ECG Findings You Don’t Want To Miss

pmiCME Updates
San Francisco, California

October 10, 2012
11:00 AM – 12:15 PM

Content Collaborator:
Session 3 ECG Findings You Don’t Want To Miss

Learning Objective
1. Interpret ECG findings that are commonly encountered in the office setting and that should not be overlooked.

Faculty

Elyse Foster, MD, FACC, FAHA
Director, Echocardiography Laboratory
Professor of Clinical Medicine
Araxe Vilensky Endowed Chair in Medicine
Department of Medicine, Cardiology
University of California, San Francisco
San Francisco, California

Dr. Elyse Foster is director of the University of California, San Francisco Adult Echocardiography Laboratory and Adult Congenital Heart Disease Service. She specializes in the assessment of cardiac function using echocardiography, including exercise and pharmacologic stress testing for ischemia detection. She also has a special interest in adult congenital heart conditions and valvular disease.

Dr Foster earned a medical degree at Tufts University School of Medicine. She completed a residency in internal medicine at Boston Medical Center, where she also completed a fellowship in cardiovascular diseases. Dr Foster is on the executive committee of the International Society of Adult Congenital Heart Disease and is on the women in cardiology committee for the American College of Cardiology. She is board certified in both internal medicine and cardiovascular disease.

Faculty Financial Disclosure Statement
The presenting faculty reports the following:

Dr Foster receives support from Evalve, Inc. and Abbott.

Suggested Reading List


ECG Findings You Don’t Want to Miss

Speaker:
Elyse Foster, MD, FACC, FAHA

Learning Objective

• Recognize ECG changes associated with easily missed or misdiagnosed clinical conditions
  • Atypical presentations of Acute Coronary Syndromes
  • Mimickers of ACS
  • EKG findings in Right Heart Disease
  • Miscellaneous diagnoses that shouldn’t be missed

Pre-test Audience Response Question: ?
Which of the following is contraindicated for the patient with Wellens’ syndrome?

1. Cardiac catheterization
2. Antiplatelet therapy
3. Echocardiogram
4. Exercise stress testing

Pre-test Audience Response Question: ?
True or false: The absence of ECG abnormalities has strong predictive value in ruling out acute pulmonary embolus.

1. True
2. False

Presenter Disclosure Information

The following relationships exist related to this presentation:
• Dr Foster receives support from Evalve, Inc. and Abbott.

Off-Label/Investigational Discussion:
In accordance with pmCME policy, faculty have been asked to disclose discussion of unlabeled or unapproved use(s) of drugs or devices during the course of their presentations.
Correct lead placement essential for accurate EKG diagnosis

- Extremities to derive limb leads
  - Upper arms and lower legs – although proximal vs. distal placement not well established
  - EKG with limb leads placed on chest, which can alter morphology

- Precordial leads
  - Placement in the 4th and 5th intercostal spaces is essential

Electrode Sites for Recording Precordial ST-Segment Elevation

Ancillary Precordial Leads

- V3R, V4R: RV ischemia/infarction
- V7, V8, V9: True Posterior Infarction

Adapted from: http://www.aacn.org

Acute Coronary Syndromes: EKG Stages After Coronary Artery Occlusion

- ST changes should be apparent in 2 or more anatomically contiguous leads
- Cabrera EKG format
  - I, aVL, aVR
  - II, III, aVF

Threshold values for ST segment elevation

- Clinical context should always be considered
- Men ≥ 40 yrs: J point elevation
  - ≥ 0.2 mV (2 mm) in V2 and V3
  - ≥ 0.1 mV (1 mm) in all other leads
- Men <40 yrs: J-point elevation
  - ≥ 0.25 mV (2.5 mm) in V2 and V3
- Women: J-point elevation
  - ≥ 0.15 mV (1.5 mm) in V2 and V3
  - > 0.1 mV (1 mm) in all other leads.
Ancillary Leads: RV infarction and Posterior Infarction

- Men and women:
  - J-point elevation
    - $\geq 0.05$ mV (0.5 mm) in V3R and V4R, except for males $< 30$ yrs
    - $\geq 0.05$ mV (0.5 mm) in V7 through V9
  - J-point depression
    - $\geq -0.05$ mV (-0.5 mm) in V2 and V3
    - $\geq -0.1$ mV (-1 mm) in all other leads.

