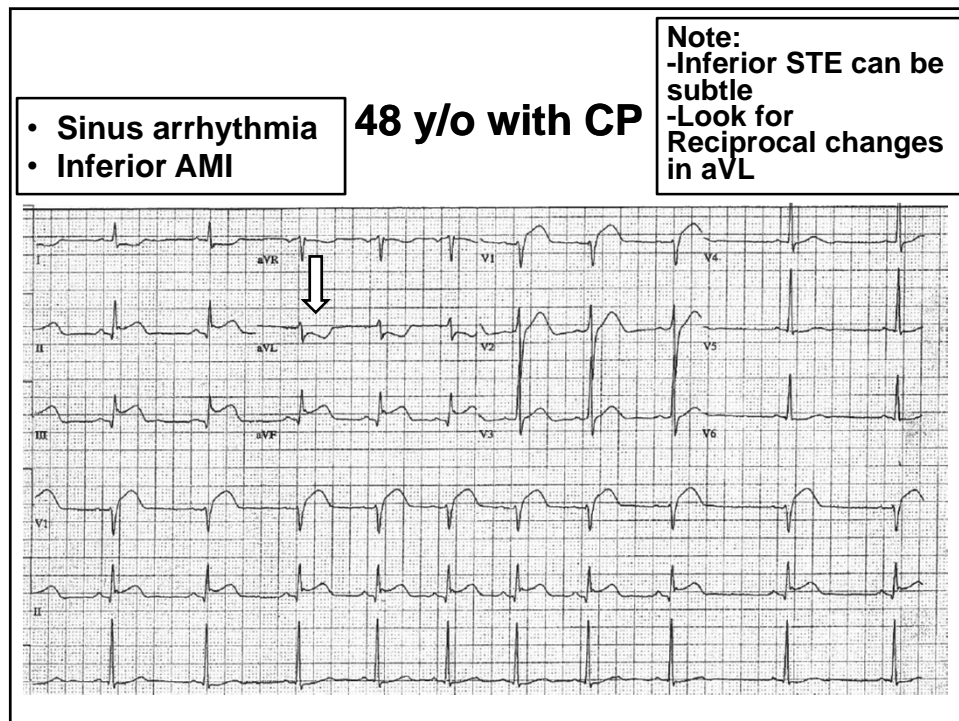
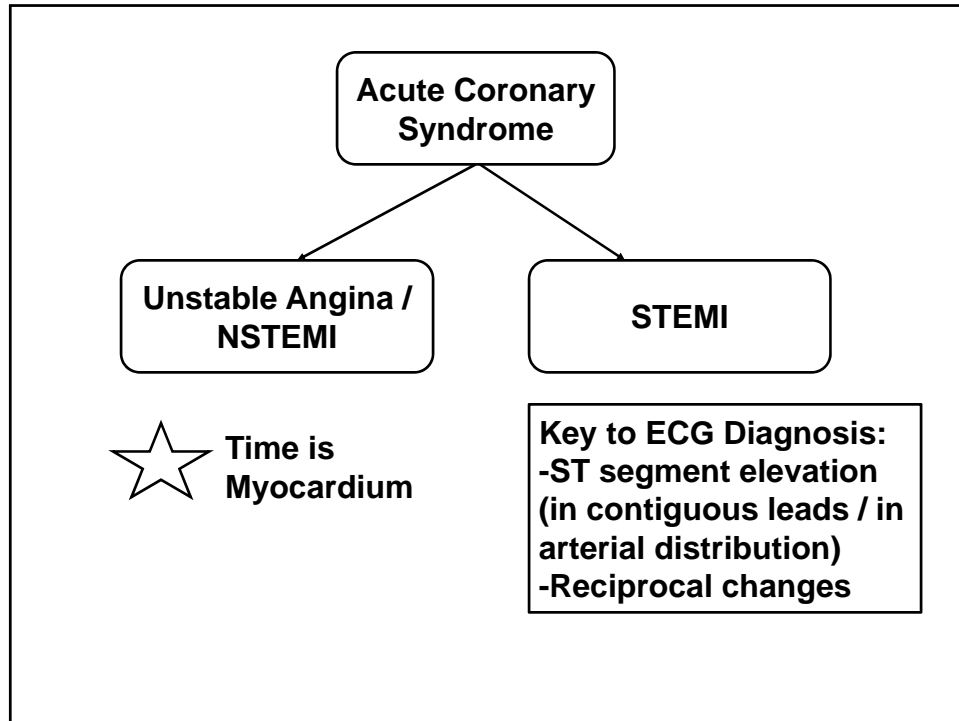


Office ECG Interpretation

**Jason Evanchan, DO
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The Ohio State University Wexner Medical Center**

Outline of topics

- **High risk ischemia**
- **T wave inversions**
- **LBBB / RBBB / RVOT PVC**
- **Atrial activity detection**
- **ECGs in the young adult at risk for SCD**

