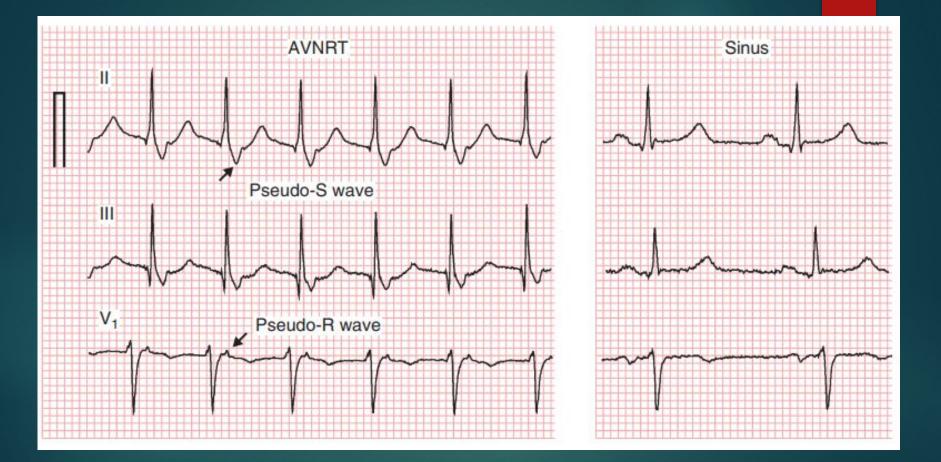
## Typical AVNRT

- 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

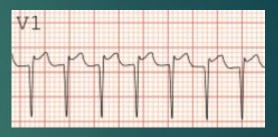




# My example may not have been the best for this phenomena...









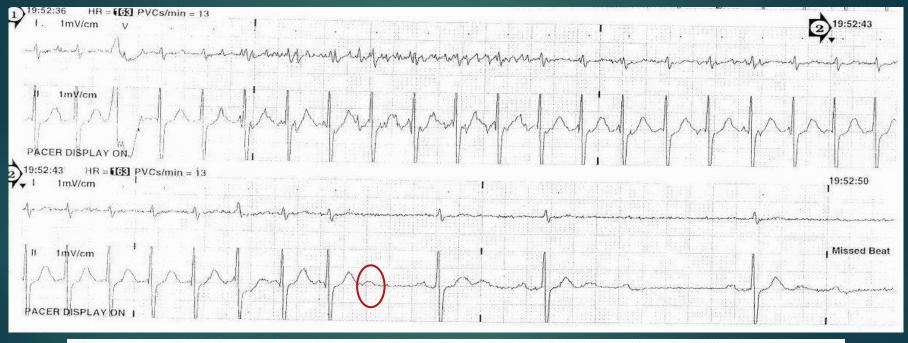
## Breaking a tachycardia

- 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



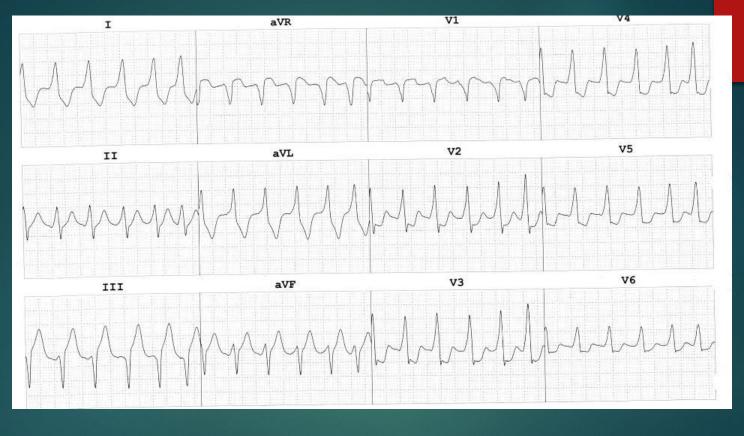


## Longterm Management of AVNRT

- 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

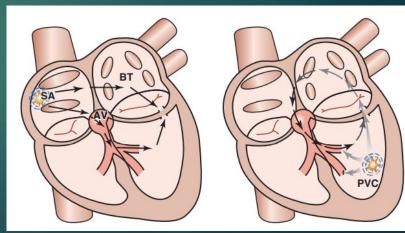


## AV Reentrant Tachycardia AVRT

- Second most common SVT
- Uses accessory path of <u>Myocardial tissue</u> 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

