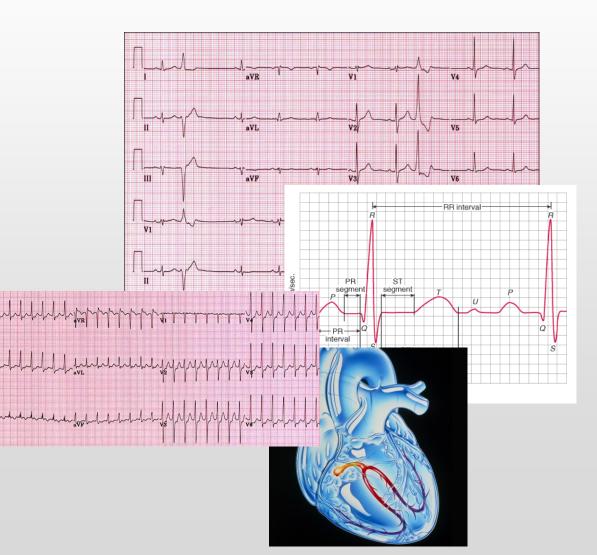
ARRHYTHMIA EVALUATION

ABHISHEK K. BHAGAT, M.D.

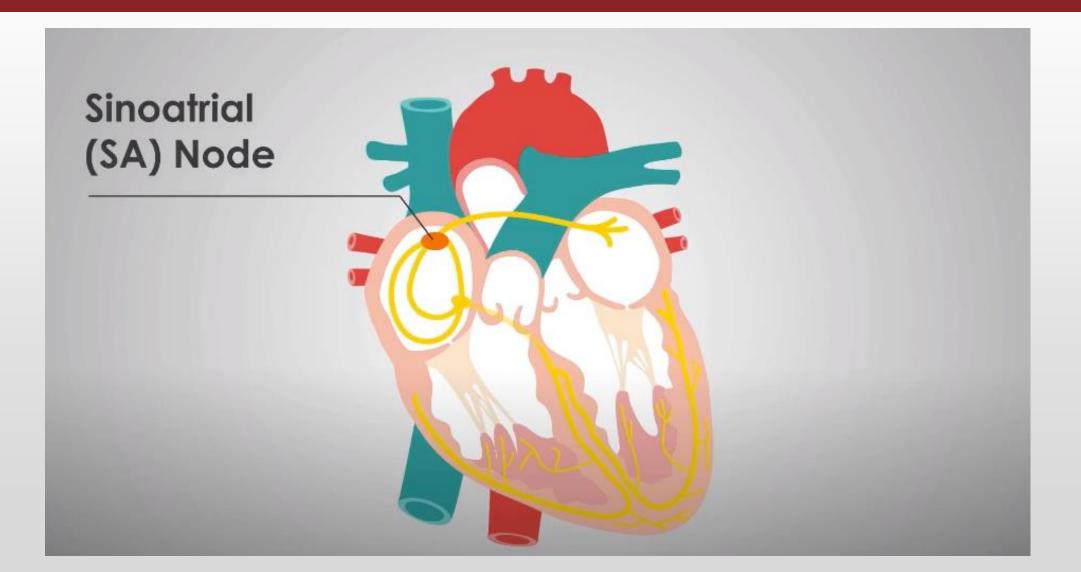


Objectives

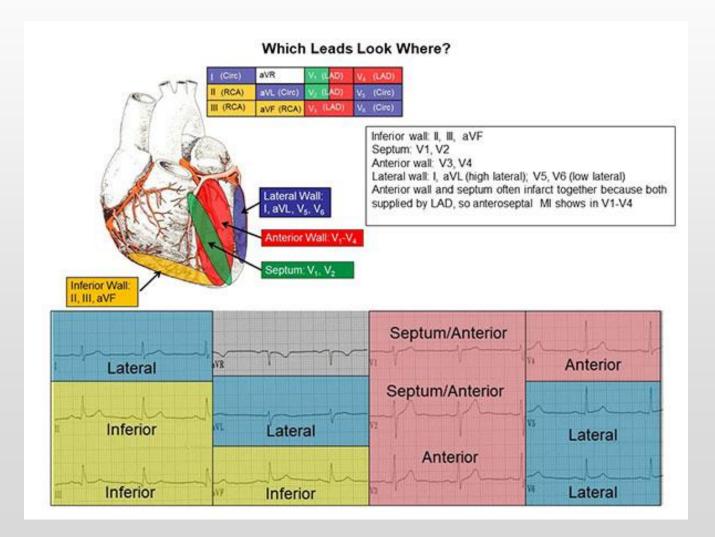
- Cardiac Conduction System
- EKG Basics
- AV Blocks
- Mechanism of Arrhythmias
- Evaluating SVTs
- Identifying Brugada



