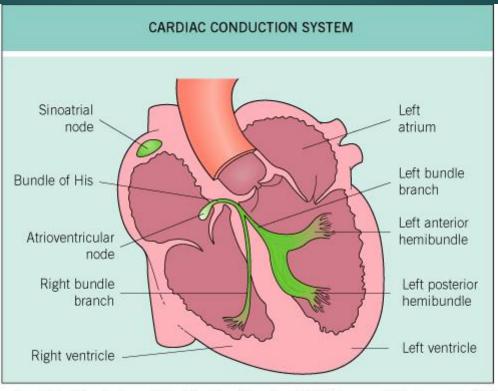
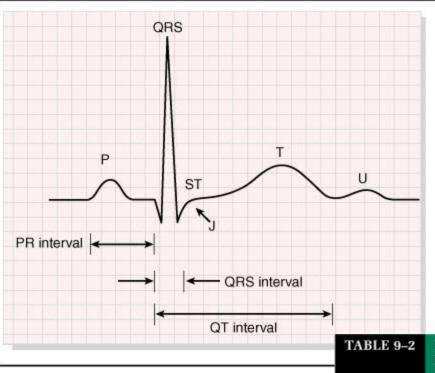
Fast & Slow
Tachy & Brady
Arrhythmias



DAVID STULTZ, MD, FACC KPN HEART & VASCULAR AUGUST 7, 2017



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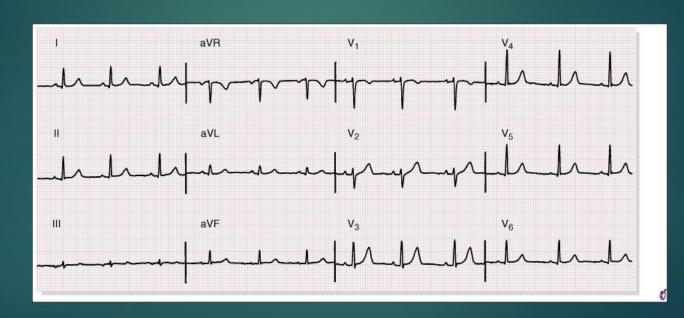
Normal Values for Durations of Electrocardiographic Waves and Intervals in Adults

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

^{*}See text for further discussion.

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Normal 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

1st Degree AV Block

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

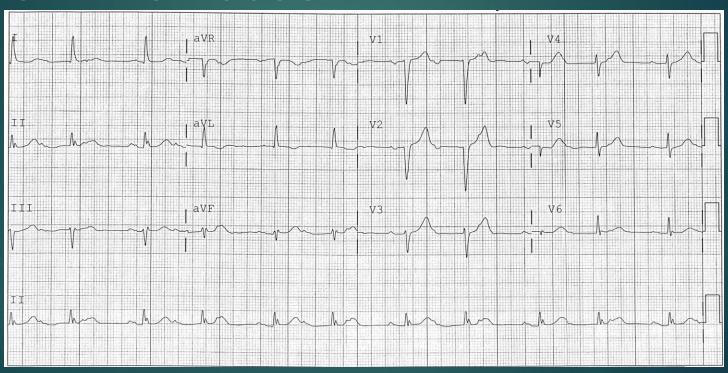


2nd Degree AV Block Type 1 - Wenkebach

▶ P-R interval prolongs until QRS is dropped

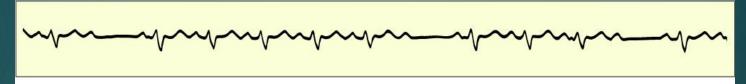


2nd Degree AV Block Type 1 - Wenkebach



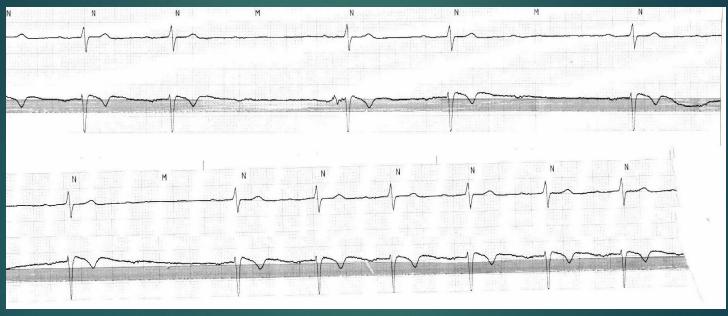
2nd Degree Heart Block Type 2

▶ PR interval remains constant, QRS drops unexpectedly



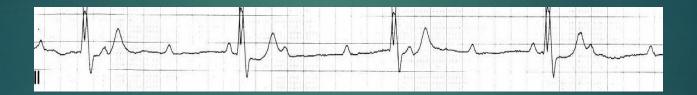
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2nd Degree Heart Block Type 2