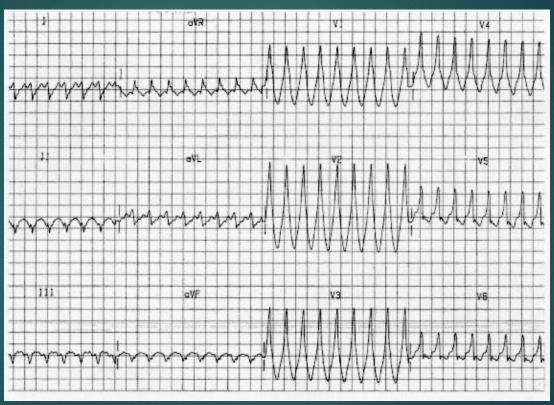
- Antegrade conduction path
  - ▶ In normal conduction, ventricles activated 1<sup>st</sup> by accessory path and 2<sup>nd</sup> by normal AV-His conduction
    - Preexcited ventricle, short P-R interval, delta wave
    - Variable degree of preexcitation amongst indivuiduals
    - Preexcitation can me modulated by antiarrythmics, autonomic tone
- Retrograde conduction path (25%)
  - Concealed pathways
  - not apparent on normal EKG
- Large electrical loop
- slower rates than AVNRT



## 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)

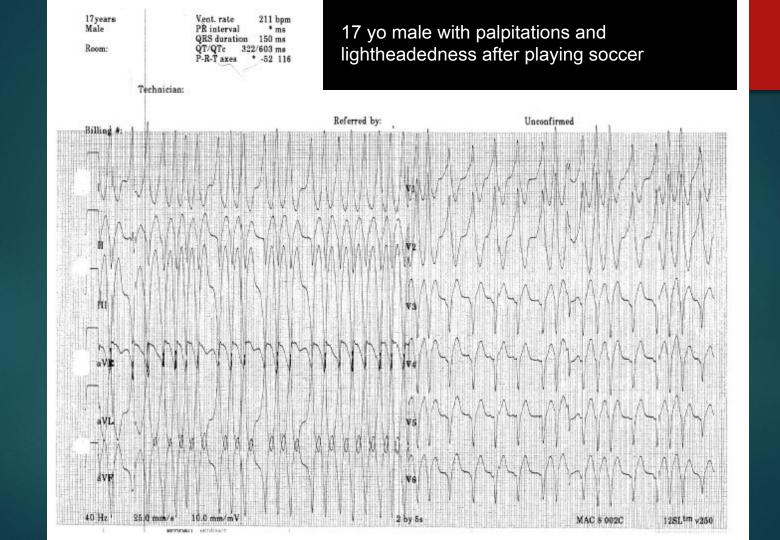