Cardiac Conduction System

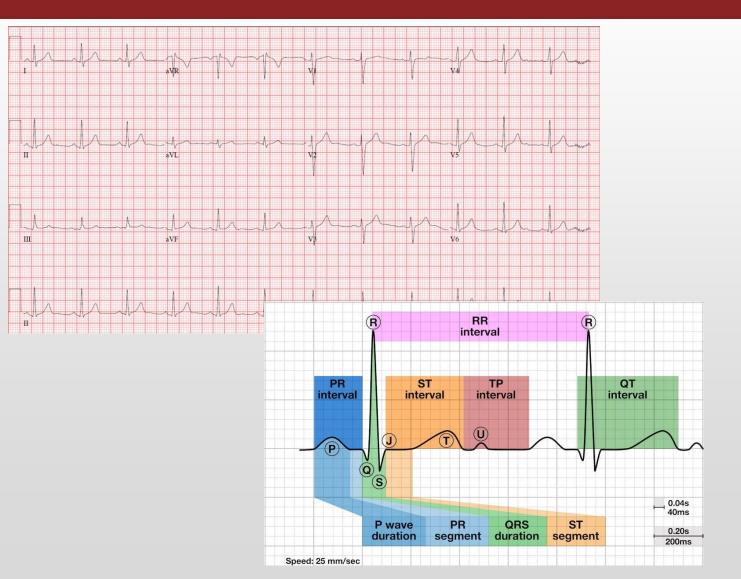


EKG BASICS



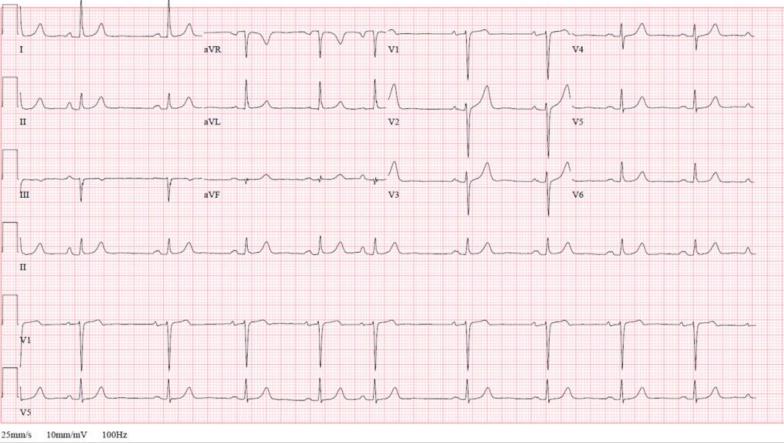
EKG Basics

- Rate and rhythm
- Axis
- PR interval (120 200 ms)
- QRS complex (60 100 ms)
- ST segment and T wave, Q wave
- QT interval (prolonged QTc: >450 (m) or >460 (f)
- Extra: R-wave progression, LVH, low voltage, left/right atrial enlargement, etc.