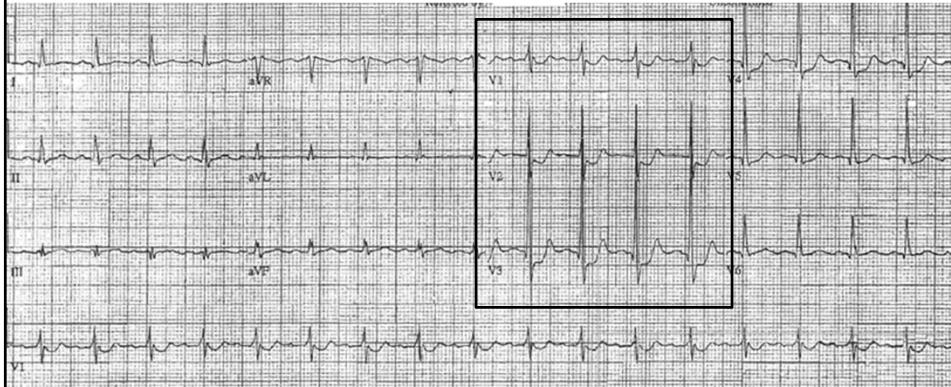


- Sinus tach
- Posterior MI, acute or recent

Key:

- With ST segment depression in V1-V3, consider posterior STEMI
- R wave in these leads can represent posterior Q waves

66 y/o male with CP and diaphoresis



66 y/o male with CP and diaphoresis



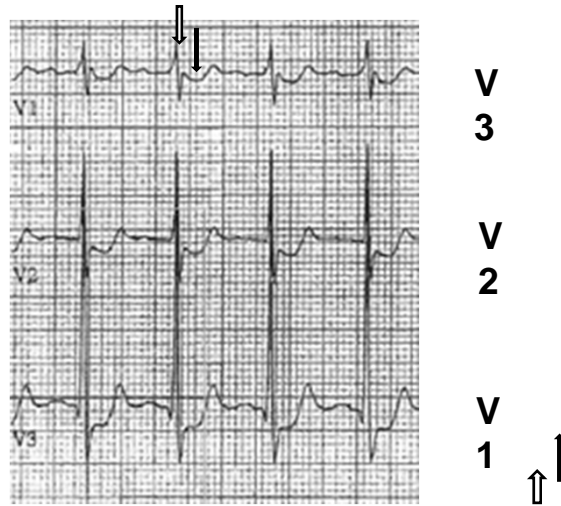
**V
3**

**V
2**

**V
1**



66 y/o male with CP and diaphoresis



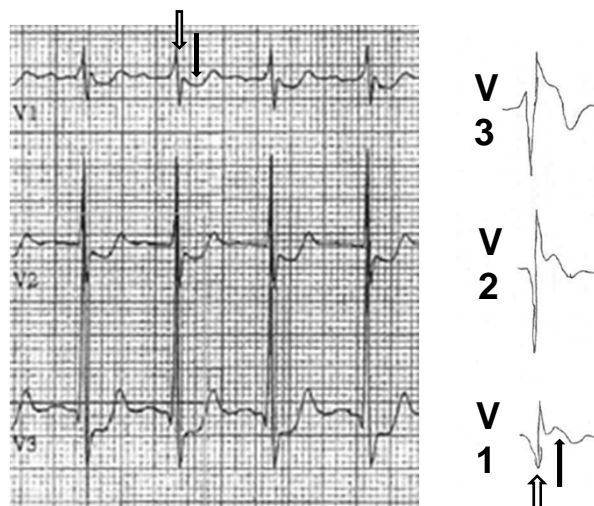
V
3

V
2

V
1



66 y/o male with CP and diaphoresis



V
3

V
2

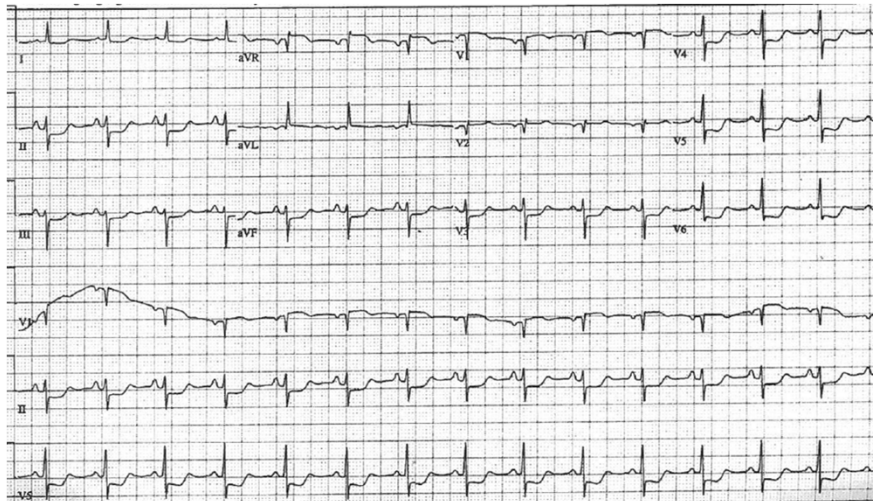
V
1



- SR
- LAFB
- ST changes suggest ischemia

53 y/o DM presents with CP

Key:
-diffuse ST segment Depression with STE in aVR suggests multivessel / LM disease



