Office ECG Interpretation

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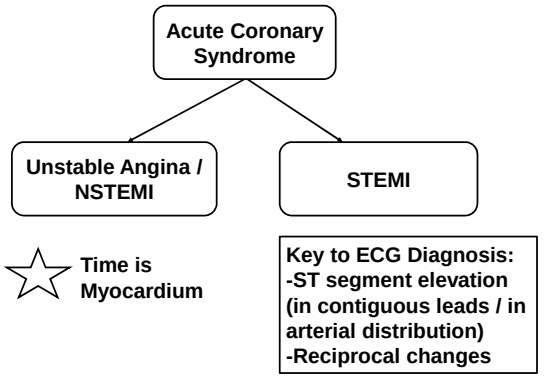
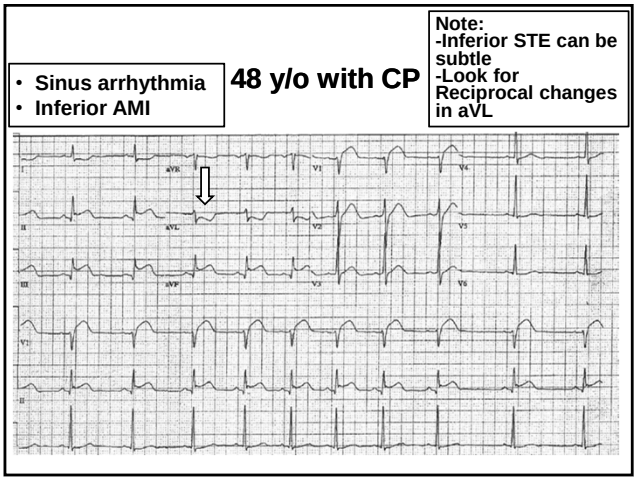
Outline of topics

