Using the 12 Lead ECG to Evaluate Palpitations & Syncope: A Case Study Approach

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Goals for the Workshop:

- Gain confidence when evaluating a patient with CC: palpitations or syncope

- Develop a differential diagnosis and perform a focused H & P

- ECG Interpretation
  - * What to look for
  - * What the clues mean

- Next step in evaluation and management

- When to refer
Case #1
Rapid Pulse Paxton

- A 17 year old male presents to PCP’s office with complaints of intermittent palpitations x 2 months. Occurs at rest, does not wake from sleep, not associated with physical exertion. Typically lasts 4-5 minutes, feels lightheaded and “is aware of heart beating fast”
- Denies chest pain or SOB.
- PMH: Negative         Surgical Hx: Negative       No meds       NKDA
- Family Hx: Negative for early MI or Sudden Cardiac Death (SCD)
- Social Hx: High school senior, previously played soccer, now involved with choir and theatre
- Nonsmoker, no alcohol, no illicit drug use
- Caffeine: No coffee or tea, consumes 1-2 energy drinks/day
- Your differential diagnosis includes…
Differential Diagnosis

- **Arrhythmias**
  - PACs, PVCs, SVT, VT

- **Simulants**
  - Caffeine
  - OTC or illicit (Sympathomimetics, cocaine or amphetamines)

- **Hypertrophic Cardiomyopathy (HCM)**

- **Metabolic**
  - Hypoglycemia
  - Anemia (females)

- **Other?**
Palpitations

- Definition: “an unpleasant awareness of the forceful, rapid or irregular beating of the heart”

- Described as:
  - “rapid fluttering in the chest”
  - “flip-flopping in the chest”
  - “pounding sensation in the chest or neck”

- These descriptions may actually help determine the cause
Causes of Palpitations

- Cardiac
  - Arrhythmia
  - Cardiac shunt
  - Valvular heart disease
  - Pacemaker
  - Atrial myxoma
  - Cardiomyopathy

- Psychiatric
  - Panic attack/Panic disorder
  - GAD
  - Somatization
  - Depression

- Catecholamine Excess
  - Stress
  - Exercise

- Medications
  - Sympathomimetic agents
  - Vasodilators
  - Anticholinergics drugs
  - Beta blocker withdrawal

- Habits
  - Cocaine, Amphetamines
  - Caffeine, Nicotine

- Metabolic Disorders
  - Hypoglycemia
  - Thyrotoxicosis
  - Pheochromocytoma
  - Mastocytosis

- High Output States
  - Anemia, Pregnancy, Fever

Palpitations Etiology

Study of 190 patients presenting to a University Medical Center
CC: Palpitations  Able to determine etiology in 84%

- Emergency Department
  - 43% Cardiac
  - 31% Psychiatric
  - 10% Miscellaneous
    - Medication-induced
    - Thyrotoxicosis
    - Caffeine
    - Cocaine, amphetamines
    - Anemia

- Medical Clinic
  - 21% Cardiac
  - 45% Psychiatric
  - 18% Miscellaneous

Diagnostic Evaluation of Palpitations

- History
- Physical Examination
- 12 Lead ECG
- Limited laboratory testing
  - Blood glucose
  - TSH
  - CBC or H&H
  - Other: Urine βHCG, toxicology screen, metanephrines
- Ambulatory Monitoring - may be helpful
- Specialized testing - rarely needed
History

- Age at onset of palpitations
  - < 20 years
    - Favors SVT due to accessory pathway (WPW, LGL)
    - PVCs or VT with HCM or congenital long QT (Tdp)
  - Older
    - Favors paroxysmal SVT, atrial tachycardia, atrial fibrillation
    - Ventricular arrhythmias due to structural or coronary artery disease (CAD)

- Description of palpitations
  - Regular or Irregular rhythm?
  - Rapid or slow rate?
  - “Flip-Flopping” – PACs, PVCs, Atrial fib
  - “Fluttering”- sustained SVT or VT
  - “Pounding in neck” – AV dissociation, PVCs, VT, Third degree heart block
Physical Exam