*Courtesy (with permission) of Eric S Williams, MD from University of Indiana

ST segment elevation

Differential Diagnosis of ST segment elevation

Myocardial injury / infarction from acute vessel occlusion
Prinzmetal angina
Post-myocardial infarction: from ventricular aneurysm
Acute pericarditis
Normal Variant such as early repolarization pattern
Repolarization from LVH and LBBB
Intracranial hemorrhage (typically with deep TW inversion)
Takotsubo's cardiomyopathy
Brugada pattern (RBBB-pattern with STE in precordial leads)
Acute pulmonary embolism (right precordial leads)

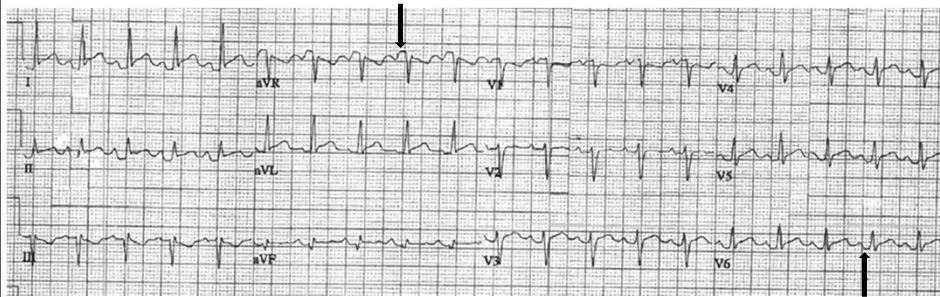
Modified from Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, Tenth Ed. Pg 145

- Sinus Tachycardia
- Acute Pericarditis

Key:

- Diffuse ST segment elevation
- No reciprocal changes
- PR depression (PRE in aVR)

42 y/o with chest pain



T wave inversion

- Normal ECG

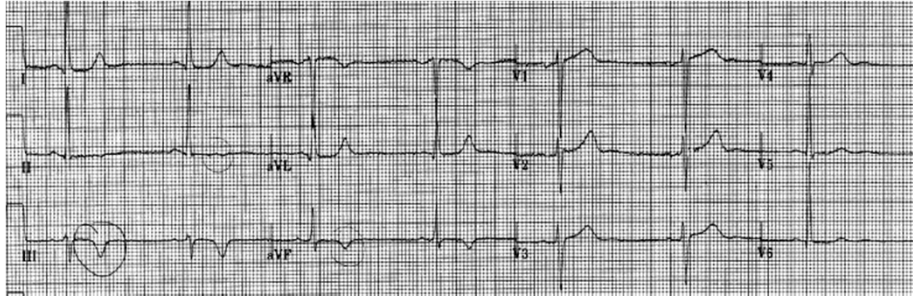
Key:

- Normally T wave is inverted in V1, aVR, and often III
- If upright in V1 can be sign of ischemia
- Juvenile T waves: inverted V1-V3



- Sinus bradycardia
- Inferior TWI c/w ischemic

61 y/o with CP and elevated trop

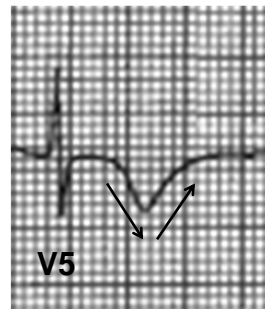


T wave inversion

ST-T wave abnormality secondary to....



Left ventricular hypertrophy



Ischemia

-SR
-LVH
-ST changes
secondary to LVH
-LA abnormality

LVH

Key:
-DX based on voltage
criteria of QRS
-Supported by other
characterizations (LAE,
LAD, secondary ST-T
wave abnormalities,
prolonged intrinsicoid
defection)



LVH

Key:
-Sensitivity <50%,
specificity can be >85%
-Limitations include young
age, body habitus

	Criteria
Cornell criteria	S in V3 + R in aVL \geq 28 mm (men) S in V3 + R in aVL \geq 20 mm (women)
Sokolow-Lyon criteria	S in V1 + R in V5 or V6 $>$ 35 mm R in aVL $>$ 11 mm
Romhilt-Estes point system 4 points = "probable" 5 points = "definite"	Any limb lead R wave or S wave $>$ 20 mm (3 points) or S in V1 or S in V2 \geq 30 mm (3 points) or R in V5 or V6 \geq 30 mm (3 points) ST-T wave abnormalities (not on dig) (3 points) LA abnormalities (3 points) LAD \geq 30 degrees (2 points) QRS duration \geq 90 msec (1 point) Intrinsicoid defection in V5 or V6 \geq 50 msec (1 point)