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

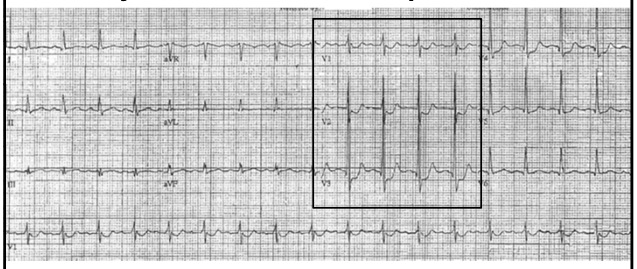




- Sinus tach
- Posterior MI, acute or recent

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



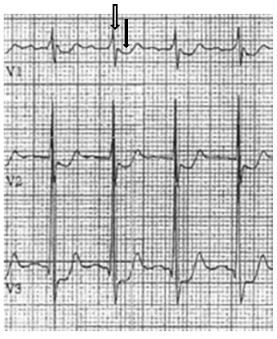
66 y/o male with CP and diaphoresis

V 3

V 2

V 1

66 y/o male with CP and diaphoresis

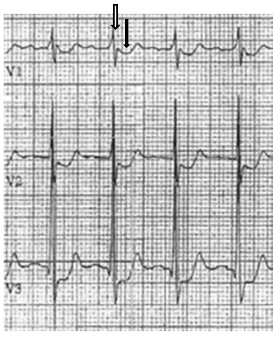


V 3

V 2

V 1

66 y/o male with CP and diaphoresis



V 3

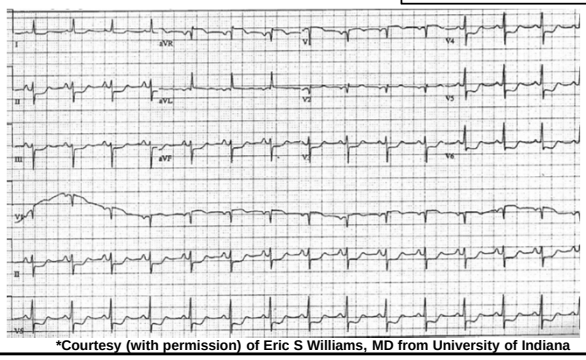
V 2

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

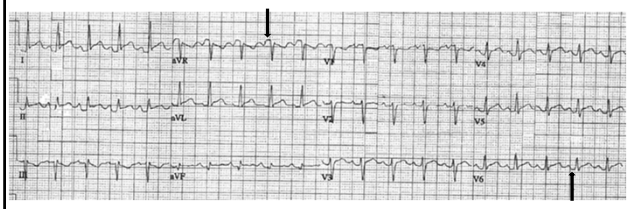
Differential Diagnosis of ST segment elevation
Myocardial injury / infarction from acute vessel occlusion
Prinzmetal angina
Post-myocardial infarction: from venticular aneurysm
Acute pericarditis
Normal Variant such as early repolarization pattern
Repolarization from LVH and LBBB
Intracranial hemorrhage (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 Tachycaria**
- **Acute Pericarditis**

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

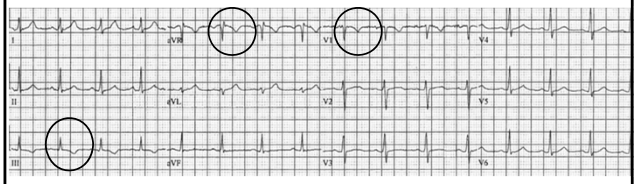
42 y/o with chest pain



T wave inversion

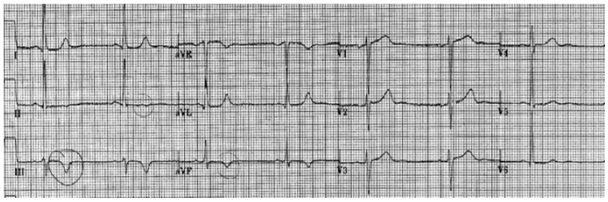
- **Normal ECG**

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



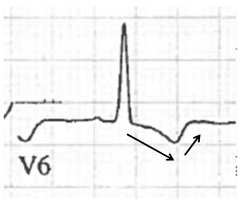
- **Sinus bradycardia**
- **Inferior TWI c/w ischemic**

61 y/o with CP and elevated trop

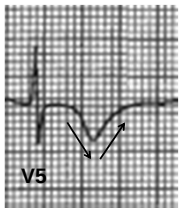


T wave inversion

ST-T wave abnormality secondary to….



Left ventricular hypertrophy

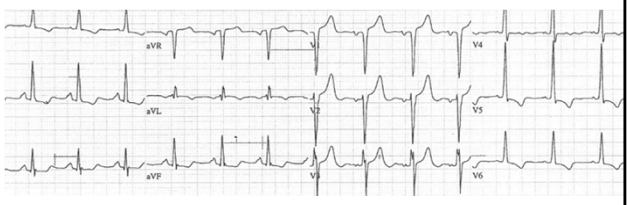


Ischemia

-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 abnromalities, prolonged intrinsicoid defection)



LVH

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

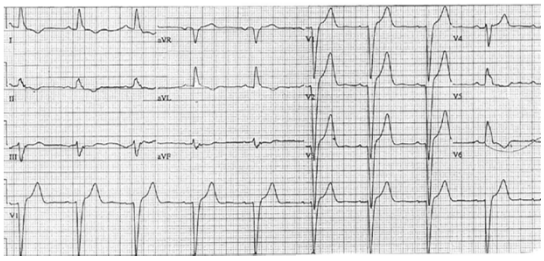
	Criteria
Cornell criteria	S in V_3 + R in aVL ≥ 28 mm (men) S in V_3 + R in aVL ≥ 20 mm (women)
Sokolow-Lyon criteria	S in V_1 + R in V_5 or V_6 > 35 mm R in aVL > 11 mm
Romhilt-Estes point system *4 points = "probable"* *5 points = "definite"*	Any limb lead R wave or S wave > 20 mm (3 points) *or* S in V_1 or S in V_2 ≥ 30 mm (3 points) *or* R in V_5 or V_6 ≥ 30 mm (3 points) ST-T wave abnormalities (not on dig) (3 points) LA abnormalities (3 points) LAD ≥ 30 degrees (2 points) QRS duration ≥ 90 msec (1 point) Intrinsicoid defection in V5 or V6 ≥ 50 msec (1 point)

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

-sinus bradycardia
-LBBB

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 defection)
-secondary ST / T wave abnormalities
-typically LAD