## Antidromic AVRT



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



## 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!!

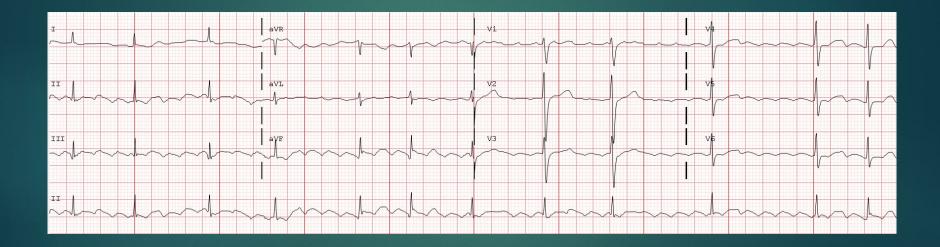
### **Board Question**

- 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"
  - ▶ MbMs

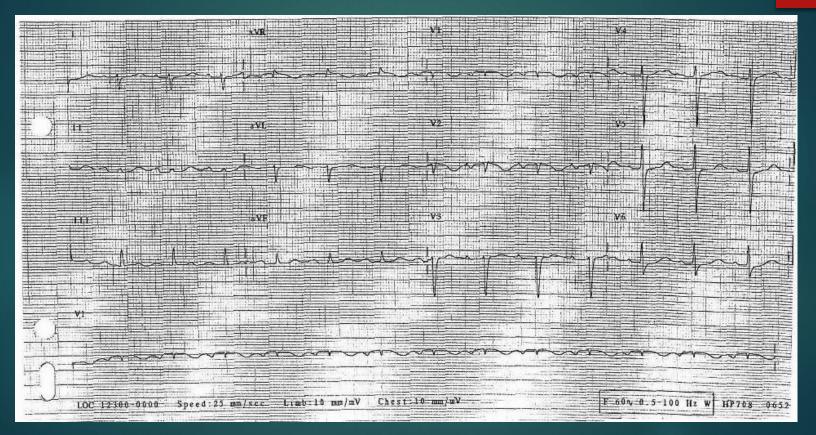
## Etiology of symptomatic recurrent VT

- 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

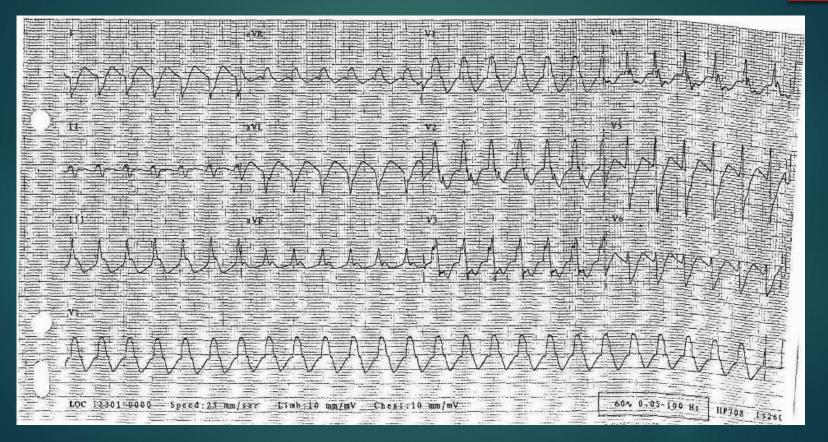
### Baseline EKG



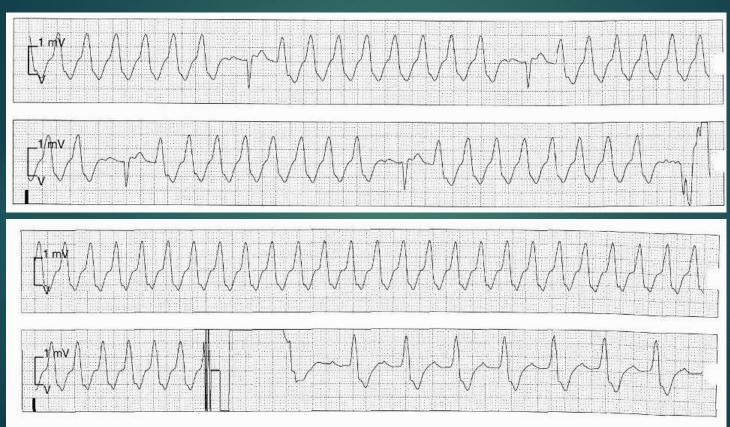
## EKG following code



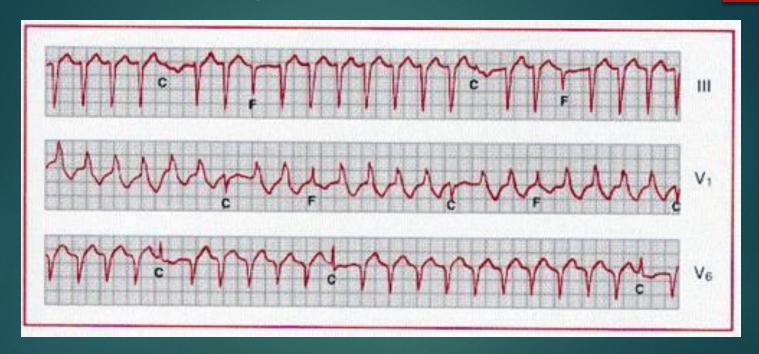
## EKG next evening...