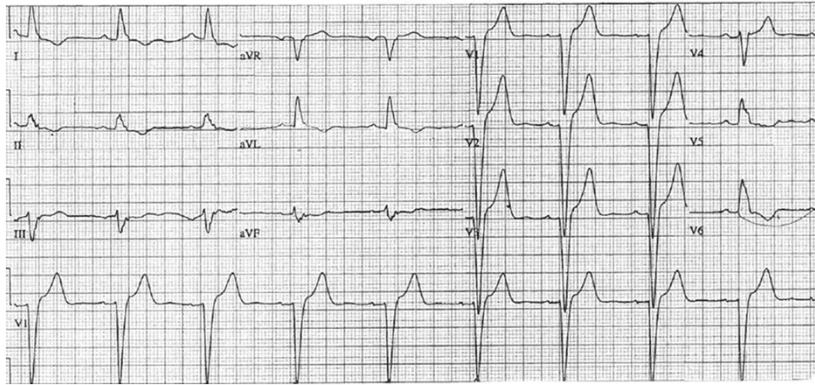
Modified from Braunwald's Heart Disease: A Textbook of Cardiovascular
Medicine, Tenth Ed. Pg 129

**-sinus bradycardia
-LBBB**

49 y/o CAD history

Keys to diagnosing LBB:

- QRS > 120 ms
- Broad, notched or slurred R wave in I, aVL, V6. Deep S wave V1, V2
- Absence of septal q waves in I, V5, V6 prolonged intrinsicoid deflection)
- secondary ST / T wave abnormalities
- typically LAD



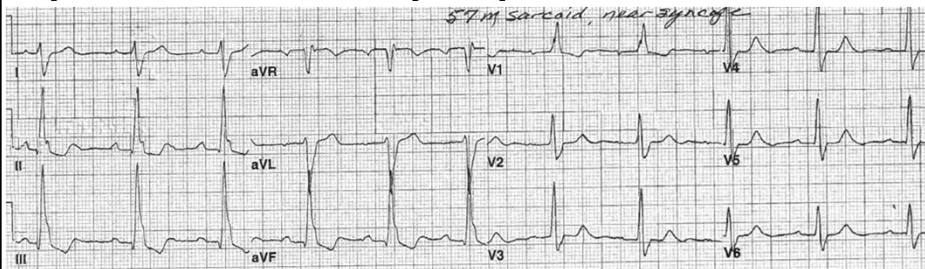
LBBB: additional notes

- ~1% of general population
- -Following AMI, myocarditis (sarcoidosis)
- -Functional / rate-related (long-short)
- Prognosis:
 - depends on type / severity of any concurrent underlying heart disease / other conduction disease
 - Independent predictor of all-cause mortality in pts with CAD, after MI, with congestive heart failure
- Challenging in pts with chest pain
- Should lead to evaluation of HTN, CAD, CM
- CRT if EF <35%
- Abnormalities in coronary blood flow
 - Vasodilator stress

-SR with 1st degree AV block
 -RBBB
 -Left posterior fascicular block

Keys to diagnosing LBBB:
 -QRS > 120 ms
 -rsR' pattern V1 and V2 (R' taller than r)
 -Wide, slurred S wave in I, V6
 -typically normal axis
 -If axis deviation consider LAFB / LPFB

57 y/o with sarcoidosis, presents with near syncope



RBBB: additional notes

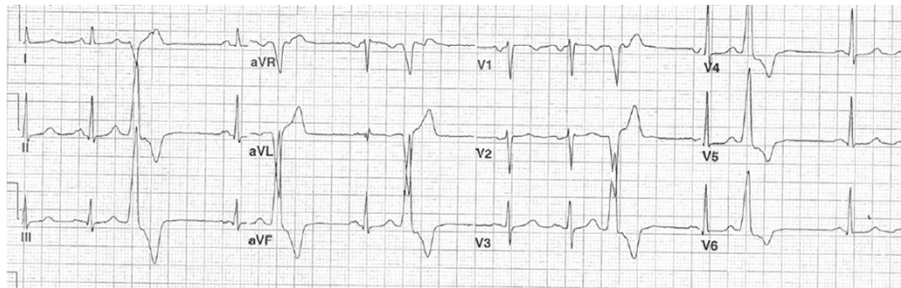
- Can be associated with structural heart disease (cor pulmonale, PE, myocarditis, HTN, CHD)
- Does not interfere with DX of MI b/c the initial 0.04 sec forces are normal
- Can exercise with stress testing
- Prognosis tied to underlying heart disease (excellent with structurally normal heart)
- mimickers such paced rhythm, Brugada