Positive Predictive Accuracy and Sensitivity of Several ECG Parameters to Detect Myocardial Infarction

<table>
<thead>
<tr>
<th>ECG Finding Patients who had MIs (PPV) (%)</th>
<th>MI Patients (Sensitivity) (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. $&gt; 1$ mm ST-segment elevation or Q waves in $\geq 2$ leads</td>
<td>461/605 (76) 461/1024 (45)</td>
</tr>
<tr>
<td>2. New ischemia or strain $&gt; 1$ mm, ST-segment depression in $\geq 2$ leads</td>
<td>203/531 (38) 203/1024 (20)</td>
</tr>
<tr>
<td>3. Other new ST-segment or T wave changes of new ischemia or strain</td>
<td>147/693 (21) 147/1024 (14)</td>
</tr>
<tr>
<td>4. Old infarction, ischemia or strain</td>
<td>50/644 (8) 50/1024 (5)</td>
</tr>
<tr>
<td>5. Other new or old abnormality</td>
<td>56/1147 (5) 56/1024 (5)</td>
</tr>
<tr>
<td>6. Nonspecific ST-T changes only</td>
<td>72/1433 (5) 72/1024 (7)</td>
</tr>
<tr>
<td>7. Normal</td>
<td>35/2062 (2) 35/1024 (3.4)</td>
</tr>
</tbody>
</table>

Suspected ACS with initial Non-diagnostic ECG

- Should be repeated q20 minutes especially in presence of on-going pain
- Increases sensitivity of ECG

ECG Findings: Acute Coronary Syndromes (ACS) and ACS Mimickers

Case 1: 62 yo male with onset of chest pain during “Dragon Boat Race” practice

- One prior episode of pain 5 days earlier that awoke him
- Became extremely weak with substernal (SS) chest discomfort 10 mins after practice
- Unrelieved with sips of water, friend called 911

ECG on arrival to ER
Notable findings on this ECG include:

1. ST-segment elevations in anterior precordial leads (V1-V4)
2. ST-segment elevations in inferior leads (II, III, aVF)
3. Peaked T wave in anterior precordial leads
4. ST depressions in II, III, aVF

EKG on arrival to ER

Which of the following is suggested by these ECG findings?

1. left anterior descending coronary occlusion
2. right coronary artery occlusion
3. left circumflex coronary occlusion
4. normal coronary anatomy

Cath lab activated...

- Extensive left anterior descending artery (LAD) thrombus with total LAD occlusion
- Extracted thrombus and drug eluting stent (DES) placed
- Developed accelerated idioventricular rhythm (AIVR)

Which of the following is the preferred management for AIVR in this scenario?

1. Immediate electrical cardioversion
2. Observation and monitoring
3. Amiodarone or procainamide
4. Beta blocker or calcium channel blocker
5. Ablation
ECG Pre-Discharge

ECG changes in Acute MI: Key Points

- ECG evidence of ischemia or infarction
- evolves over hours, days and weeks
- varies in sensitivity and specificity
- must be considered in clinical context
- Corroborate with presenting symptoms and biomarkers to confirm diagnosis

Case 2: 42 yo man c/o episodes of stuttering chest discomfort over past week

- Became prolonged (30 min) after softball game on day of presentation, some increase in discomfort during inspiration
- Cardiac risk factors: dyslipidemia
- Physical exam:
  - Essentially normal
  - No rub or S3
- After oxygen and aspirin in ambulance, pain free upon ER arrival at 9:50 pm

Which of the following ECG changes in this patient warrant activation of the cath lab?