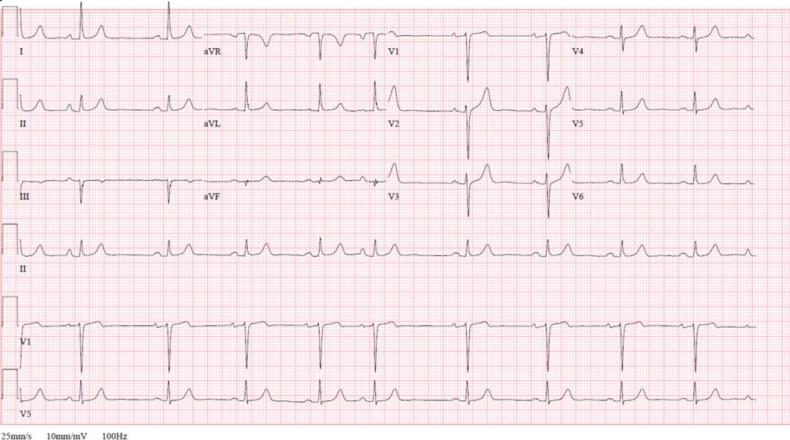
45M w/ medical hx significant for Grave's disease presents to your clinic complaining of occasional "skipped beats" sensation. Diagnosis?

- A. Atrial fibrillation (rate controlled)
- B. Sinus rhythm w/ premature atrial contraction (PAC)
- C. Atrial flutter (rate controlled)
- D. First degree AV block
- E. Mobitz 2 AV block



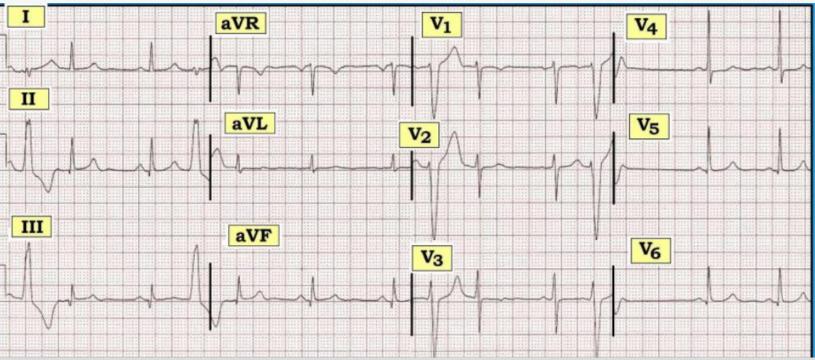
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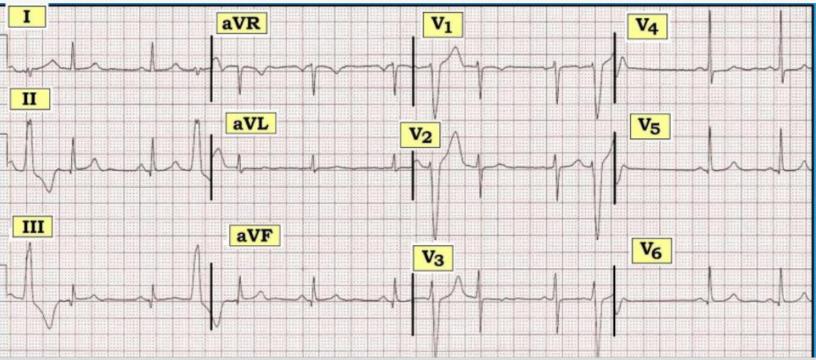
55F w/ medical hx significant for HTN and ESRD on HD presents complaining of intermittent palpitations. Diagnosis?

- A. Atrial fibrillation
- B. Ventricular bigeminy
- C. Sinus rhythm w/ premature atrial contractions (PACs)
- D. Sinus rhythm w/ premature ventricular contractions (PVCs)
- E. Focal atrial tachycardia