## Rhythm Strip



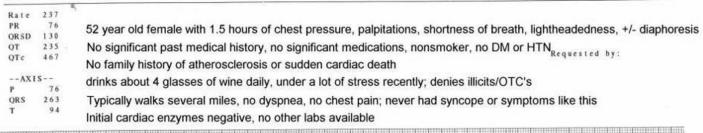
## Fusion and Capture Beats

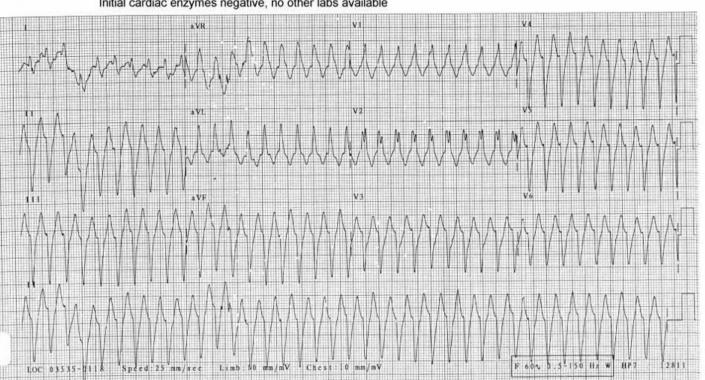


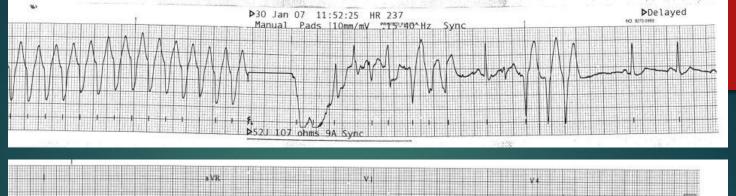
During the course of a tachycardia characterized by widespread, abnormal QRS complexes, the presence of fusion beats and capture beats provides maximum support for the diagnosis of VT

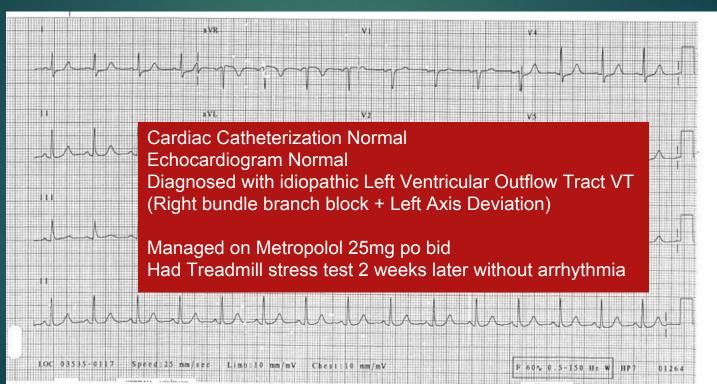
## Acute management of VT

- 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 antiarrhymic
  - ▶ 50J synchronized shock



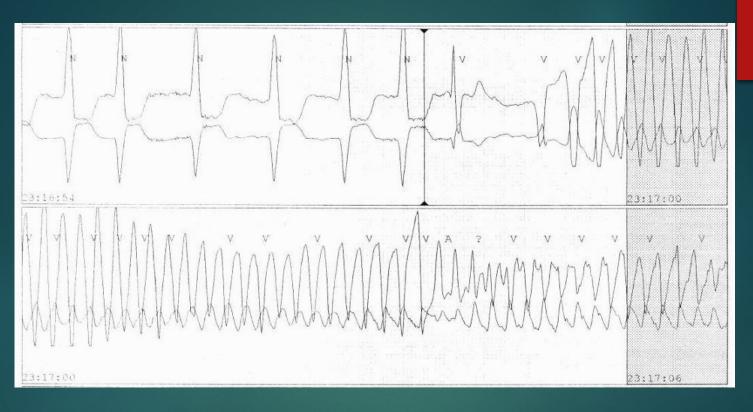






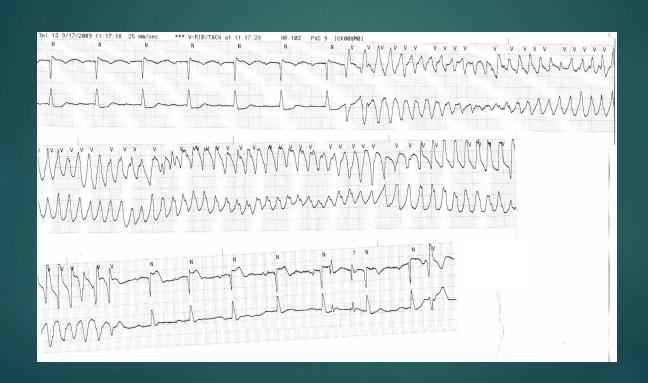
#### Torsades de Pointes

- 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

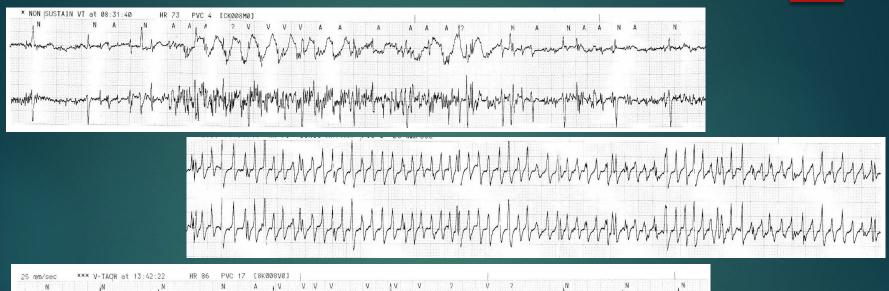
### Another Torsades...