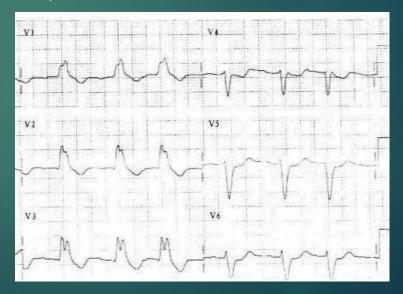
3rd degree Heart Block

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



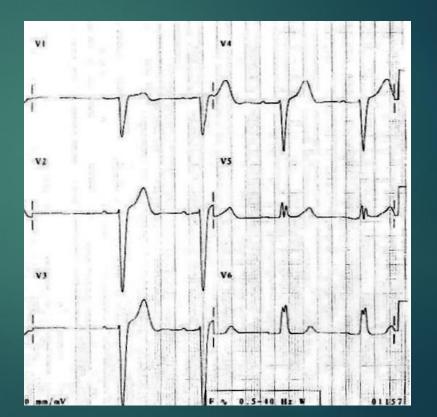
Bundle Branch Blocks

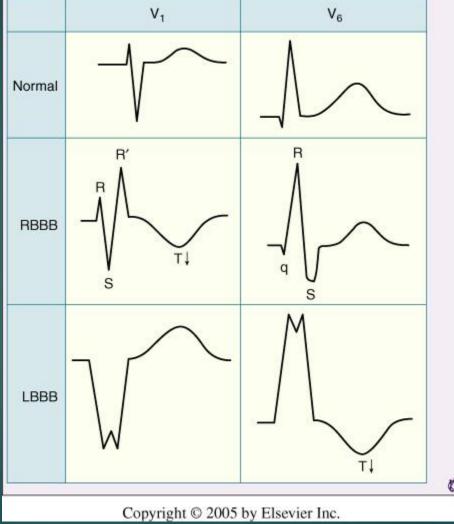
- Right Bundle Branch Block
 - ▶ QRS duration >120ms (3 small boxes)
 - ▶ rsR' in V1
 - 'Rabbit Ears'



Bundle Branch Blocks

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





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 (V₅ and usually leads I and aV_i

Small or absent initial r waves in right precordial leads V₂) followed by deep S waves

Absent septal q waves in left-sided leads

Prolonged intrinsicoid deflection (>60 msec) in V₅ and

Complete right bundle branch block

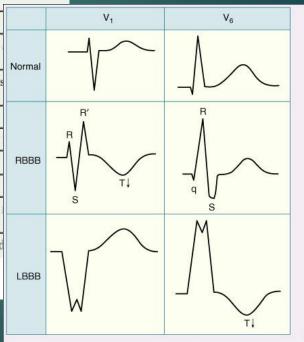
ORS duration ≥120 msec

Broad, notched R waves (rsr', rsR', or rSR' patterns) in precordial leads (V_1 and V_2)

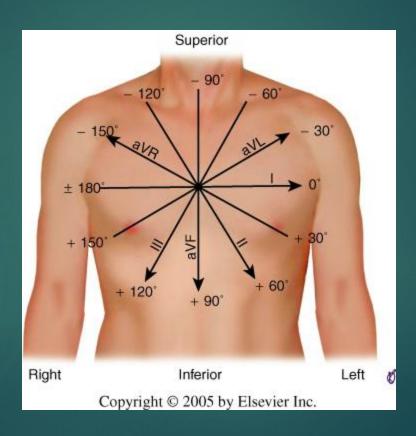
Wide and deep S waves in left precordial leads (V5 and

*Criterion required by some authors.