1. 1 – 1.5 mm J point elevation V2, V3, V4
2. Axis + 110
3. Poor R wave progression
4. S wave V6
5. None of the above

Key Point

- Pain free
- Does not meet threshold for elevation
  - Men ≥ 40 yrs: J point elevation
    - ≥ 0.2 mV (2 mm) in V2 and V3
    - ≥ 0.1 mV (1 mm) in all other leads
EKG #1 - 10 pm
No pain

EKG #2: 1:30 am
No recurrent pain, cardiac enzymes WNL

Pt sent to cath lab . . .
- 99% proximal LAD occlusion
- Bare metal stent (BMS) placed
- Discharged on:
  - aspirin (ASA)
  - clopidogrel
  - statin
  - metoprolol

Discharge EKG - Day 3
Acute Coronary Syndromes: Wellens’ Syndrome

- LAD - T wave syndrome
  - Proximal LAD stenosis
  - Deep symmetric T waves in multiple precordial leads (V1 - V4)
  - No ST elevations
  - Often associated with reversible wall motion abnormality


Wellens’ Syndrome: Key Points

- History of chest pain
  - present in 14% - 18% of patients admitted for unstable angina

- ECG pattern observed in pain-free state; it may change or appear more normal in presence of pain (ie, “pseudonormalization”)


Wellens’ Syndrome: Key Points

- Characteristic ECG findings:
  - Little/no cardiac enzyme elevation
  - No pathologic precordial Q waves
  - Little/no ST-segment elevation
  - No loss of precordial R waves
  - Symmetric deeply inverted T waves in leads V2 and V3
  - Less commonly, biphasic T waves in leads V2 and V3


Wellens’ Syndrome: Key Points

- Manage aggressively
  - Approx 75% of patients who are not revascularized (coronary artery bypass surgery (CABG) or angioplasty) will develop extensive anterior wall infarction within days

- Patients typically have at least a 50% stenosis of the LAD on angiography

- Stress testing contraindicated; may induce left anterior wall MI


Acute Left Main Thrombosis

- STE aVR
  - Suggestive of left main disease or equivalent with diffuse subendocardial ischemia
  - May be associated with STE V1, reciprocal STD aVF, V2, V4

Case 3: 65 yo man with chest pain for 1 hour

- H/O HTN and dyslipidemia
- Recent complaints of dyspnea on exertion (DOE), scheduled for exercise treadmill test (ETT)
- Sudden onset nausea, vomiting, chest discomfort and extreme weakness
- Called 911
- In ER, BP 70/50 mm Hg, HR 50, cool extremities, poor mentation, low urine output, lungs clear with marked JVD

Most likely cause of ECG findings: ?

1. Acute occlusion of the RCA
2. Acute occlusion of the left circumflex coronary artery
3. Acute pericarditis
4. None of the above

Dx: Inferior infarction with RV involvement

- Proximal RCA occlusion
  - ST elevation in III > II
  - ST depression in I, aVL
  - Acute RV injury
    - ST elevation in V1, V3R
- Acute LCx occlusion
  - STE in II = III
  - ST elevation I, aVL
  - ST depression V1 – V3

Right-sided leads
Patient sent to cath lab

- Total proximal right coronary artery (RCA) occlusion
- 60% left main lesion
- Received bare metal stent to RCA
- Refused surgery

Right Ventricular Infarction Complicating Acute Inferior Myocardial Infarction

Diagnosis of Acute MI in Presence of left bundle branch block (LBBB)

Key Points

- RBBB usually does not obscure diagnosis of acute ischemia or infarction
- LBBB:
  - New or Uncertain LBBB in setting of acute chest pain warrants STEMI management
  - Pre-existing
    - Sgarbossa score \( \geq 3 \)
      - High specificity – 90%
      - Low sensitivity – 35%

Case 4: 53 yo man with chest pain x 13 hours

- Midsternal, occasional pleuritic component
- PMH: mild renal insufficiency, monoclonal gammopathy
- PE: BP 155/90 mm Hg, no murmurs or rubs

Notable Findings on this ECG include:

1. Left axis deviation
2. Hyperacute T waves V2, V3, V4
3. Diffuse J point elevation I, aVL, V2 – V4
4. PR segment depression II, III, aVF
Case 4: 53 yo man with chest pain x 13 hours