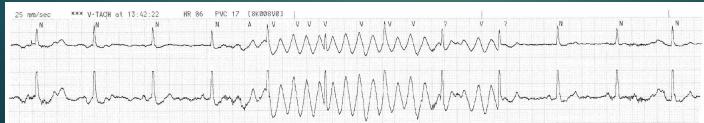


#### Acute treatment of Torsades

- 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<sup>st</sup> Degree AV Block

>200 ms from onset of P wave to onset of QRS

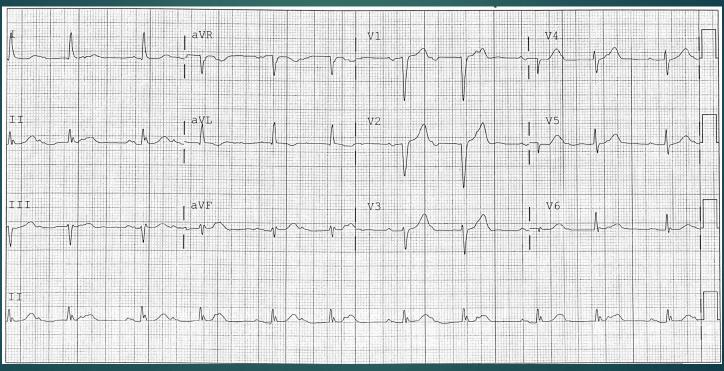


## 2<sup>nd</sup> Degree AV Block Type 1 - Wenkebach

P-R interval prolongs until QRS is dropped

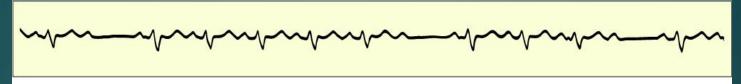


# 2<sup>nd</sup> Degree AV Block Type 1 - Wenkebach



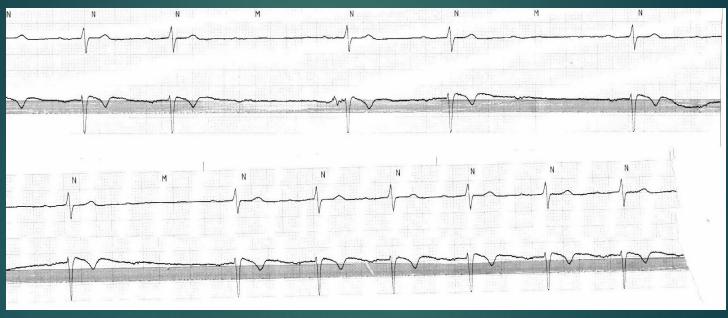
## 2<sup>nd</sup> Degree Heart Block Type 2

▶ PR interval remains constant, QRS drops unexpectedly



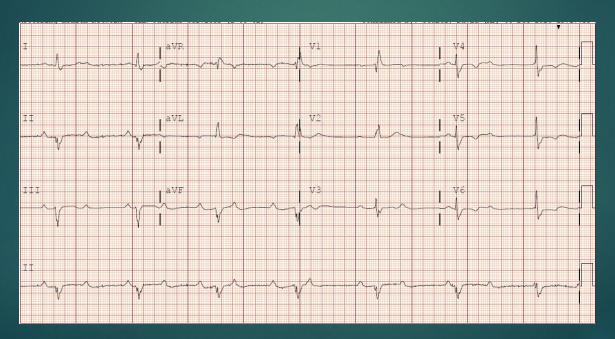
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## 2<sup>nd</sup> Degree Heart Block Type 2