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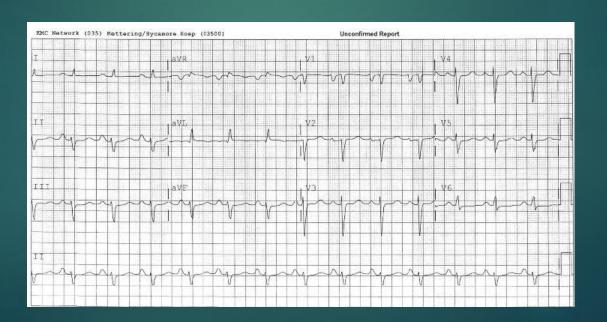


Axis

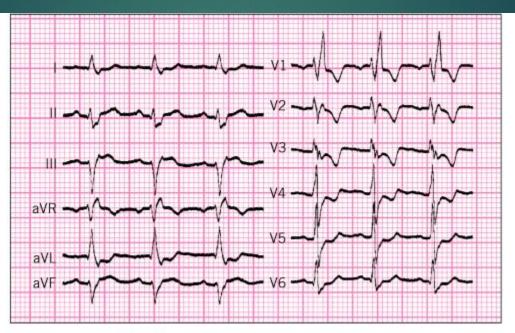


Left Anterior Fascicular Block

- ► Frontal Axis -45 to -90 degrees
- ▶ QRS <120ms
- 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



Fascicular Blocks

QRS Duration <120ms

LAHB (LAFB)

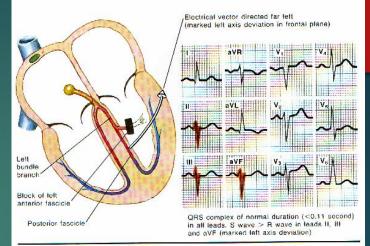
Severe LAD without explanation

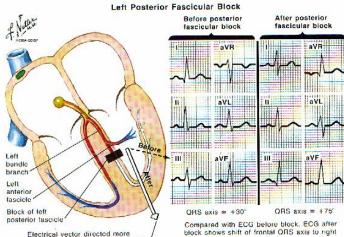
- •Deep S waves in II, III, aVF
- •Frontal Axis <-45 to -60 degrees
- •Positive in I, Negative in aVF
- •Not explained by LBBB, LVH, inferior infarct

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





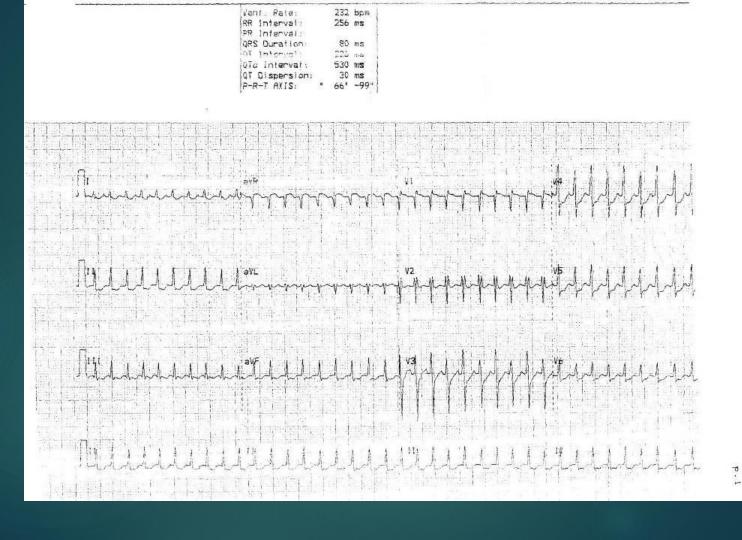
right than before block, but usually within normal QRS axis range

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	

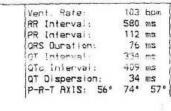
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Case presentation

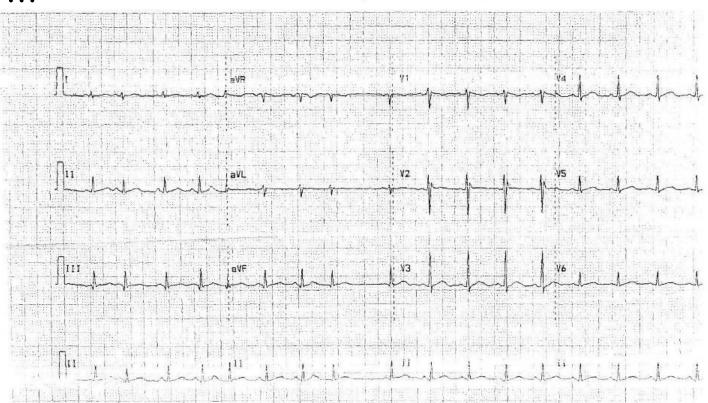
- 21 year old white female presents to the emergency room with palpitations for 1 hour
- Mild lightheadedness, no syncope
- No significant past medical history
- No meds except OCP
- Cramming for exams, took no doze and Red Bull this morning after pulling an all-nighter