- Cardiac & Pulmonary Auscultation
- Mitral Valve Prolapse (MVP)
  - Mid-systolic click, occasionally a mid-systolic murmur
  - PACs, SVT, A fib
  - PVCs, nonsustained VT

- Hypertrophic Cardiomyopathy (HCM)
  - Systolic murmur at left sternal border, increases with Valsalva maneuver
  - Atrial fibrillation, VT

- Dilated Cardiomyopathy and Heart Failure
  - S3 or S4 gallops, systolic murmur if MR
  - Signs of failure: Pulmonary congestion, hepatomegaly, peripheral edema
  - Laterally displaced PMI
  - Atrial fibrillation, PACs, PVCs, VT
12 Lead ECG Interpretation

- A - Analyze rhythm
  - Measure PR, QRS and QT

- B - Bundle Branch Block

- C - Chamber enlargement (Atrial and Ventricular)

- D - Determine axis

- E - Evaluate each area:
  - **Inferior** (II, III, AVF)
  - **Anterior/Septal** (V1-V4)
  - **Lateral** (I, AVL, V5, V6)
  
  For:
  - **Injury:** ST Elevation
  - **Ischemia:** ST Depression, T wave inversion
  - **Infarction:** Q waves or Loss of R waves
ECG Findings

- Step A) Analyze Rhythm:
  - Rare to capture arrhythmia on ECG, may see Atrial fib/flutter or VT
Arrhythmias

- PACs

- PVCs
Cardiac Intervals

- Normal
  - PR Interval
    - 120 ms - 200 ms (0.12 - 0.20 sec)
  - QRS
    - 40 ms - 100 ms (0.04 - 0.10 sec)
  - QT/QTc
    - 360 ms - 440 ms (0.36 - 0.44 sec)
Further Diagnostics

- **Echocardiogram**
  - Structural heart disease, HCM
  - Evaluate murmur
  - ECG Changes: Prior MI, L BBB, LVH

- **Ambulatory Monitoring**
  - Holter Monitor
    - Continuous 24-48 hour recording, patient keeps diary records time & symptoms
    - 2-3% arrhythmia detection
  - Event Recorders
    - External, patient triggered, 2-4 weeks
    - Implantable Loop Recorder, up to 36 months
    - 8-20% arrhythmia detection
    - More cost effective
Case #1
Rapid Pulse Paxton

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- Family Hx:  Negative for early MI or Sudden Cardiac Death (SCD)
- Social Hx:  High school senior, previously played soccer, now involved with choir and theatre
- Nonsmoker, no alcohol, no illicit drug use
- Caffeine:  No coffee or tea, consumes 1-2 energy drinks/day
- Additional History?  Physical Exam?  Diagnostics?
Sinus Bradycardia 52     PR 0.12     QRS 0.11
QT/QTc  0.40/0.37     QRS Axis  +58°
Wolff-Parkinson-White (WPW)
Accessory pathway
Allows early depolarization of ventricles

- Short PR < 120 ms
- Wide QRS
- Delta wave
Wolff-Parkinson-White (WPW)

Orthodromic conduction (80-90%)

Antegrade conduction across AV node and Retrograde conduction across accessory pathway during SVT

Normal sinus rhythm
Primary Care Management

- Obtain Echo
  - Structural heart disease
- Cardiology referral
  - Electrophysiology study
- Treatment
  - Avoid stimulants
  - Medications
    - Flecainide, Propafenone (Avoid verapamil or digoxin)
    - Radiofrequency Ablation (90-95% effective)
      - Symptomatic tachyarrhythmias
      - Occupation at high risk (pilots, truck drivers, athletes)
Post-Ablation
“Early Repolarization”
Case #2
Palpitations Peggy

- 65 year old woman presents with intermittent palpitations. “Feels like her heart is doing flip-flops in her chest.” Lasts approximately 20-60 minutes and becomes SOB & very fatigued. First noticed about 3 days ago. No chest pain, but chest feels heavy and hard to get full deep breath. No recent illness, travel or sick contacts. No fever or chills, Denies GI symptoms.