Patient developed nausea and near syncope

- Rushed to cath lab
- Clean coronaries
- Labs: troponin normal, BNP normal, ESR 35
- Treated with ibuprofen 800 mg 3x/day

Dx: Pericarditis
Ibuprofen tapered over 3 weeks but pain recurred

Notable Findings on this ECG include:

1. Diffuse ST elevation
2. PR segment depression
3. Junctional rhythm
4. True posterior MI
5. None of the above

Patient treated with colchicine

- Pain continued
- Effusion enlarged and required surgical drainage
- Pathology non-specific, no etiology identified
- Eventually pain resolved with slowly tapered ibuprofen and colchicine

Most recent ECG
ECG changes in pericarditis compared to STEMI: Key Points

- ST elevation may be diffuse
  - J point elevation
  - Rarely > 5 mm
  - Retains normal concavity
- No reciprocal ST depression
- PR segment depression
  - II, III, aVF
  - Elevation in aVR “knuckle sign”
- ST segments normalize before T waves invert
- No Q waves

ECG changes Pericarditis compared to Early Repolarization: Key Points

- Involves limb leads in pericarditis, rare in early repolarization
- Ratio of ST elevation to T wave amplitude > 0.25 in V6 strongly favors acute pericarditis
- PR segment changes

Other causes of ST elevation:

- Ischemic Heart Disease
  - Coronary artery spasm (reversible)
  - Old MI with persistent STE (aneurysm)
- Left ventricular hypertrophy (V1, V2)
- Myocarditis
- Massive PE
- Hyperkalemia
- Hypothermia
- Brugada syndrome

Brugada Syndrome

- Cause of sudden death
- Usually adult onset
- M > F
- Sodium channel mutation

Case 5: 87 yo woman transferred due to subarachnoid hemorrhage (SAH)

- H/O bioprosthetic aortic valve replacement, coronary arteries normal in 2005
- Otherwise doing well with good neurologic improvement

Day 2 of admission: Patient developed heart failure

- Troponin 1.7, BNP > 3000 pg per milliliter
- Cath deferred because of contraindication to anticoagulation
The most likely diagnosis is: ?

1. Acute LAD occlusion
2. Cerebral T waves
3. Pulmonary embolism
4. Acute aortic valve thrombosis

Day 2 of admission: Patient developed heart failure

- Troponin 1.7, BNP > 3000 pg per milliliter
- Cath deferred because of contraindication to anticoagulation

Echocardiogram

- Dx: “Cerebral T waves”
- Neurogenically-mediated cardiac injury

Pre-Discharge to Rehab Facility

Neurogenically-mediated Cardiac Injury: Key Points

- ECG changes common in patients with SAH and stroke
- TW changes precordial: 25-35%
- QT prolongation: 25%-35%
- ST segment deviations: 25%
- Prominent U wave: 10-25%

Takotsubo cardiomyopathy: Key Points

- Stress induced cardiomyopathy bears similarities to neuro-cardiogenically mediated injury
- Post-menopausal women most often affected (90% cases)¹
- Can mimic ACS, characterized by:
  - Chest pain and dyspnea
  - Reversible left apical ballooning
- Often dismissed as “anxiety”
- Preceded by intense emotional or physical stress
- Pts have lower incidence of traditional cardiac risk factors (eg, smoking, HTN, family hx)¹
- 95% patients recover completely by 8 weeks¹²

². Pilgrim TM & Wyks TR. Int J Cardiol. 2008;124(3):263-266
Case 6: 67 yo man admitted with chest pain of sudden onset

- Severe chest pain with pleuritic component
- HR 120 irreg/irreg, BP 90/70 mm Hg, JVD to angle of jaw, lungs clear, cor distant heart sounds
- CXR clear
- First troponin: 5.6 ng/mL

Most likely cause of ECG changes is: ?