After intervention



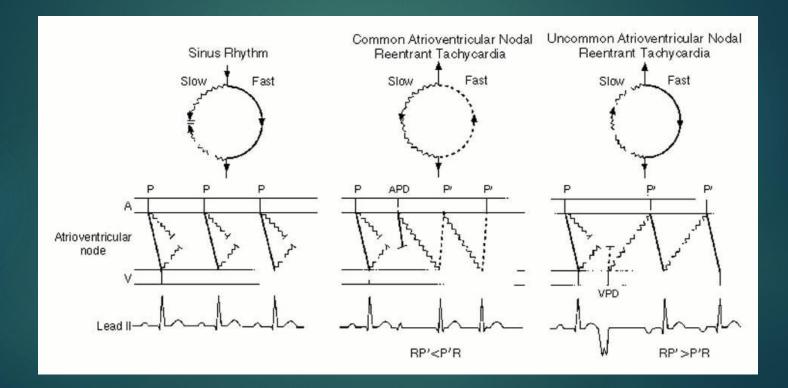




General Mechanism of Nodal Dependent SVT

- ▶ 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



AV Node Reentrent Tachycardia AVNRT

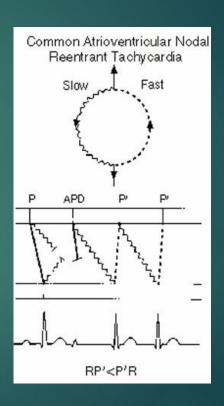
- 60% of all SVT's (most common)
- ▶ 70% are female
- Mostly patients age 30-40's
- ▶ 90% Typical (Slow-Fast)
 - Antegrade limb has slow conduction, retrograde is fast
- ▶ 10% Atypical
 - ► Fast-Slow
 - ▶ Slow-Slow
 - ▶ Fast-Fast