- PMH: COPD, T2DM, hypertension, hypothyroidism

- Social History: 1 ppd smoker x 40+ years. Drinks one gin & tonic every evening and 1-2 cups of coffee every AM

- Meds: Metformin 1000 mg BID, Lisinopril 20 mg daily, HCTZ 12.5 mg Levothyroxine 88 mcg daily. Spiriva and Albuterol inhalers

- What’s on your differential?
Differential Diagnosis

- Arrhythmia
  - PACs, PVCs
  - Atrial Fibrillation or Multifocal Atrial Tachycardia (MAT)
- ACS, Heart Failure
- Pulmonary/COPD/Hypoxia
- Metabolic
  - Thyrotoxicosis
  - Electrolytes
  - Hypoglycemia
- Anxiety

DIAGNOSTICS:
- ECG
- Labs
  - CBC
  - BMP/CMP
  - TSH
  - BNP
  - Troponin

MAT has 3 or more P wave morphologies due to multiple atrial pacemakers
Chamber Enlargement

- Atrial Enlargement
  - Normal P wave
    - Amplitude 1-2.5 mm
    - Duration < 110 ms
  - RA Enlargement P Pulmonale
    - P wave amplitude > 2.5 mm
  - LA Enlargement P Mitrale
    - P wave duration ≥ 120 ms

- Causes: Valve disorders, COPD, heart failure, PAH
- Increased risk for PACs, A Fib, A Flutter, MAT
Ventricular Enlargement and Strain

- **RV Enlargement**
  - Tall R waves in V1 and V2
  - Deep S waves in V5 and V6
- **RVH**
  - R wave in V1 + S wave V5 > 10.5 mm and RAD

- **LV Enlargement**
  - Tall R waves in V5 and V6
  - Deep S waves in V1 and V2
- **LVH**
  - R wave in V5 or V6 + S wave in V1 or V2 ≥ 35 mm

“Normal R wave Progression”
Heart rate 82  PR unable to measure
QRS 0.08  QT 0.42  Axis + 60°
Atrial Fibrillation

- **Causes:** COPD, hyperthyroidism (thyrotoxicosis), HTN, mitral valve dysfunction, ischemic heart disease, rheumatic heart disease, PE, heart failure, binge drinking “holiday heart”

- **Symptoms:**
  - Paroxysmal (duration < 7 days)
  - Persistent (duration > 7 days) or Chronic (continuous)
    - Loss of “atrial kick” decreased stroke volume & cardiac output
    - Heart Failure symptoms?
    - Increased risk thrombus/emboli formation

- **Treatment:**
  - Identify cause: ACS, heart failure, valvular heart disease, thyroid, etc.
  - Rate or rhythm control
  - Anticoagulation therapy
**CHA$_2$DS$_2$-VASc**

Stroke Risk Assessment Tool

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<th>Points</th>
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<td>H</td>
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- **C** - CHF
- **H** - HTN
- **A** - Age ≥ 75 yrs
- **D** - Diabetes Mellitus
- **S** - Prior Stroke/TIA/VTE
- **V** - Vascular Disease (Prior MI, PAD)
- **A** - Age 64-74 years
- **Sc** - Sex category (female gender)
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<tr>
<th>CHA$_2$DS$_2$-VASc Score</th>
<th>Risk Category</th>
<th>Guidelines</th>
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<tr>
<td>0</td>
<td>Low</td>
<td>No antithrombotic therapy</td>
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| 1                       | Moderate     | No antithrombotic therapy  
|                         |              | Tx with oral anticoagulant or ASA may be considered |
| ≥ 2                     | High         | Oral anticoagulant |

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<td>9 points</td>
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Case #3
Blacked-out Ben

- 21-year-old man presents with syncopal episode while playing pickup basketball game last evening. Team mates witnessed him collapse, was unresponsive for 30-60 seconds with agonal breathing. No seizure activity, no incontinence. Denies prodromal symptoms.