1. Acute left main thrombosis
2. Takotsubo syndrome
3. Acute pulmonary embolus
4. Pericardial tamponade
5. None of the above

ECG/Echo: Acute RV Strain

- McConnell’s sign: right ventricle (RV) dysfunction with apical sparing (echo)
- CT scan: bilateral pulmonary emboli
- Pt received thrombolysis with TPA
- Clinically improved and discharged on enoxaparin bridging to warfarin

Acute pulmonary embolus: key points

- Associated ECG findings, often transient:
  - an S1 Q3 T3 pattern
  - a prominent S wave in lead I
  - a Q wave and inverted T wave in lead III
  - sinus tachycardia
  - T wave inversion in leads V1 - V3
  - Right Bundle Branch Block
  - Low amplitude deflections

Acute pulmonary embolus: key points

- Common diagnosis in patients presenting with chest pain
- Absence of ECG abnormalities has no significant predictive value
- Certain ECG changes can provide important clues as to Dx of PE


Case 7: 34 yo woman presents to your office c/o progressive exertional dyspnea X 1 yr

- Severe shortness of breath and mild chest discomfort walking one block
- H/O cocaine and methamphetamine use
- 2 pregnancies, 8 and 10 yrs earlier, uncomplicated
- PE: BP 90/60 mm Hg, HR 100, O2 sat 98%,
  - JVD to angle of jaw
  - Holosystolic murmur at left sternal border (LSB)
- HIV negative

Findings on the ECG suggest:  

1. Right arm – left arm lead reversal
2. Dextrocardia
3. Right ventricular hypertrophy
4. None of the above

ECG

ECG Findings: Other Right Heart Disease

ECG Findings: Right ventricular hypertrophy

- Right axis deviation (>90)
- RV1 >7 mm
- RV1 + SV5 or V6 >10 mm
- R/S ratio in V1 >1 or S/R ratio in V6 <1
- Incomplete right bundle branch block
- ST-T wave abnormalities (“strain”) in inferior leads
- Right atrial hypertrophy (P pulmonale)
- S1 S2 S3 pattern (particularly in children)

The most appropriate next step in evaluating this patient is:

1. Cardiac catheterization
2. Chest CT
3. 2D echo
4. Pulmonary function studies

Right ventricular hypertrophy: Key Points

- Differential diagnosis includes:
  - Pulmonary arterial hypertension
    - Primary
    - Secondary
  - Chronic thromboembolic disease
  - Eisenmengers syndrome
  - Pulmonary stenosis
  - Other
- Aggressive evaluation in the symptomatic adult

Summary: Key Take Home Messages

- ECG remains a first line diagnostic tool
- Critical in the diagnosis of Acute Coronary Syndromes despite lack of sensitivity and specificity
  - ST Elevation a sign of acute coronary thrombosis warranting cath lab activation although there are many mimickers
  - ST Depression a sign of subendocardial ischemia often demand, may represent non-STEMI
- ECG often points to acute and chronic right heart disease
60 yo woman with hypertension  
c/o mild palpitations B/P = 160/90

What is the diagnosis?

1. Type 1 second-degree AV block  
2. Atrial fibrillation (AF) with pre-existing left bundle branch block (LBBB)  
3. RV conduction delay  
4. Ventricular tachycardia

Atrial fibrillation with pre-existing LBBB

- Sometimes confused with ventricular tachycardia (VT)  
- Irregularly irregular rhythm suggests AF  
- Features of typical LBBB  
  - wide QRS >120 ms (3 small squares)  
  - No secondary R wave in lead V1  
  - No lateral Q waves  
  - Lack of Concordance in the precordial leads

60 yo woman with hypertension

A 70-year-old man c/o exercise intolerance

What is the diagnosis?

1. Sinoatrial block  
2. Sinus pause  
3. First degree heart block  
4. Second degree heart block  
5. Third degree heart block
Complete Heart Block

- P waves not conducted to the ventricles because of block at the AV node
- P waves show no relation to the QRS complexes. They ‘probe’ every part of the ventricular cycle but are never conducted
- Ventricles are depolarized by a wide complex ventricular escape rhythm at a rate of 36

**A 90 yo woman presents c/o syncope**

---

“Trifascicular Block”

- Delay in the RBBB and L Anterior Fascicle
- PR prolongation may be due to
  - Delay in left posterior fascicle
  - AV nodal delay
- Suggests advanced conduction system disease and warrants monitoring in symptomatic patient
  - May require PM insertion

**A 25-year-old man c/o bouts of tachycardia**

---

What is the diagnosis?