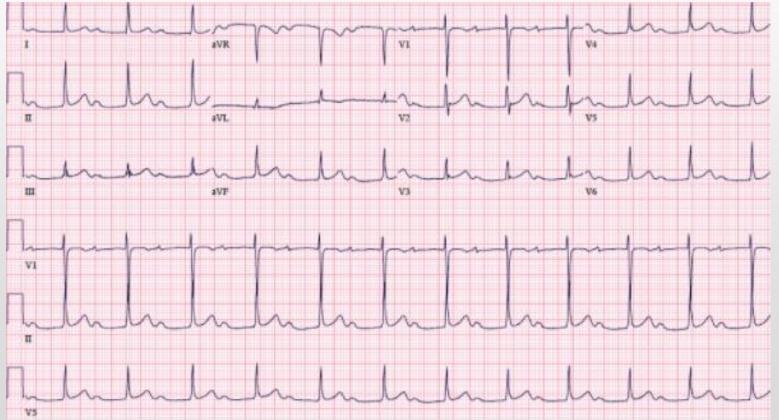
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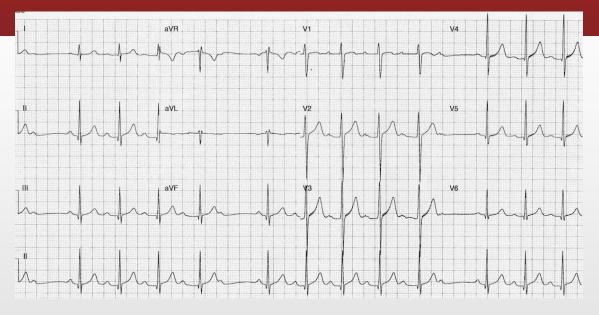
1st Degree AV Block

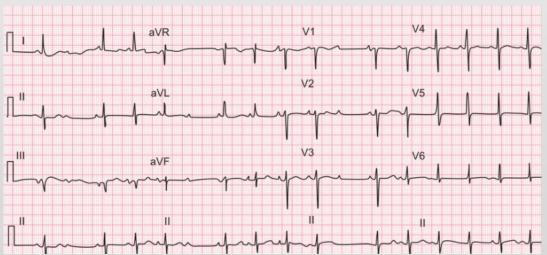
- Slow conduction through the AV node delay of impulse from atria to ventricles
- PR interval >200 ms (>300 ms "marked" 1st degree AV block)
- Causes: fibrotic changes in conduction system in elderly, CAD, electrolyte disturbances (hypoK), inflammation/infection (ex. endocarditis, Lyme), infiltrative diseases (ex. sarcoidosis)
- Management: asymptomatic no further evaluation. Symptomatic (without reversible causes) – permanent pacemaker.



2nd Degree AV Block

- Mobitz 1 (Wenckebach) progressive PR interval prolongation followed by a nonconducted P wave
- Most common site of block is in AV node
- Causes: CAD (RCA), inflammation/infection, iatrogenic (AVN blockers, post-cardiac surgery), hyperkalemia, hypothyroidism
- Mobitz 2 PR interval remains unchanged prior to a non-conducted P wave
- Most common site of block is below the AV node – bundle of His and bundle branches





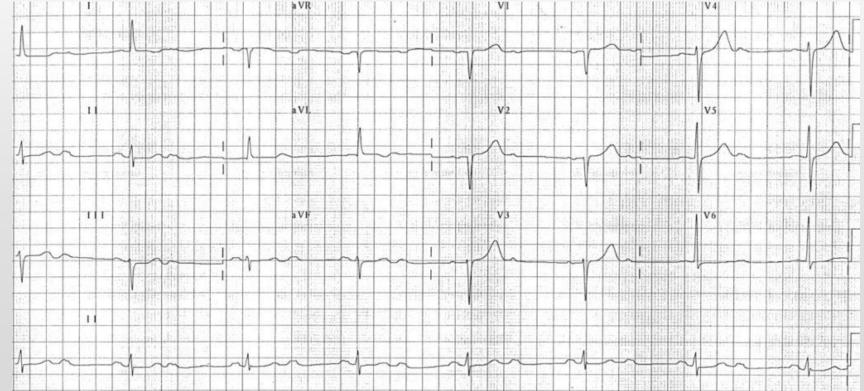
3rd Degree (Complete) AV Block

- No atrial impulses reach the ventricle. Ventricular depolarization maintained by junctional or ventricular escape rhythm – AV dissociation
- Causes: fibrotic changes in conduction system in elderly, CAD, electrolyte disturbances (hypo/hperK), inflammation/infection (ex. endocarditis, Lyme), infiltrative diseases (ex. sarcoidosis)
- High risk for sudden cardiac death
- Management: permanent pacemaker