- PMH: Seasonal allergic rhinitis, no prior syncopal episodes, no history TBI
- Surgical History: Wisdom teeth extraction age 20
- Family History: Negative for early MI, SCD
- Social History: Single, Junior in college, civil engineering degree
- Non smoker, occasional beer on weekends, no illicit drugs
- Caffeine: Cup of coffee every morning, Starbucks mocha on Sundays
Syncope

Definition: Transient loss of consciousness characterized by unresponsiveness, loss of postural tone & spontaneous recovery

Causes:
- Cardiac:
  - Outflow obstruction (AS, HCM, PE)
  - Arrhythmias (VT, Torsades de Pointes, Sick Sinus Syndrome, AV block)
- Vasovagal (response to pain, panic, fright, exercise)
- Orthostatic hypotension
- Carotid sinus stimulation (position change, turning head, tight collar)
- Situational (micturition, defecation, coughing)
Evaluation of Syncope

- **History**
  - Description of Event
  - Preceding or Precipitating events
    - Exercise – during or following exercise is concerning for cardiac etiology
    - Startle or acute arousal- consider Long QT syndrome
    - Postural Changes – vasovagal
    - Pain or emotional stress - vasovagal

- **Physical Exam**
  - Cardiac Auscultation
  - PMI
  - 12 Lead ECG
Case #3  Blacked out Ben
Sinus rhythm rate 90     PR 0.16      QRS 0.08      QT 0.48     QTc 0.59
Long QT Syndrome (LQTS)

Disorder of myocardial repolarization (T wave) Results in prolonged QT interval

Normal QT interval  360-440 ms
QT Interval affected by:  Age, gender & heart rate

Abnormal:  Men  > 470 ms
            Women  > 480 ms
            Any time  > 500 ms
Causes of Long QT Syndrome (LQTS)

- **Primary/Congenital**
  - Genetic (Romano-Ward syndrome)

- **Acquired**
  - Medication-induced
  - Electrolyte Imbalance
    - Hypokalemia
    - Hypomagnesemia
    - Hypocalcemia
  - CNS: SAH, Stroke
  - Cardiac: HF, LVH, CAD
  - Hypothermia
Acquired Causes of LQTS

- Medication-Induced – most block K+ channel in phase 3
  - Antiarrhythmics: Quinidine, procainamide, sotalol, dofetilide, amiodarone
  - Macrolide antibiotics: Azithromycin, clarithromycin, erythromycin
  - Fluoroquinolones: Ciprofloxin, levoflroxacin
  - Antifungals: Fluconazole “azoles”
  - Metronidazole
  - Psych: Haloperidol (IV), TCAs, SSRIs, trazadone, atypical antipsychotics
  - Opioids: Hydrocodone, buprenorphine, methadone
  - GI: *Cisapride, chronic PPIs, ondansetron
  - Nonseadating antihistamines: *terfenadine, *astemizole, hydroxyzine
  - CYP450 3A4 inhibitors or inducers: Phenytoin, grapefruit juice, azoles, macrolides, St John’s wort

- Electrolytes: Potassium, Magnesium, Calcium
- * Taken off market
Torsades de Pointes (TdP)

Polymorphic ventricular tachycardia

Management:
Identify & treat underlying cause
Cardiology referral, genetic testing
Family members evaluated
Dual chamber pacemaker, AICD
Case #4 Laceration Larry

- 48-year-old Asian man presents to PCP with a laceration to his left eyebrow. He thinks he passed out, as he woke up on his bathroom floor in a pool of blood.
- HPI: Had just stepped out of the shower. Passed out once a few months ago.
- PMH: HTN
- Family History: T2DM, HTN, Father died in his sleep at age 55. Paternal uncle SCD age 52.
- Medications: Lisinopril 10 mg daily, ASA 81 mg daily.
- Social History: Divorced, works as computer programmer.
- Nonsmoker, no alcohol or illicit drugs.
What is on your differential?

- Postural hypotension
- Vasovagal
- Arrhythmia
  - Ventricular (VT, TdP, long QT)
  - Supraventricular
  - Bradycardia, AV Block
- Seizure
- TIA
- Other?

What diagnostic tests would you consider?