1. Wolff-Parkinson-White (WPW) syndrome
2. Atrial Fibrillation
3. Atrial Flutter
4. Paroxysmal Supraventricular Tachycardia
5. Ventricular Tachycardia

**Wolff-Parkinson-White (WPW) syndrome**

- Short PR interval, less than 3 small squares (120 ms)
- Slurred upstroke to the QRS indicating pre-excitation (delta wave)

- Broad QRS
- Secondary ST and T wave changes
A 21 yo man with a long history of palpitations and, recently, blackouts

What is the diagnosis?
1. Paroxysmal Supraventricular Tachycardia
2. Ventricular Tachycardia
3. Atrial fibrillation (AF)
4. Atrial flutter with 2:1 AV conduction

Wolff-Parkinson-White syndrome with AF

- Irregularly irregular, wide complex tachycardia
- Impulses from the atria are conducted to the ventricles via either
  - both the AV node and accessory pathway producing a broad fusion complex or
  - just the AV node producing a narrow complex (without a delta wave) or
  - just the accessory pathway producing a very broad 'pure' delta wave
- People who develop this rhythm and have very short R-R intervals are at higher risk of VF

Atrial fibrillation in WPW: Key points

- Avoid beta blockers, digoxin and verapamil
- Cardioversion if unstable
- Acute medical therapy with procainamide:
  - May convert due to effects on atrium
  - Slows VRR due to effects on accessory pathway
  - Ibutilide and amiodarone may be effective
- Chronic medical therapy
  - Flecainide, propafenone or dofetilide
- Radiofrequency (RF) ablation therapy of choice

A 22 yo woman with prolonged vomiting

Which of the following ECG elements suggests hypokalemia?
1. Slight ST segment elevations
2. Absent U waves
3. Small or absent T waves
4. Complete heart block
A 22 yo woman with prolonged vomiting

Hypokalemia
The patient’s serum potassium was 1.8 mmol/L

Key ECG findings:
• small or absent T waves
• prominent U waves
• first or second degree AV block
• slight depression of the ST segment

A 58 yo man on hemodialysis presents with profound weakness after a weekend trip

Hyperkalemia
The patient’s serum potassium was 9.6 mmol/L

Key ECG Findings:
• small or absent P waves
• atrial fibrillation
• wide QRS
• shortened or absent ST segment
• wide, tall and tented T waves
• ventricular fibrillation

Which of the following ECG elements suggests hyperkalemia?

1. Prominent P waves
2. Narrow QRS complex
3. Elevated ST segment
4. Wide, tall and tented T waves
5. Ventricular tachycardia
Summary: Key Take Home Messages

• Dysrhythmias
  – 12 lead EKG can be important in athletic screening for congenital propensity to arrhythmias/SD
    • WPW
    • Brugada Syndrome
    • Long QT syndrome
    • Short QT syndrome
  – Conduction system disease may have typical and atypical presentations in the elderly
    • Syncope
    • Fatigue
    • Exercise intolerance
    • Altered mental status

Post-test Audience Response Question: ?

Which cardiac dysrhythmia is most often associated with successful reperfusion?

1. Accelerated idioventricular rhythm
2. Ventricular fibrillation
3. Atrial flutter
4. Wenckebach (type I) 2nd degree AV block

Post-test Audience Response Question: ?

Which of the following is contraindicated for the patient with Wellens’ syndrome?

1. Cardiac catheterization
2. Antiplatelet therapy
3. Echocardiogram
4. Exercise stress testing

Post-test Audience Response Question: ?

True or false: The absence of ECG abnormalities has strong predictive value in ruling out acute pulmonary embolus.

1. True
2. False