49 y/o CAD history



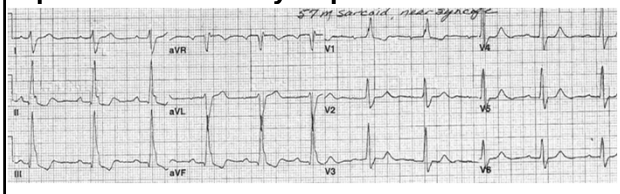
LBBB: additional notes

- ~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 then 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



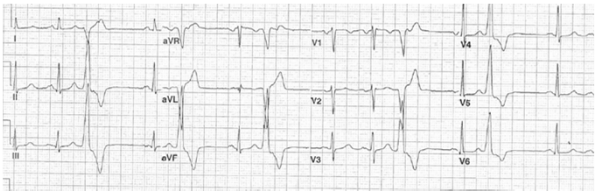
RBBB: additional notes

- 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

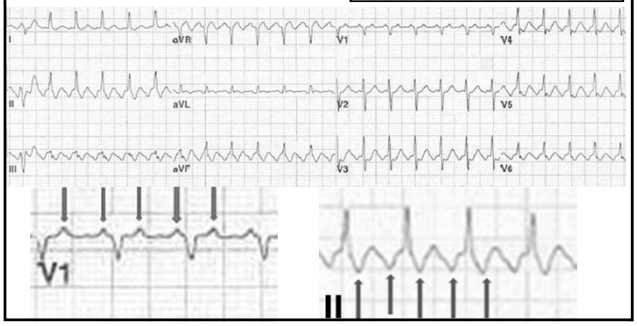
56 y/o with palpitations



-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

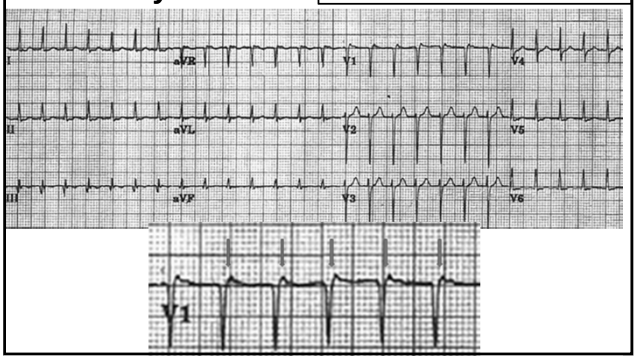
74 y/o with palps



-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)

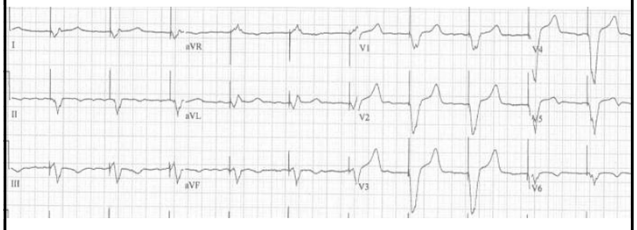
21 y/o women



-Ventricular paced rhythm
-Atrial fibrillation

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

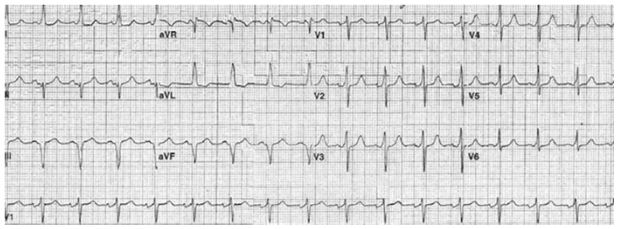
72 y/o, NICM, ICD



-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

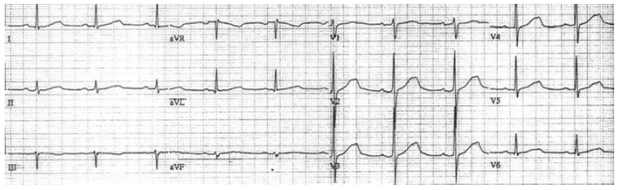
Brugada Syndrome (BrS). Expert Consensus Recommendations on BrS Diagnosis
1. **BrS is diagnosed** in pts with ST segment elevation with **type 1** morphology ≥ 2 mm in ≥1 lead among the right precordial leads (V1, V2), positioned in the 2nd, 3rd, or 4th intercostal space occurring either **spontaneously 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/APHRS Expert Consensus Statement on the Diagnosis and Management of Patients with Inherited Primary Arrhythmia Syndromes