- Postural vitals
- ECG
- Holter monitor
- EEG
- Head CT
- Labs?
- Other?
Case #4
Sinus rhythm rate 85   PR .16   QRS .10   QT .38   QTc .43
Brugada Syndrome

- Genetic, Asian
- Male > Female 8:1
- Na+ channel disorder
- Pseudo-RBBB pattern in V1-V2
- ST elevation V1 and V2
- J-point elevation ≥ 2 mm
- Sloped ST segment
- T wave inversion V1-V3

Type 1 “Coved” ST elevation
Type 2 “Saddle back” ST elevation
Let’s compare...

Brugada
- J Point elevation
- ST elevation
- T wave inversion

Right BBB
- QRS $\geq 0.12$ sec
- rSR’ complex in V1
- Terminal R wave in V1
- T wave opposite polarity
Brugada Syndrome: Clinical Presentation

- Sudden Cardiac Arrest
  - Due to ventricular tachyarrhythmia (VF or Torsades)
- Syncope
- Palpitations
  - Due to ventricular tachyarrhythmias or A Fib
- Nocturnal agonal respirations
  - SUNDS- Sudden Unexpected Nocturnal Death Syndrome
  - SUDS – Sudden Unexpected Death Syndrome
  - Lai Tai “Death during sleep” in Thailand
  - Bangungut “To rise and moan in sleep followed by death” in Philippines
  - Pokkuri "Unexpected sudden cardiac death at night” in Japan
Evaluation & Management of Brugada Syndrome

- **Evaluation:**
  - R/O structural heart disease
    - Echocardiogram or Cardiac MRI
    - Stress testing
  - Genetic testing
    - SCN5A – Cardiac Na+ channel gene (15-30% have mutation)
    - Sodium channel blocker challenge (Flecainide)

- **Management:**
  - AICD

*In other words… Refer to Cardiology*
Case #5
Soccer-playing Sarah

- Sarah is a 14-year-old teenage girl. While at soccer practice yesterday, she passed out and had what bystanders described as a seizure. She presents to her PCP’s office the following day with her mother.
- “I was playing soccer, and the next thing I remember is waking up with everyone standing over me. I’m fine.” Denies headache, N/V, trauma
- T 98.7  P 68 regular  R 16  BP 108/64  SpO2 100% on room air
- Past Medical/Surgical History: Negative, no history of seizure disorder
- Medications: Ibuprofen PRN for muscle aches or menstrual cramps
- Allergies: NKDA
- What is on your differential?
Differential Diagnosis

- Seizure
- Hypoglycemia
- Arrhythmia
  - SVT
  - Ventricular tachycardia
  - LQTS and Torsades
  - Bradycardia
- Hypertrophic Cardiomyopathy
- Postural Hypotension
- Other?
Hypertrophic Cardiomyopathy (HCM)

- Genetic disorder, myocyte hypertrophy
- Asymmetric hypertrophy, involves intraventricular septum
- Obstruction of left ventricular outflow tract (LVOT)
- Presentation:
  - Asymptomatic
  - Chest pain, dyspnea
  - Palpitations
  - Dizziness, fatigue
  - Syncope or SCD
History and Physical Exam

- HISTORY:
  - No prodromal symptoms
  - Negative for chest pain, dyspnea, palpitations, prior syncope
  - No family history of SCD

- PHYSICAL EXAM:
  - Normal with the exception...
  - Hear a systolic murmur at left lower sternal border
  - Increases in intensity with Valsalva (decreased preload)
Case #5 Soccer Playing Sarah

A) Sinus rhythm rate 62
1\textsuperscript{st} degree AV block
PR 220  QRS 80  QT 420

B) Abnormal QRS in V1
QRS 80 ms

C) No chamber enlargement

D) LAD \(-30\) degrees

E) * T wave inversions
V4-V6, I and AVL
* Poor R wave progression V1-V3
* Q waves I & AVL
“Pseudo-infarct” pattern with LVH or RVH
ECG Interpretation in Athletes “Seattle Criteria”