- SR
- Frequent monomorphic PVCs

Keys to RVOT tachycardia / PVCs

- PVCs / VT in left bundle morphology, inferior axis, with transition V2-V3
- Typically structurally normal heart
- Can be amenable to ablation

56 y/o with palpitations

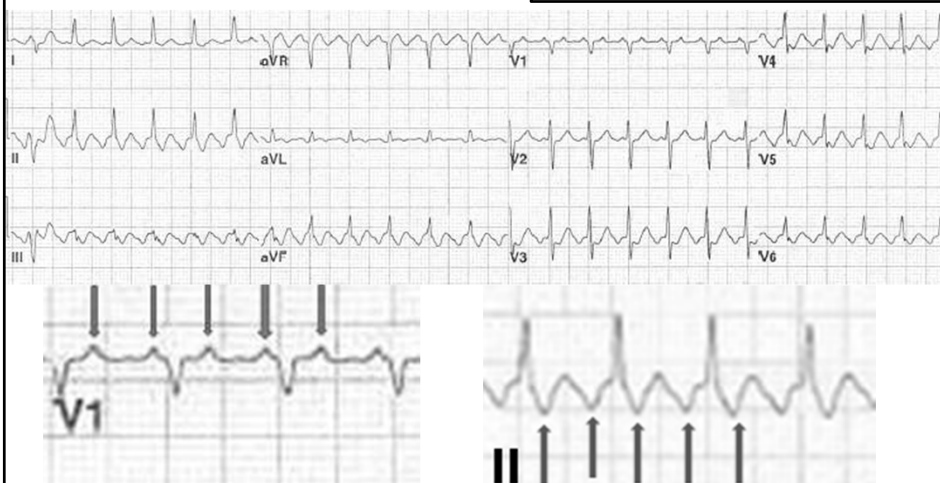


- A flutter
- AV block 2:1

Key:

- Suspect atrial flutter when the ventricular rate is ~150 bpm
- Search for P waves (hidden in ST-segment / T waves)
- Flutter waves obscure ST segment

74 y/o with palps

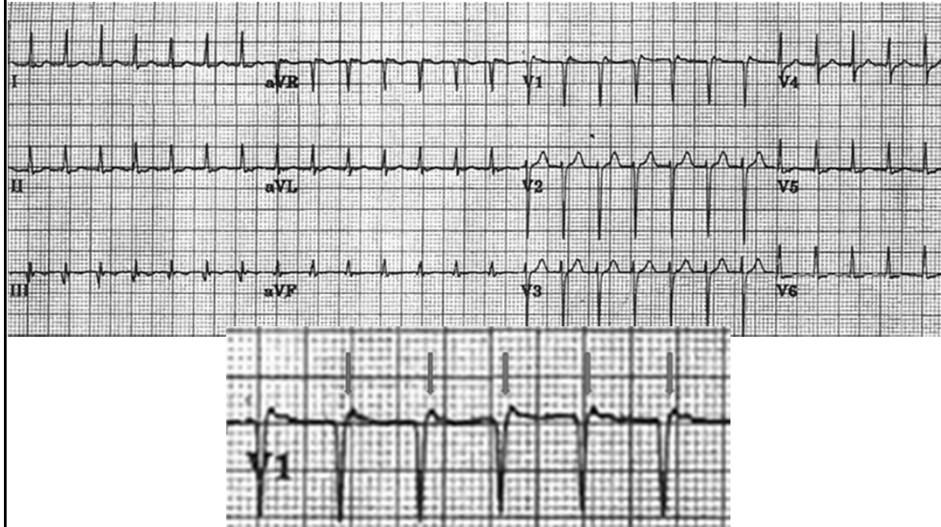


-SVT (likely AVNRT)

Key:

- Find the P wave (compare to previous ECG for r')
- Assess how close the P wave is to the QRS complex (short RP tach)

21 y/o women

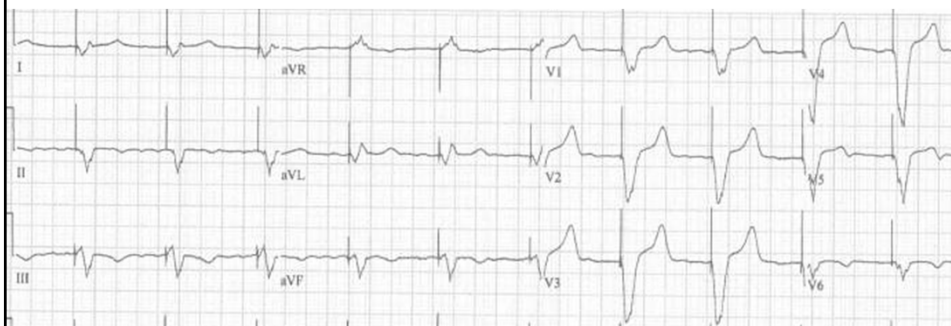


**-Ventricular paced rhythm
-Atrial fibrillation**

Key:

- In patients with ventricular paced rhythm, look for atrial activity
- The "computer" often "misses" atrial fibrillation in this setting

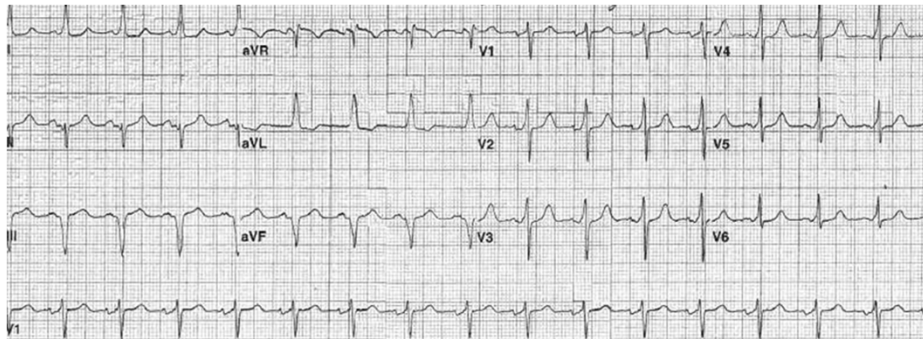
72 y/o, NICM, ICD



-WPW pattern
-Sinus rhythm

Key:
-Delta wave
-Short PR interval
-Can mimic MI

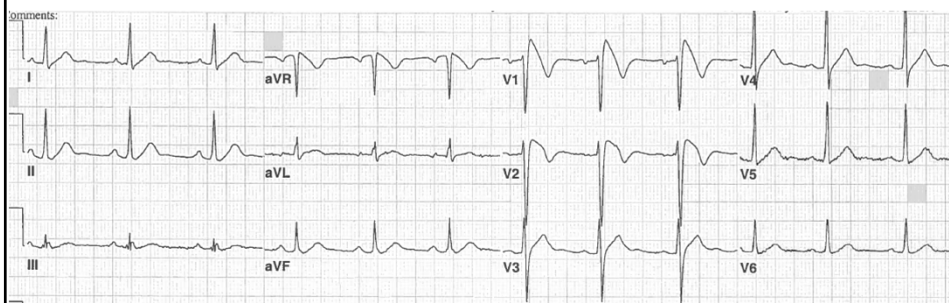
**34 y/o male;
insurance exam**



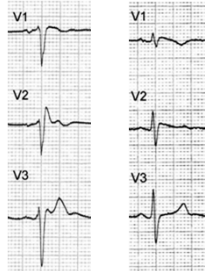
-Sinus rhythm
-Brugada Pattern (type I)

Key:
-RBBB with STE in the
right precordium
-Can have variable
morphologies
-Risk for VF and SCD

22 y/o with syncope



Type 2 Type 3



Author: Napolitano C, Priori SG.
Brugada syndrome. Orphaned J
Rare Dis. 2006 Sep 14;1:35
(CC BY 2.0)

Wilde AA, et al. Circ
2002; 106: 2514-19

Brugada Syndrome (BrS). Expert Consensus Recommendations on BrS Diagnosis

1. **BrS is diagnosed** in pts with ST segment elevation with **type 1 morphology** ≥ 2 mm in ≥ 1 lead among the right precordial leads (V1, V2), positioned in the 2nd, 3rd, or 4th intercostal space occurring either **spontaneously or after provocative drug test** with IV administration of Class I antiarrhythmic drugs.

2. **BrS is diagnosed** in pts with **type 2 or type 3** ST-segment elevation in ≥ 1 lead among the right precordial leads (V1, V2), positioned in the 2nd, 3rd, or 4th intercostal space when a **provocative drug test with IV administration of Class I antiarrhythmic drugs induces a type I ECG morphology**

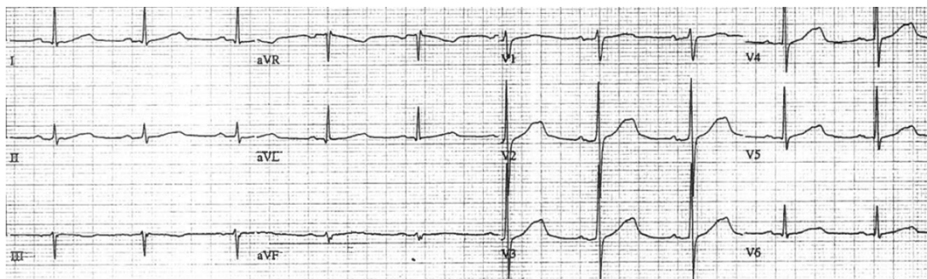
2013 HRS / EHRA/APHS Expert Consensus Statement on the Diagnosis and Management of Patients with Inherited Primary Arrhythmia Syndromes