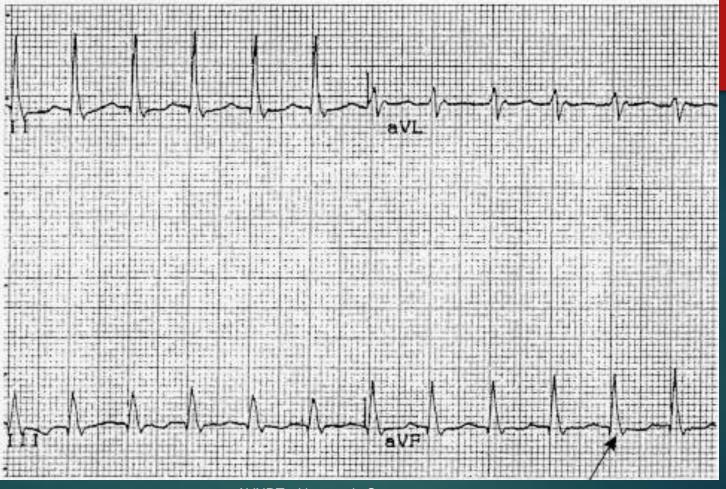
Typical AVNRT

- 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

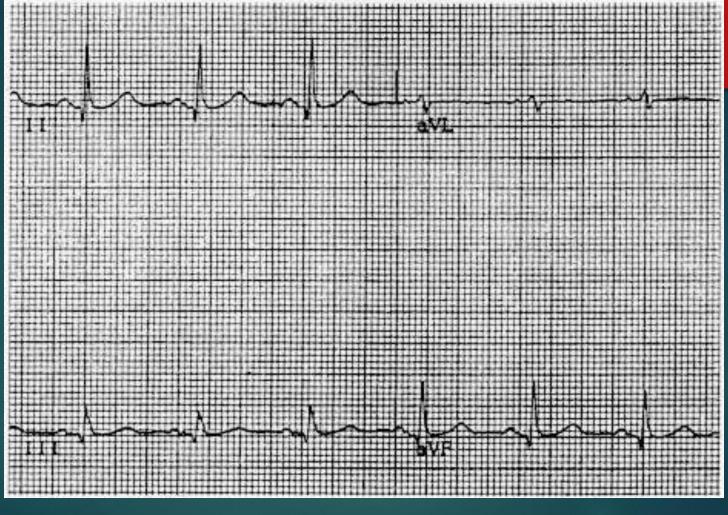
EKG Features of AVNRT

- 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

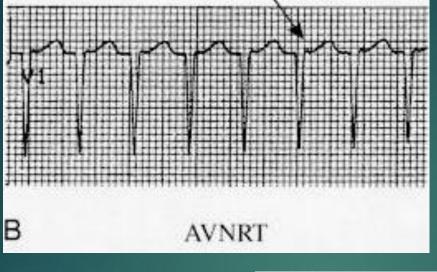




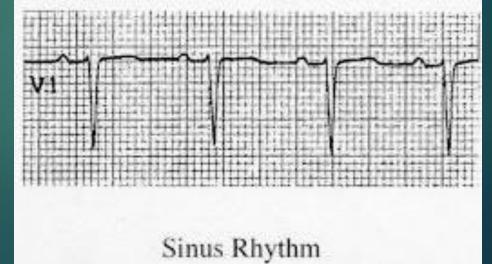
AVNRT with pseudo S wave



Chauhan NSR



AVNRT with pseudo R waves



Chauhan

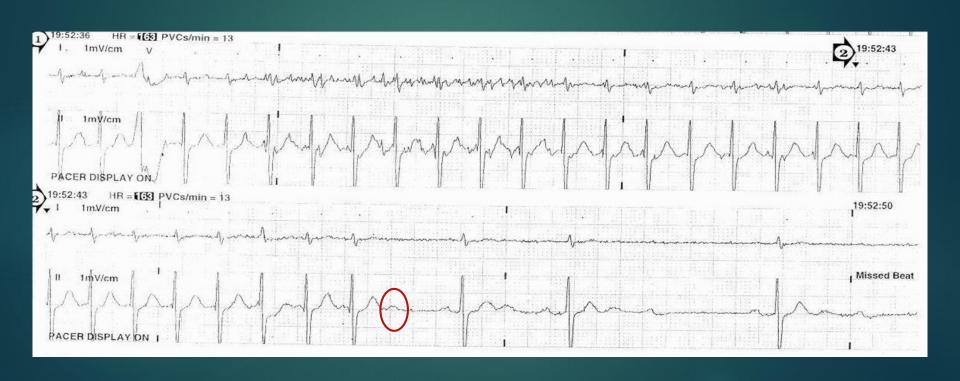
Breaking a tachycardia

- 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 1st by accessory path and 2nd 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)

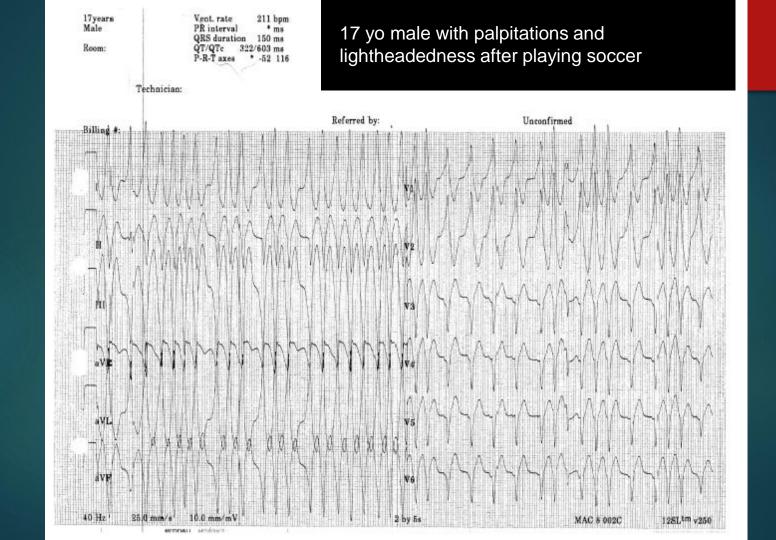
Antidromic AVRT



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!!