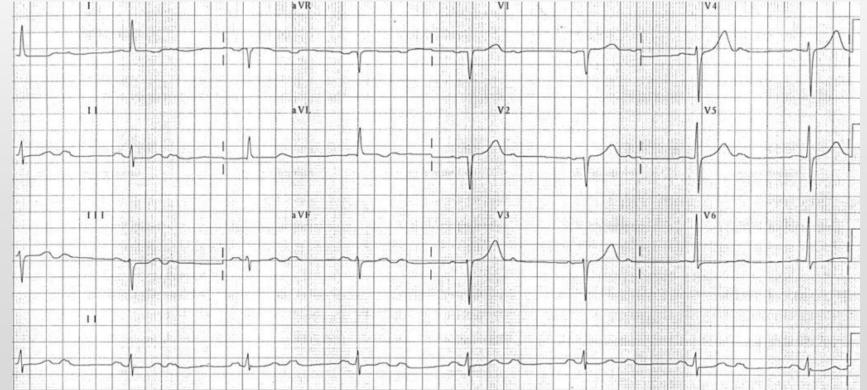
65F w/ medical hx significant for DM2 and ESRD on HD presents complaining of feeling tired and lightheaded for 1 day. Labs show K of 2.7. Diagnosis?

- A. Complete heart block
- B. Sinus bradycardia w/ PACs
- C. Mobitz 1 (Wenckebach) AV block
- D. Atrial fibrillation w/ slow ventricular response
- E. Mobitz 2 AV block



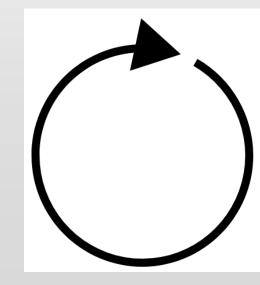
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- E. Mobitz 2 AV block



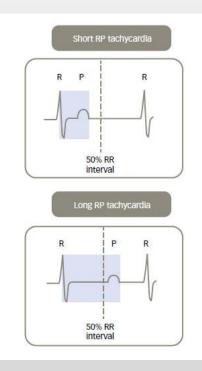
Mechanism of Arrhythmias

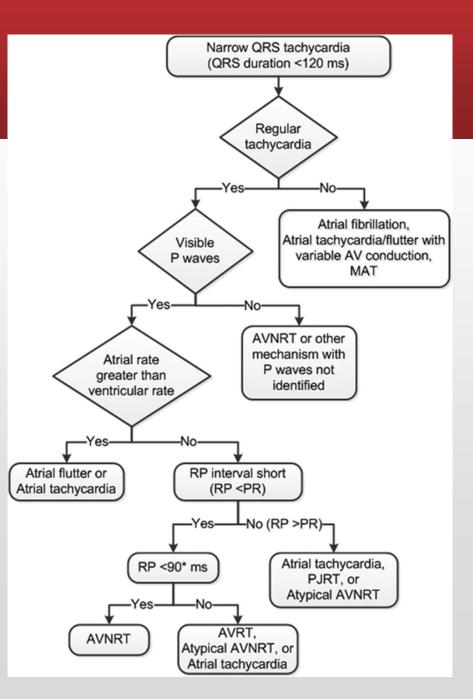
- Arrhythmias arise if impulse formation is abnormal or impulse propagation is abnormal
 - Abnormal impulse formation is due to abnormal automaticity or triggered activity
 - Many structures of the heart possess automaticity (able to depolarize spontaneously) ex. SA node, parts of atrial myocardium, His-Purkinje system, etc.
 - Triggered activity (after depolarization) is depolarization which occurs during repolarization (early depolarization) or after repolarization (late depolarization)
 - These depolarizations can cause extra cardiac contraction
 - Triggered activity (after depolarizations) can cause extra contractions but they do not cause persistent arrhythmias (unless the contraction triggers another arrhythmia mechanism – ex. re-entry)
- Abnormal impulse propagation (re-entry)
 - Re-entry means that the depolarizing impulse moves around itself in a circle
 - Ex: atrial flutter, AVNRT, VF