-Sinus rhythm
-Prolonged QT

37 y/o with congenital
QT prolongation

Key:

- Measure longest QT (well seen)
- Assess for secondary causes (medications / electrolytes)
- QTc = QT interval \div sq rt RR interval



*Courtesy (with permission) of Eric S Williams, MD from University of Indiana

Long QT Syndrome (LQTS) Expert Consensus Recommendations on LQTS Diagnosis

1. LQTS is diagnosed:
 - a. In the presence of a LQTS risk score of ≥ 3.5 in the absence of a secondary cause for QT prolongation *and / or*
 - b. In the presence of an unequivocally pathogenic mutation in one of the LQTS genes or
 - c. In the presence of a QT interval corrected for HR using Bazett's formula (QTc) ≥ 500 msec in repeated 12 lead ECGs, and in the absence of a secondary cause for QT prolongation.
2. LQTS can be diagnosed in the presence of a QTc btw 480-499 msec in repeated 12 lead ECGs in a patient with unexplained syncope in the absence of a secondary cause for QT prolongation and in the absence of a pathogenic mutation

2013 HRS / EHRA/APHRs Expert Consensus Statement on the Diagnosis and Management of Patients with Inherited Primary Arrhythmia Syndromes

1993-2011 LQTS Diagnostic Criteria

Findings	Points
A. QTc (in the absence of medications known to effect these ECG features)	3
≥ 480 msec	2
460-479 msec	1
450-459 msec (in males)	1
B. QTc 4 th min of recovery from exercise	2
C. Torsades de pointes	1
D. T wave alternans	1
E. Notched T wave in 3 leads	0.5
F. Low HR for age	
Clinical History	
A. Syncope	
With stress	2
Without stress	1
B. Congenital Deafness	0.5
Family History	
A. Family members with definite LQTS	1
B. Unexplained SCD below the age of 30 in immediate family member	0.5

Schwartz et al. Circ 88: 782,1993

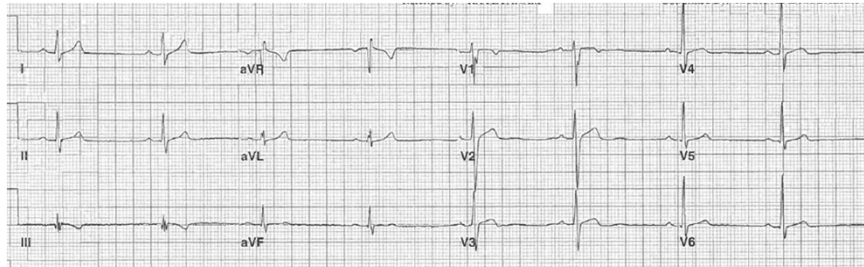
Keating. Circ 85: 1973, 1992

Schwartz et al. Circ 124: 2181-4

**-Sinus rhythm
-Short QT**

**Key:
-Risk for SCD with
structurally normal heart**

18 y/o



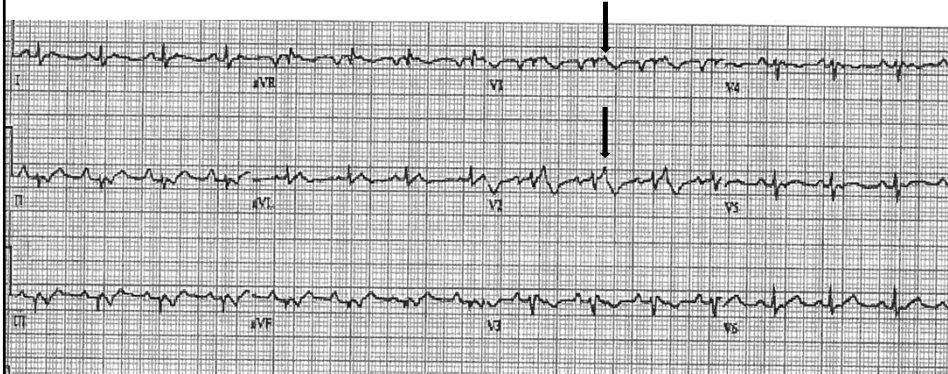
**Short QT Syndrome (SQTS). Expert Consensus
Recommendation on SCQS**

1. SQTS is diagnosed in the presence of a $QTc \leq 330$ msec
2. SQTS can be diagnosed in the presence of a $QTc < 360$ msec and one or more of the following: a pathogenic mutation, family h/o SCD at ≤ 40 , survival of a VT / VF episode in the absence of heart disease

**2013 HRS / EHRA/APHRS Expert
Consensus Statement on the Diagnosis
and Management of Patients with
Inherited Primary Arrhythmia Syndromes**