- Summit on ECG interpretation in athletes February 2012 in Seattle

- Standardized criteria to distinguish
  - Normal physiological changes “Athlete’s Heart”
  - Pathological changes associated with SCD

- Free online training module
  [http://learning.bmj.com/ECGathlete](http://learning.bmj.com/ECGathlete)
"Athlete’s Heart"
Normal ECG Variants

- Sinus bradycardia ≥30 bpm
- Sinus arrhythmia
- Ectopic atrial rhythm
- Junctional escape rhythm
- 1st degree AV Block (PR > 200 ms)
- 2nd degree AV Block Type I
- Incomplete RBBB
- Isolated QRS Voltage criteria for LVH
  - NOT associated with: LA enlargement, LAD, ST depression, T wave inversion or pathological Q waves

Common training-related ECG changes, normal variants in athletes
Do NOT require further evaluation in Asymptomatic athletes, Ages 14-35

- Early Repolarization
  - ST elevation
  - J-point elevation
  - J waves
  - Terminal QRS slurring
- Convex (domed) ST elevation with T wave inversion in V1-V4 in black athletes
“Seattle Criteria”
Abnormal ECG Findings in Athletes

- Suggests presence of pathological CV disease
- Requires further diagnostic evaluation

- T wave inversion
  - > 1 mm in two or more leads V2-V6, II and AVF or I and AVL (excludes III, AVR, V1)

- ST segment depression
  - > 0.5 mm in two or more leads

- Pathologic Q waves
  - > 3 mm depth, > 40 ms duration, in two or more leads (except III and AVR)

- Complete L BBB
  - QRS ≥ 120 ms

- Intraventricular conduction delay (IVCD)
  - Any QRS duration > 140 ms
“Seattle Criteria”
Abnormal ECG Findings in Athletes

- **LAD**
  - -30 to -90 degrees

- **LA Enlargement**
  - P wave duration > 120 ms, M-shaped P in Lead II, biphasic in V1

- **RVH pattern**
  - R wave in V1 + S wave in V5 > 10.5 mm AND RAD > 120 degrees

- **Ventricular pre-excitation (WPW)**
  - Short PR (< 120 ms) Delta wave and wide QRS (> 120 ms)

- **Long QT interval**
  - QTc > 470 ms men  > 480 ms women  any QTc > 500 ms

- **Short QT interval**
  - QTc < 320 ms
“Seattle Criteria”
Abnormal ECG Findings in Athletes

- Brugada-like ECG pattern
  - Downsloping ST segment elevation
  - Negative T wave in > two leads V1-V3
- Profound sinus bradycardia
  - < 30 bpm or sinus pauses ≥ 3 seconds
- Atrial tachyarrhythmias
  - SVT, A Fib, A Flutter
- PVCs
  - ≥ 2 PVCs per 10 second tracing
- Ventricular arrhythmias
  - Couplets, triplets and non-sustained VT
Evaluation of HCM

- 12 lead ECG
- 24 hour Holter if palpitations or syncope
- Echocardiogram/Doppler
- Cardiac MRI if echo indeterminate
- Genetic testing/counselling
- Screening of relatives
- Consider coronary angio if angina, VT or cardiac arrest
- EP study if atrial arrhythmias or WPW

- In other words, refer to cardiology...

Duke Heart Center Criteria
Pathologic vs Physiologic LVH

- **Favors HCM**
  - LV wall thickness > 15 mm
  - Unusual LVH patterns
  - LV cavity < 45 mm
  - Marked LA enlargement
  - Bizarre ECG patterns
  - Abnormal LV diastolic filling
  - Female gender
  - Family history of HCM

- **Favors Athlete’s Heart**
  - LV wall thickness < 13 mm
  - LV cavity > 55 mm
  - Normal diastolic filling
  - Normal LA size
  - Male gender
  - LV wall thickness decreases w/deconditioning
  - No family history of HCM
  - Max Vo2 > 45 ml/kg/min or > 110% predicted
Sudden Cardiac Death (SCD)