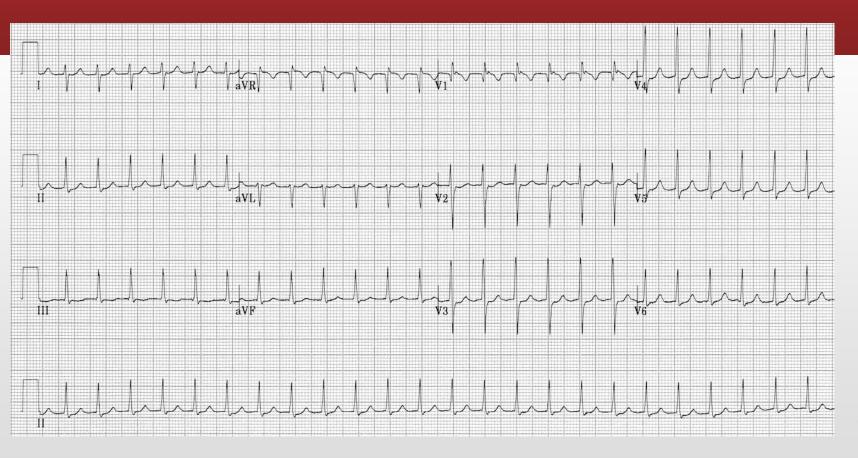


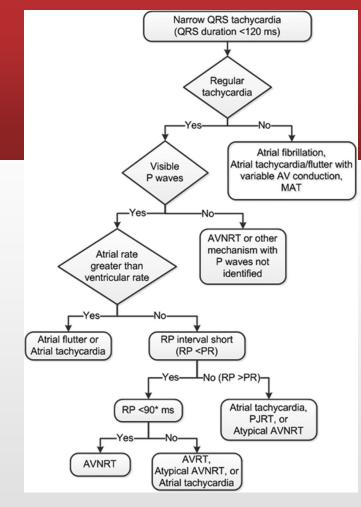
SVT

- Tachycardia originating His bundle or above
 - Inappropriate sinus tachycardia, AT (focal/multifocal), atrial flutter, atrial fibrillation, AVNRT, accessory pathway-mediated tachycardias (WPW)