Concern for ARVC

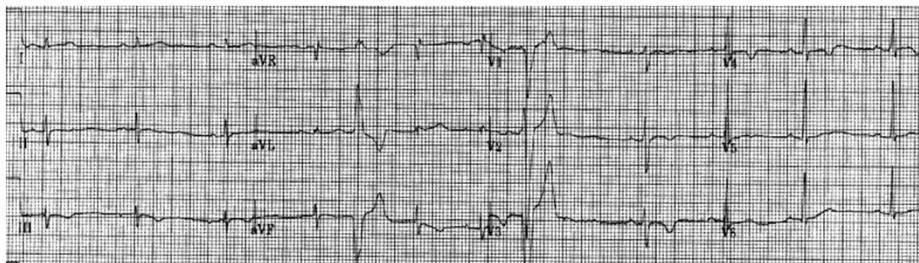
Key for Arrhythmogenic RV cardiomyopathy
 -Task Force on DX of ARVC (combination of echo, MRI, arrhythmias, conduction abnormalities, BX data, Family HX)
 -Epsilon waves (poor sensitivity)
 -TW inversion antero-septal leads
 -Late potential on SAECG
 -PVCs, VT with left bundle morphology



-Sinus rhythm
 -Anterior TW inversion
 -PVC in left bundle morphology

Key for Arrhythmogenic RV cardiomyopathy
 -Task Force on DX of ARVC (combination of echo, MRI, arrhythmias, conduction abnormalities, BX data, Family HX)
 -Epsilon waves (poor sensitivity)
 -TW inversion antero-septal leads
 -Late potential on SAECG
 -PVCs, VT with left bundle morphology

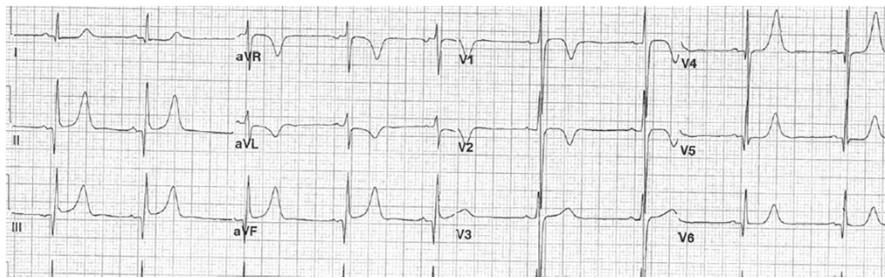
**21 y/o with exercise induced syncope.
 MRI, echo, and ECG c/w ARVC**



Key for HCM

- ECG is rarely normal, but findings are not often specific
- Can have prominent voltages with repolarization
- Prominent Q waves (inferior and lateral leads)
- LAD
- Deeply inverted T waves with apical variant HCM

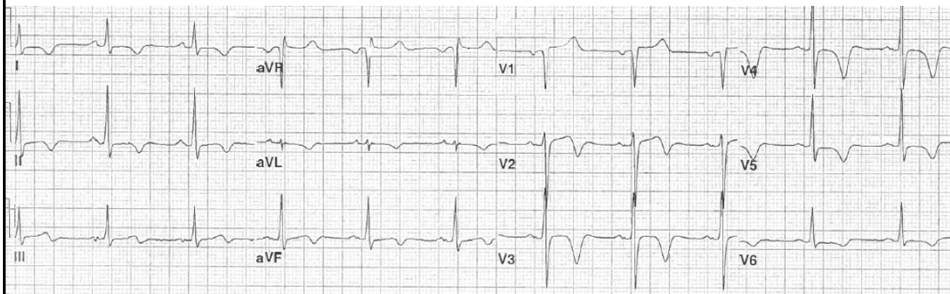
**20 y/o with HOCM. Septal hypertrophy.
Peak LVOT gradient 144 mm Hg**



**51 y/o with syncope.
FHx of SCD.
Evidence of apical
HCM on cardiac MRI**

Key for apical HCM

- Deep, symmetrical, inverted T waves anterolateral leads



Classification of Heart Block

Classification of Heart block	Notes
First Degree AV Block	PR interval > 200 msec. All P waves followed by QRS complexes
2 nd Degree, Mobitz type I (Wenckebach)	Progressive PR prolongation until a P wave is not conducted <i>Note: compare the post non-conducted beat PR interval to the PR interval immediately before</i> Typically at the level of the AV node
2 nd Degree, Mobitz type II	Intermittent or repetitive non-conducted / dropped beats without prior PR lengthening (fixed PR interval) Site of pathology is distal to the AV node
Complete Heart Block	Failure of all P wave to conduct

Thank you!

- jason.evanchan@osumc.edu
- **Special thanks to:**
 - Dr. Rick (Stephen) Schaal
 - Dr. Eric S. Williams