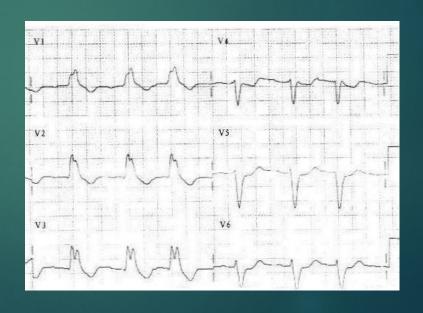
# 3<sup>rd</sup> degree Heart Block

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



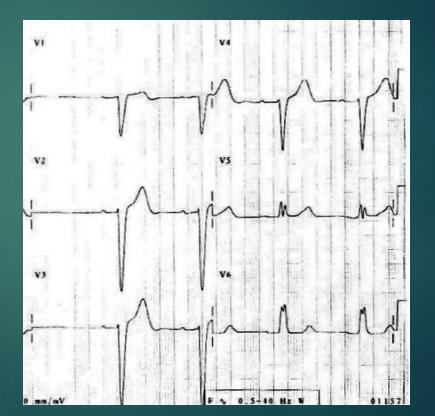
#### Bundle Branch Blocks

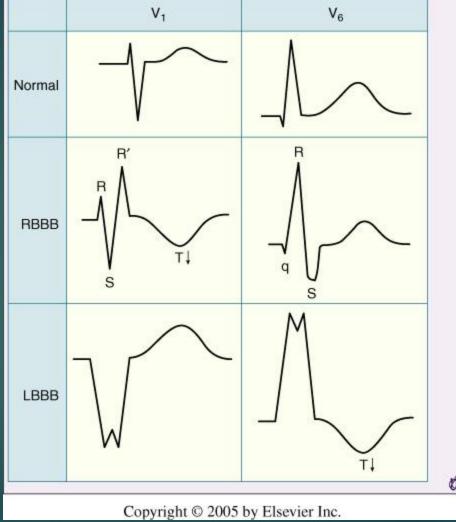
- Right Bundle Branch Block
  - QRS duration>120ms (3 small boxes)
  - ► rsR' in V1
  - 'Rabbit Ears'



#### Bundle Branch Blocks

- Left Bundle Branch Block
  - QRS duration >120ms (3 small boxes)
  - ► R in V6





#### Bundle Branch Block Criteria

TABLE 9-7

Common Diagnostic Criteria for Bundle Branch Blocks

#### Complete left bundle branch block

ORS duration ≥120 msec

Broad, notched R waves in lateral precordial leads (V<sub>5</sub> and usually leads I and aV<sub>i</sub>

Small or absent initial r waves in right precordial leads V<sub>2</sub>) followed by deep S waves

Absent septal q waves in left-sided leads

Prolonged intrinsicoid deflection (>60 msec) in V<sub>5</sub> and

#### Complete right bundle branch block

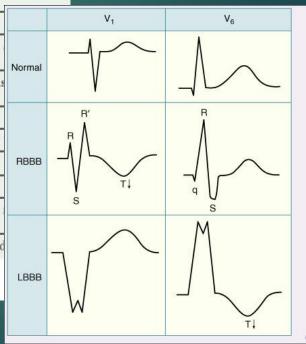
QRS duration ≥120 msec

Broad, notched R waves (rsr', rsR', or rSR' patterns) in precordial leads (V<sub>1</sub> and V<sub>2</sub>)

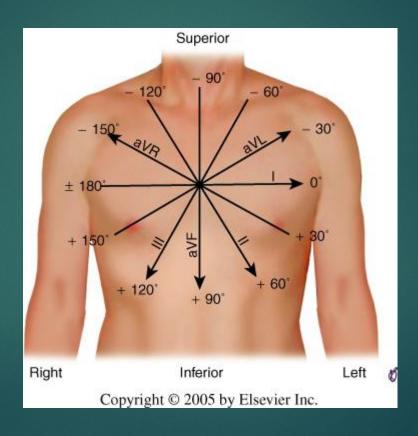
Wide and deep S waves in left precordial leads (V5 and

\*Criterion required by some authors.

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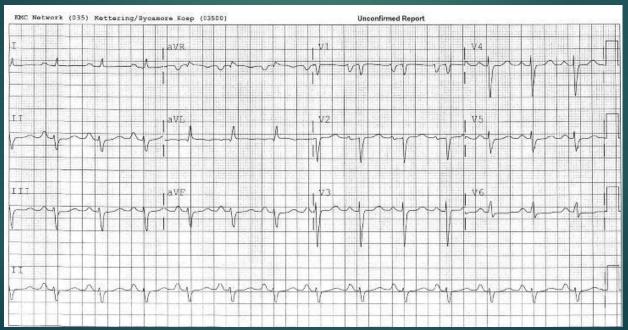