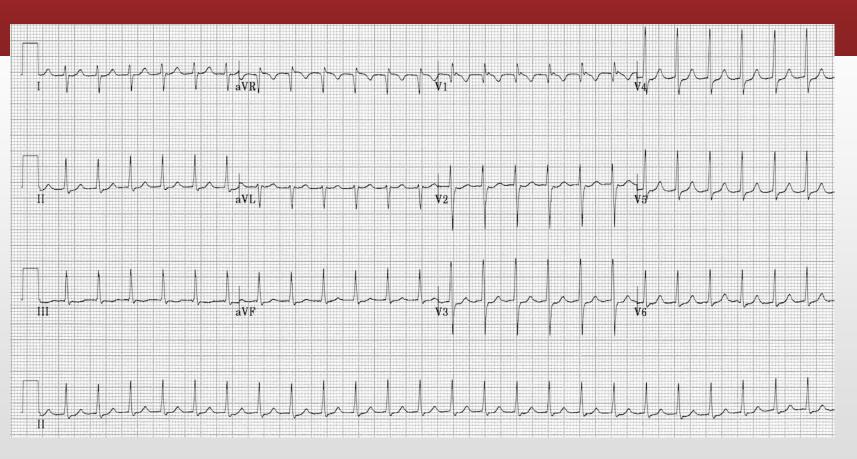


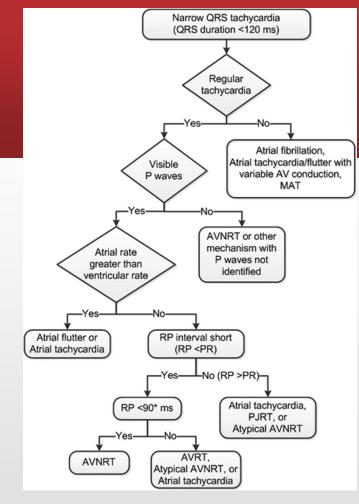




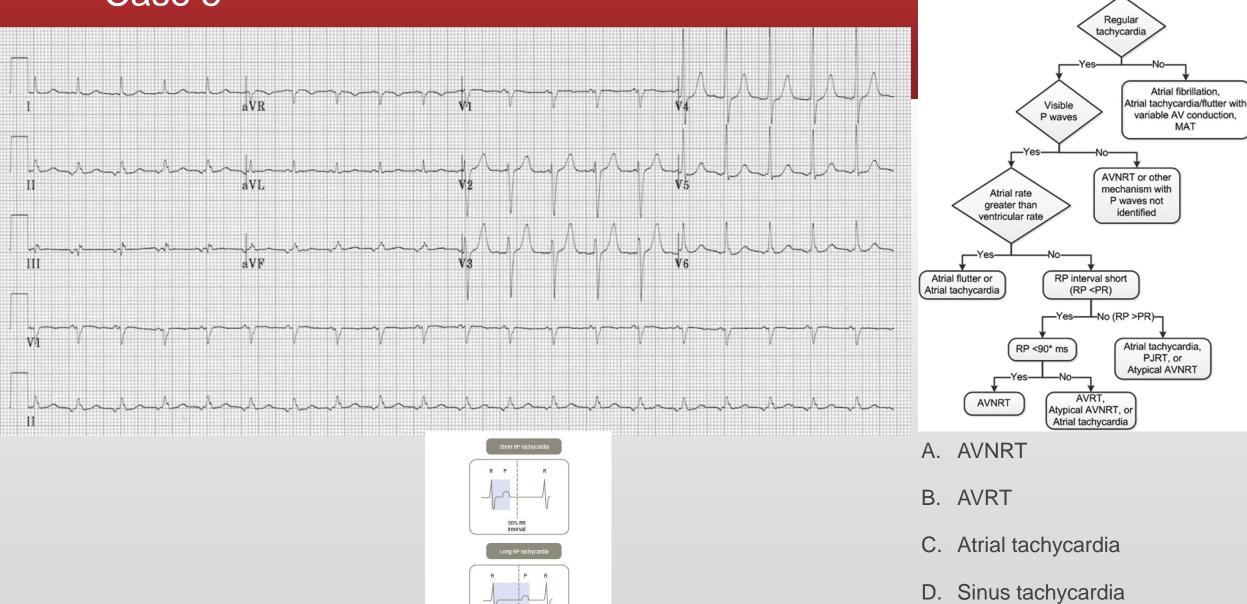


- A. AVNRT
- B. AVRT
- C. Atrial tachycardia
- D. Sinus tachycardia
- E. Atrial flutter





- A. AVNRT
- B. AVRT
- C. Atrial tachycardia
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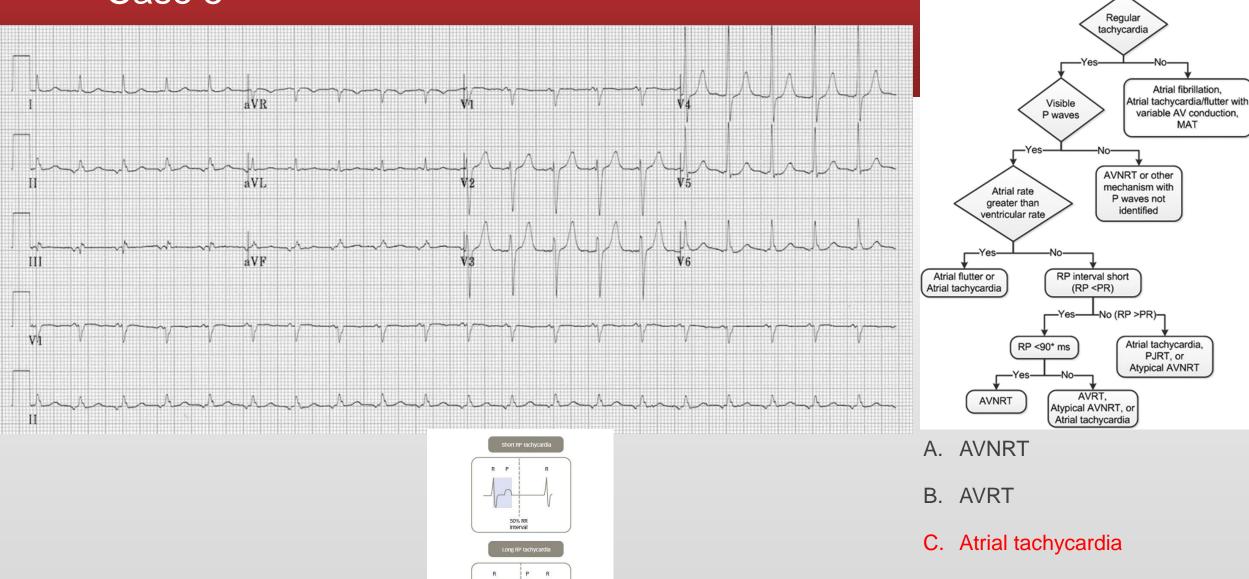