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

Ventricular tachycardia

- Wide complex, regular tachycardia
- ▶ May be "stable" or unstable
- A word on 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

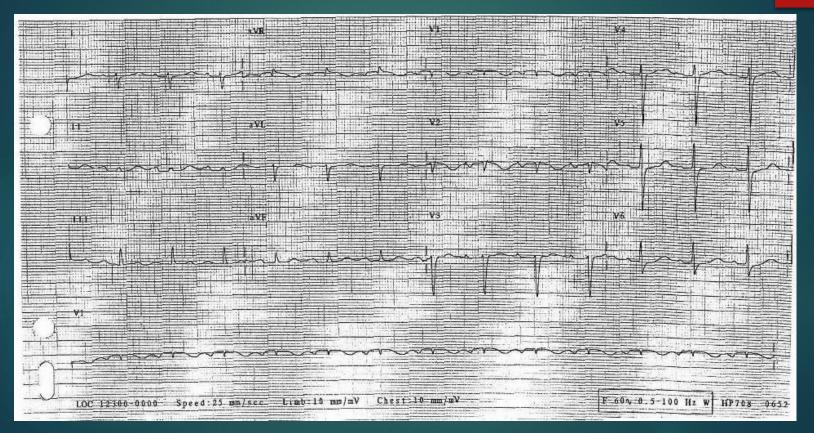
Etiology of symptomatic recurrent VT

- ▶ 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

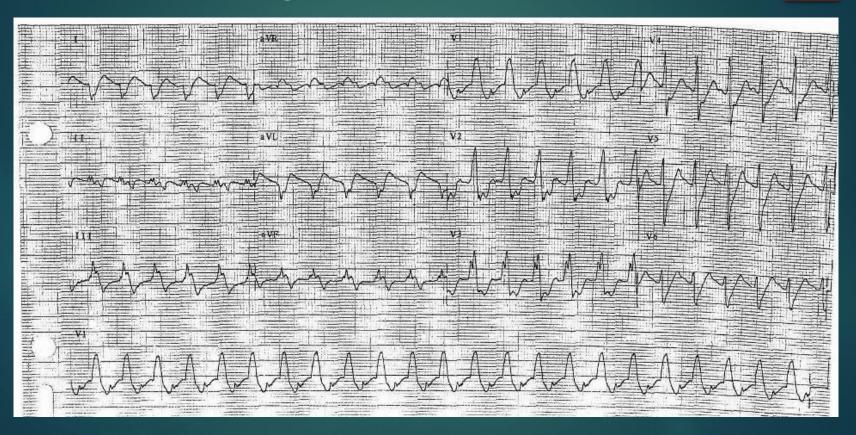
Case VT

- ▶ 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

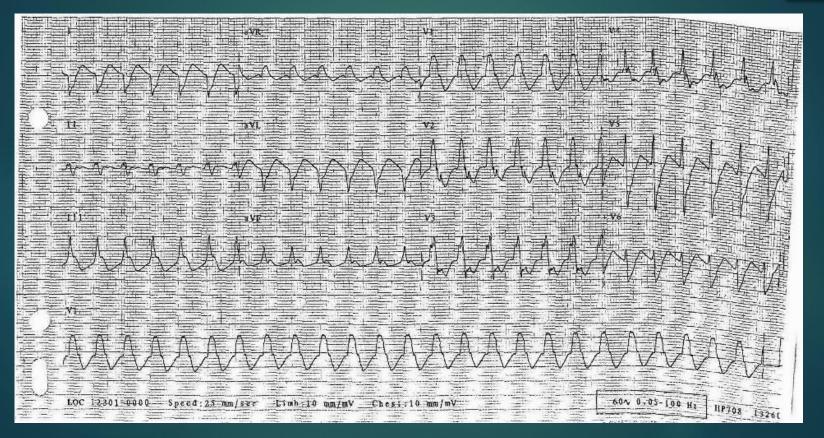
Baseline EKG



EKG following code



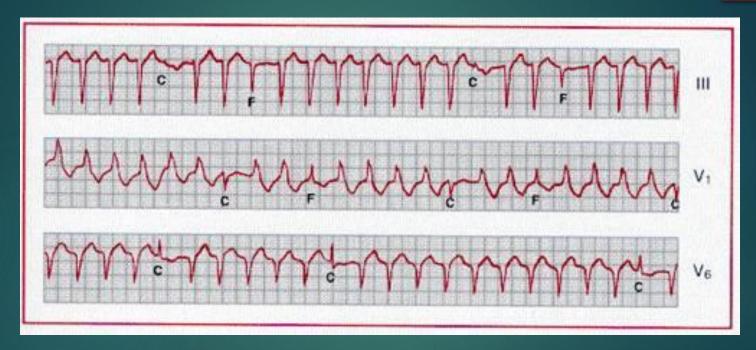
EKG next evening...