- Majority caused by ventricular arrhythmias
- Associated with CAD, HF, structural heart disease
- 5%-10% occur in structurally normal hearts
- Causes:
  - Brugada syndrome
  - Congenital or Acquired Long QT syndrome (LQTS)
  - Polymorphic VT (Torsades)
  - Idiopathic VT or VF
Confidence evaluating patient with palpitations or syncope

Focused History & Physical Exam

ECG Interpretation
* What to look for
* What the clues mean

Next step in evaluation

When to refer
Thank you for your time and participation

Comments or Questions?
Case #6  Betting Brian

Brian is a 54 year old man who presents to the ED. Developed chest pressure while golfing. Golfs twice a week. No prior history of chest pain or SOB. Sx began approximately 30 minutes PTA, after losing $100 bet on the 6th hole. Felt weak, a little lightheaded, nauseated. The guys in his foursome said he appeared pale & diaphoretic. Rated pain 5-6/10, now resolved completely after one sublingual NTG.

- **Family History:** Father had a MI in his early 60’s
- **Social Hx:** Married, lawyer, non-smoker, daily glass of wine or cocktail
- **Occasional Motrin or Tylenol, NKDA**
- **VS:** T 98.7  P 80  R 20  BP 138/84  SpO2 96%
- **Nurse has initiated an IV, place him on the monitor and obtained an ECG**
Case #6

- Normal sinus rhythm rate 75
- PR 0.16
- QRS 0.08
- QT 0.38
What would you do?

- Chest X-ray and cardiac markers are normal. Chest pain has completely resolved, his vital signs and rhythm have been stable.

- You would…
  - A) Discharge, follow up with his PCP
  - B) Arrange for cardiac stress testing
  - C) Arrange for cardiology referral
  - D) Consult cardiology to arrange for non-emergent heart cath
  - E) Consult cardiology for emergent PCI
Wellens Syndrome
Critical Stenosis of Proximal LAD

- Angina symptoms
- Negative cardiac markers
- Deeply, symmetrically inverted T waves in $V_2$, $V_3$
Wellens Syndrome

- **Type 1**
  - 75% of cases
  - Symmetrical T wave inversion V2 and V3
  - No/minimal ST changes

- **Type 2**
  - 25% of cases
  - ST elevation with T wave inversion
Wellens Syndrome: Clinical Presentation

- Angina symptoms
- Normal or minimally elevated cardiac markers
- ECG:
  - No pathologic Q-waves or loss of R-waves (signs of infarction)
  - ST segment isoelectric in V1-V3
  - Deeply, symmetrically inverted T-wave V2-V5 or V2-V6

- Indicative of critical stenosis of the proximal LAD
Wellens Syndrome: Management

- Cardiac catheterization and PCI
- Stress testing may precipitate a myocardial infarction
- Medical management ineffective
  - 75% will develop anterior MI within weeks
Critical Stenosis of Proximal LAD

LAD Supplies:
Anterior and Septal walls of LV
Bundle branches
Sinus Tachycardia rate 142
PR .12
QRS .09
QT .30
QTc .46
Pulmonary Embolism ECG Changes

- Sinus tachycardia, new onset atrial fibrillation
- Right bundle branch block pattern
- P-Pulmonale (RA enlargement)
- Cor pulmonale (RV enlargement)
- RAD
- Non-specific ST/T wave changes
- “S1 Q3 T3 Syndrome” (present 20%-30%)
ECG Changes in Pericarditis

Acute (Stage I)
ST Elevation
  Concave, J Point elevation
  Diffuse
  No reciprocal ST changes
PR segment depression
ST depression in V1 or VR leads

Changes Evolve
Stage II  Days – weeks
  ST normalize, T waves flatten
Stage III  Several weeks
  T wave inversion
Stage IV  Months
  Resolution of T waves
Case #8
Sinus tachycardia rate 110  PR .26  QRS .10  QT .32
ECG Changes

- **Hypokalemia**
  - U wave
  - Flattened T wave
  - Prolonged QT Interval

- **Hypocalcemia**
  - Prolonged QT interval
  - Lengthening of ST segment

- **Hypomagnesemia**
  - T wave inversion
  - Prolonged ST segment
  - Often associated with hypokalemia