-Sinus rhythm
-Prolonged QT

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

37 y/o with congenital QT prolongation



*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

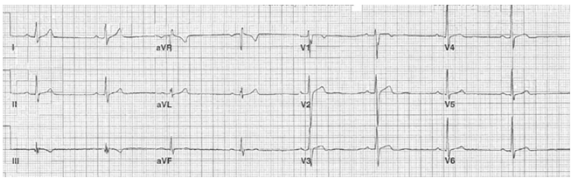
Findings	Points
A. QTc (in the absence of medications known to effect these ECG features)	
≥480 msec	3
460-479 msec	2
450-459 msec (in males)	1
B. QTc 4th min of recovery from exercise	1
C. Torsades de pointes	2
D. T wave alternans	1
E. Notched T wave in 3 leads	1
F. Low HR for age	0.5
Clinical History	
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

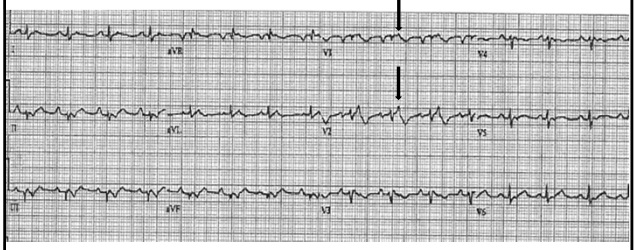
1. SQTS is diagnosed in the presence of a QTc ≤ 330 msec

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

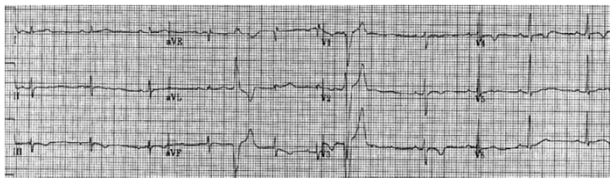
Key for Arrhythmogenic RV cardiomyopathy
-Task Force on DX of ARVC (combination of echo, MRI, arrhythmias, conduction abnormalities, BX data, Family HX)
-Epsilon waves (poor sensitivity)
-TW inversion anteroseptal leads
-Late potential on SAECG
-PVCs, VT with left bundle morphology



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

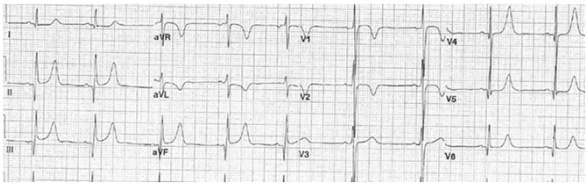
Key for Arrhythmogenic RV cardiomyopathy
-Task Force on DX of ARVC (combination of echo, MRI, arrhythmias, conduction abnormalities, BX data, Family HX)
-Epsilon waves (poor sensitivity)
-TW inversion anteroseptal leads
-Late potential on SAECG
-PVCs, VT with left bundle morphology

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



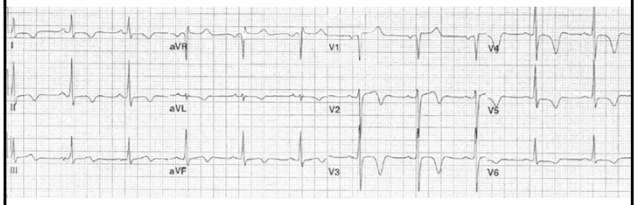
Key for HCM
-ECG is rarely normal, but findings are not often specific
-Can have prominent voltages with repolarization
-Prominent Q waves (inferior and lateral leads)
-LAD
-Deeply inverted T waves with apical variant HCM

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
2nd Degree, Mobitz type I (Wenckebach)	Progressive PR prolongation until a P wave is not conducted *Note: compare the post non-conducted beat PR interval to the PR interval immediately before* Typically at the level of the AV node
2nd 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!

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- **Special thanks to:**
  - **Dr. Rick (Stephen) Schaal**
  - **Dr. Eric S. Williams**