Rhythm Strip



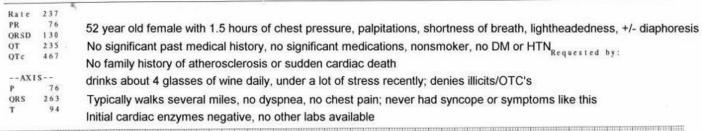
Fusion and Capture Beats

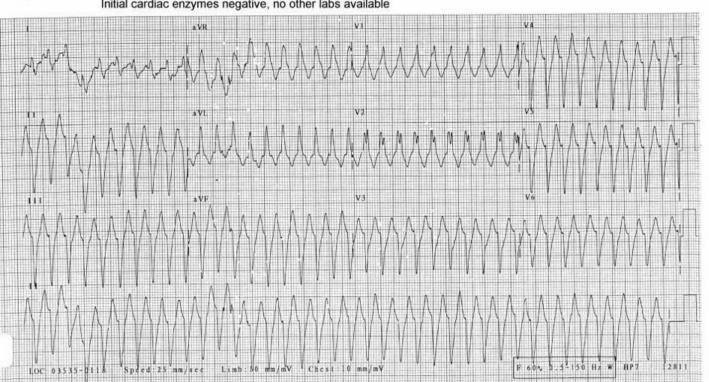


During the course of a tachycardia characterized by widespread, abnormal QRS complexes, the presence of fusion beats and capture beats provides maximum support for the diagnosis of VT

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

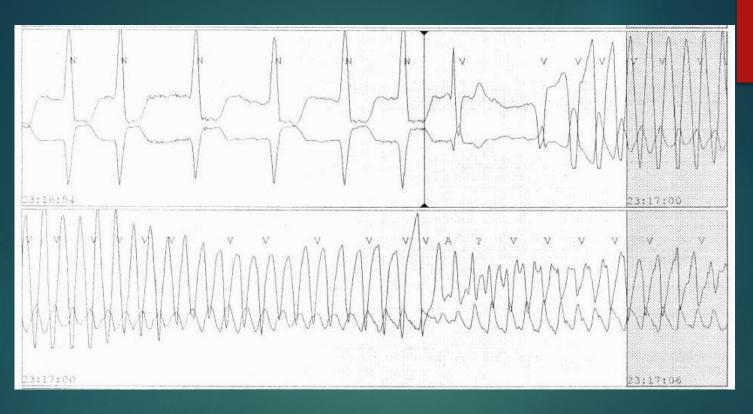






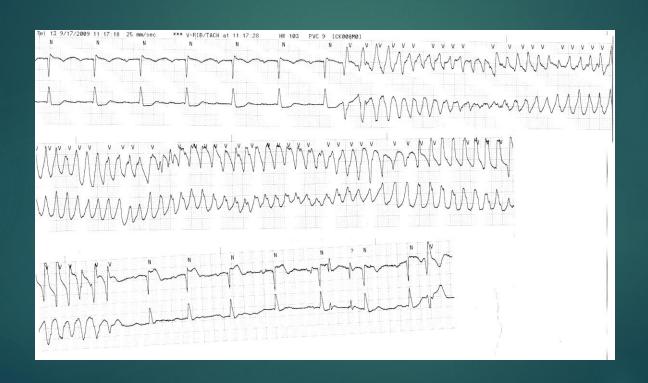
Torsades de Pointes

- ► 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

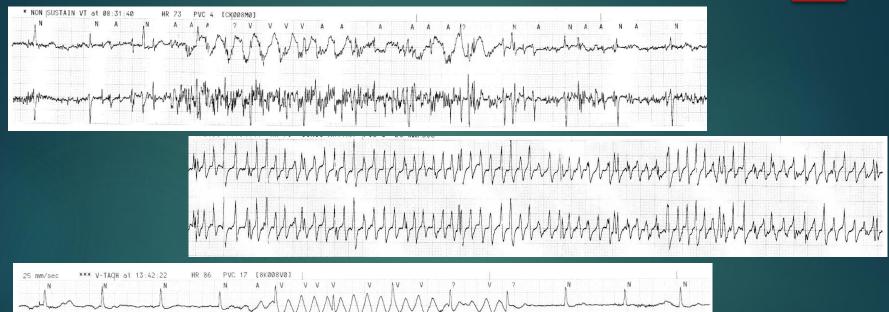
Another Torsades...

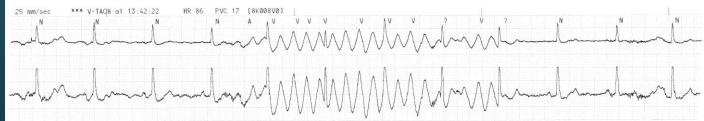


Acute treatment of Torsades

- 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 3N...