## Axis

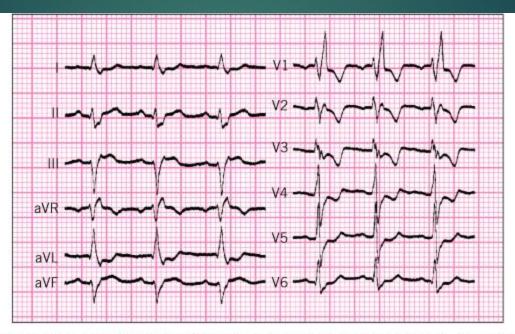


### Left Anterior Fascicular Block

- Frontal Axis -45 to -90 degrees
- QRS <120ms</p>
- rS pattern in II, II, aVF (inferior leads)



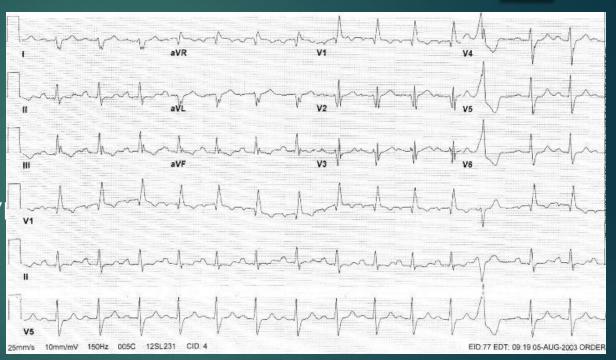
### LAFB + RBBB



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### Left Posterior Fascicular Block

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



#### Fascicular Blocks

QRS Duration <120ms

#### LAHB (LAFB)

Severe LAD without explanation

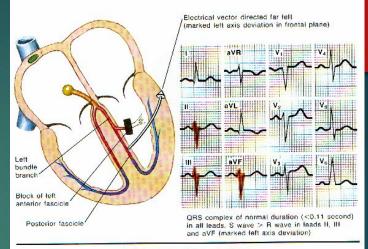
- •Deep S waves in II, III, aVF
- •Frontal Axis <-45 to -60 degrees
- •Positive in I, Negative in aVF
- •Not explained by LBBB, LVH, inferior infarct

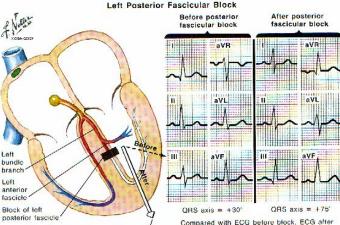
#### LPHB (LPFB)

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

#### Left Anterior Fascicular Block





Electrical vector directed more

right than before block, but usually within normal QRS axis range

block shows shift of frontal QRS axis to right

#### Fascicular Block Criteria

TABLE 9-6

Common Diagnostic Criteria for Unifascicular Blocks

#### Left anterior fascicular block

Frontal plane mean QRS axis of -45 to -90 degrees with rS patterns in leads II, III, and aV<sub>I</sub> and a qR pattern in lead aV<sub>I</sub>

QRS duration less than 120 msec

#### Left posterior fascicular block

Frontal plane mean QRS axis of ±120 degrees

RS pattern in leads I and aV1 with qR patterns in inferior leads

QRS duration of less than 120 msec

Exclusion of other factors causing right axis deviation (e.g., right ventricular overload patterns, lateral infarction)