50% RR

interval

E. Atrial flutter

Narrow QRS tachycardia (QRS duration <120 ms)

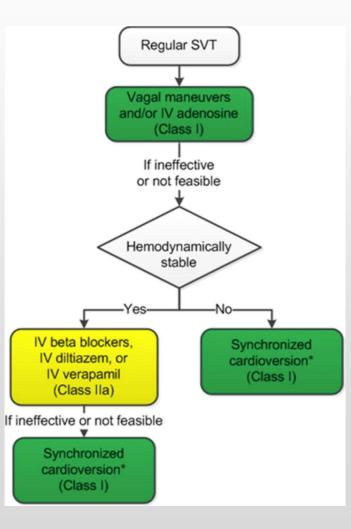


50% RR interval D. Sinus tachycardia

Narrow QRS tachycardia (QRS duration <120 ms)

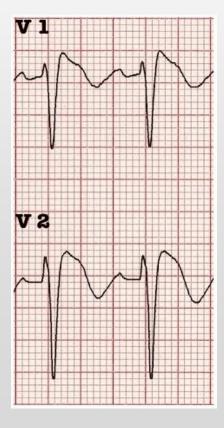
E. Atrial flutter

SVT Management



Brugada Syndrome

- Sodium channelopathy
- Sudden cardiac death in structurally normal heart (other causes include congenital LQTS, catecholaminergic polymorphic VT, idiopathic VT, etc.)



- Type 1
- >2 mm coved ST-elevation in V1-V2 followed by negative T waves



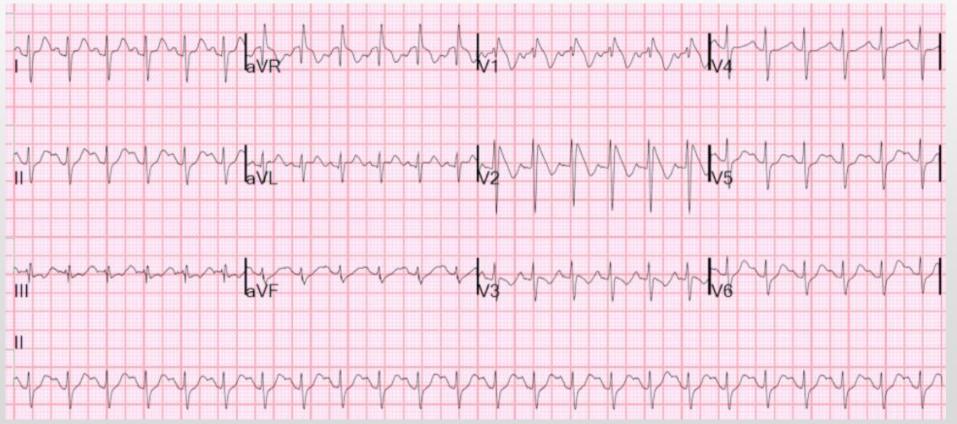
- Type 2
- "Saddle-back" ST-elevation

30M presents after suddenly losing consciousness while playing basketball. His paternal uncle died at age 35 while swimming. Diagnosis?



- A. Brugada type 1
- B. Brugada type 2
- C. STEMI
- D. Atrial fibrillation w/ rapid ventricular response
- E. Mobitz 2 AV block

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- A. Brugada type 1
- B. Brugada type 2
- C. STEMI
- D. Atrial fibrillation w/ rapid ventricular response
- E. Mobitz 2 AV block