Pseudo-Ventricular Tachycardia (artifact)

And now to Slow it down....

1st Degree AV Block

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



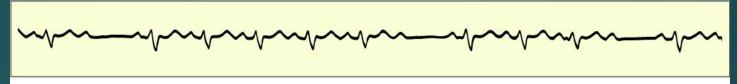
2nd Degree AV Block Type 1 - Wenkebach

▶ P-R interval prolongs until QRS is dropped



2nd Degree Heart Block Type 2

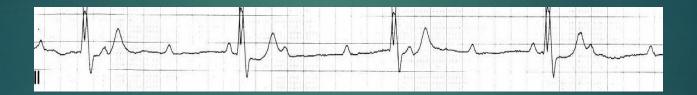
▶ PR interval remains constant, QRS drops unexpectedly



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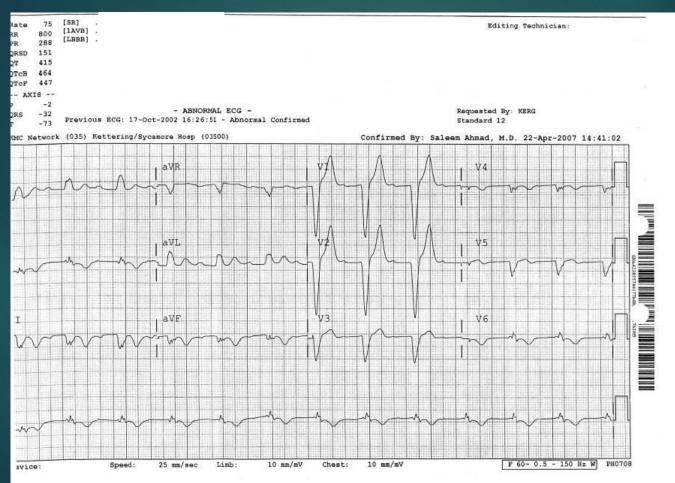
3rd degree Heart Block

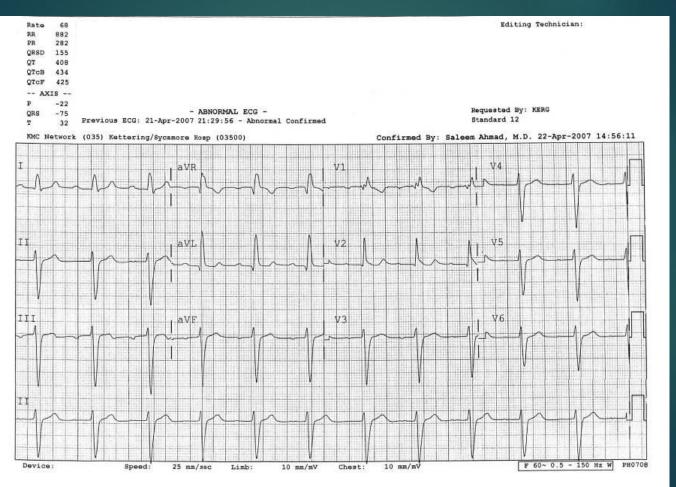
- ▶ P rate faster than QRS rate
- ▶ No correlation between P's and QRS



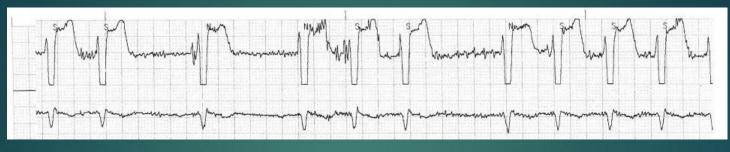
Case Presentation

- ▶ 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

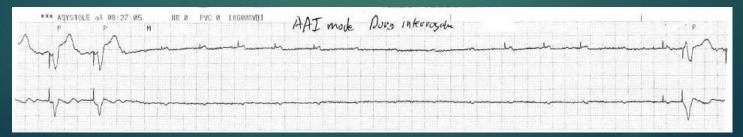




Later that night....







Board Pearls for Heart Block

- ► Think of potential causes of heart block
 - ▶ Lyme disease
 - Sarcoidosis
 - Drug overdose
 - ▶ Hyperkalemia
 - ▶ Hypothyroidism

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

- ► 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.