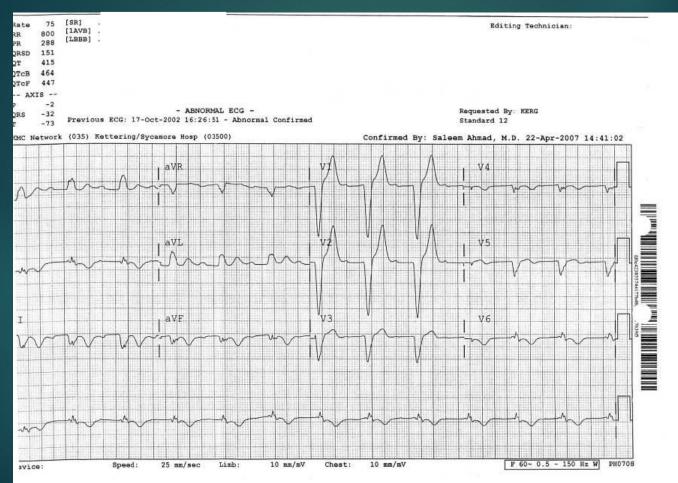
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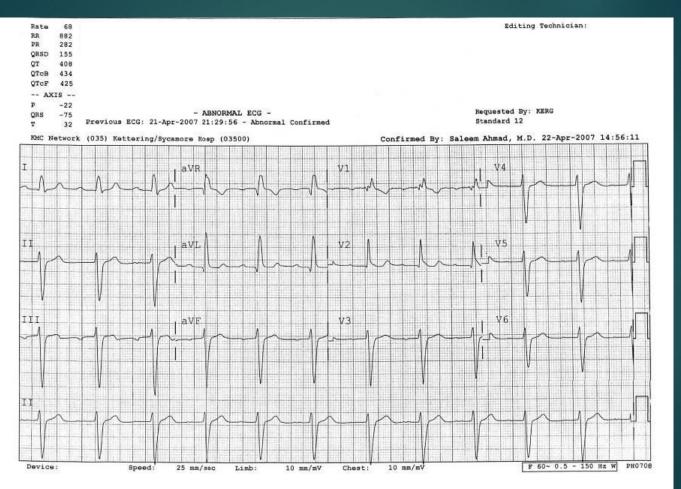
COMMON CAUSES OF ATRIOVENTRICULAR AND INTRAVENTRICULAR CONDUCTION DISTURBANCE	
Intrinsic causes	Congenital Sclerodegenerative Ischemia Trauma (surgical) Connective tissue disorders Tumors Sarcoidosis
Extrinsic causes	Drugs Autonomic disorders Hypothyroidism

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#### 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

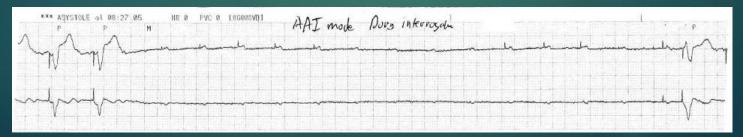




## Later that night....



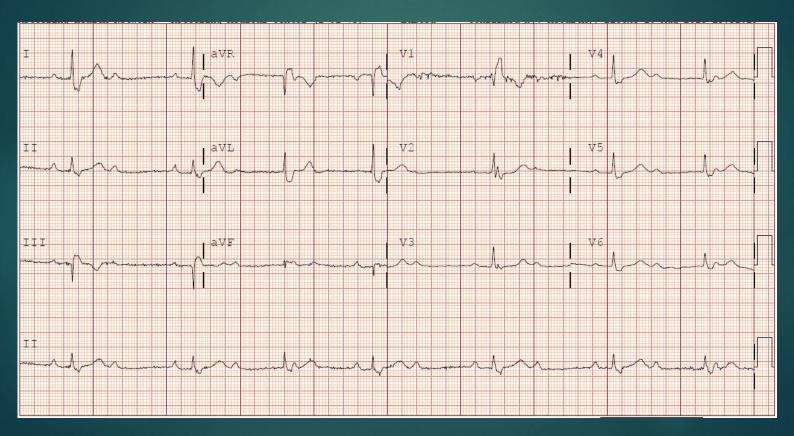




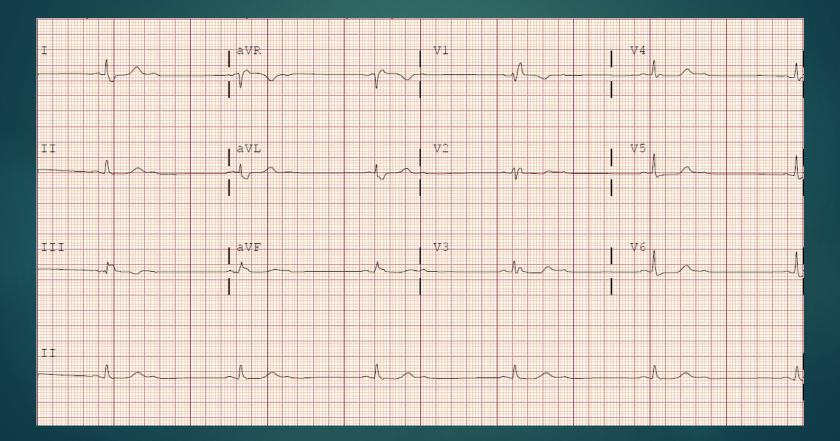
#### Board Pearls for Heart Block

- 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
- Chauhan VS, Krahn AD, Klein GJ, Skanes AC, Yee R. Supraventricular tachycardia. Med Clin North Am. 2001 Mar;85(2):193-223, ix.
- Ganz LI, Friedman PL. Supraventricular tachycardia. N Engl J Med. 1995 Jan 19;332(3):162-73.