INTRODUCTION

- Brief loss of consciousness, spontaneously resolves without intervention, back to normal baseline on return to consciousness
- Final common pathway of syncope is the same regardless of underlying cause
- Variety of causes, according to Framingham Heart Study:
  - 37% unknown
  - 21% vasovagal
  - 10% cardiac
  - 9% orthostatic
  - 7% medication related
- Diagnosis matters prognostically

VASOVAGAL SYNCOPE
Pathophysiology and Clinical Features

- Inappropriate vasodilation
- Premonition of lightheadedness, nausea, pallor, sweating
- Slow progressive onset
- Various causes:
  - Unpleasant exposure to sights, sound or smell
  - Severe pain
  - Emotional distress
  - Prolonged standing or kneeling
  - Crowded or warm space exposure
  - Cough, micturition, defecation or swallowing episode

ORTHOSTATIC SYNCOPE
Pathophysiology and Clinical Features

- Postural hypotension without appropriate autonomic response
- Typically occurs within 3 minutes after position change
- Causes vary (examples: Intravascular volume loss, medication, diseases affecting vascular tone)
- Be weary in general, this does not mean work up is done
- Studies demonstrate 43%-50% false positives in adult population

PSYCHIATRIC SYNCOPE
Pathophysiology and Clinical Features

- Most commonly patients with generalized anxiety disorder or major depression
- Some studies suggest up to 42% of “unknown” causes of syncope are in this category
- Hyperventilation induced housed in this category
- Diagnosis of exclusion

NEUROLOGIC SYNCOPE
Pathophysiology and Clinical Features

- Rare, delineate seizure from syncope
- Typically caused from transient disruption of blood flow to brain
- Examples: brainstem ischemia, vertebrobasilar atherosclerotic disease, basilar artery migration, subclavian steal syndrome
- Subarachnoid hemorrhage can also present as syncope
**MEDICATION INDUCED SYNCOPE**

- Typical mechanism through orthostasis
- Most common culprits beta-blockers, calcium channel blockers and diuretics
- Identified by thorough history

**Cardiac Syncope**

Pathophysiology and Clinical Features

**Structural Disease**

- Physical arrhythmias
  - Examples: HOCM, Aortic Stenosis, PE, MI
  - Chest pain, dyspnea on exertion

**Dysrhythmia**

- Examples: Tachycardias, bradycardias, QT syndromes, Brugada
- Consider electrolyte abnormalities
- Typically no prodrome, sudden onset

**EVALUATION**

- Despite best efforts, 40% of patients will not have diagnosis
- Goal to risk stratify from emergency department
- History, History, History
- Basic workup MUST include EKG

**Tools**

- Quinn et al, 2006 studied outcomes at 7 and 30 days with patients that presented with syncope
- The five high risk criteria had an 89% sensitivity and 52% specificity for death at 1 year.
- In a validation study of 1418 patients, death rate was found to be at 1.4% at 30 days, 4.3% at 6 months and 7.6% at 1 year.

**San Francisco Syncope Rule**

Risk stratification for patients with syncope.

When to use: Yes

- Heart Failure History: Yes
- ECG:
  - Sinus:
  - VT:
  - Bundle branch block:
  - Non-sustained ventricular tachycardia:
  - Sustained ventricular tachycardia:
- Hypertension:
- Diabetic:
- History of syncope:
- Autonomic dysregulation:
- Primary syncope:

Why use: Yes

**Define abnormal EKG?**

- Ischemic/Infarct/STEMI: Changes
- Sinus tachycardia with rate above 100 beats/min
- Tachyarrhythmia (SVT, ventricular tachyarrhythmia, atrial fibrillation with AVN, atrial flutter)
- Sinus bradycardia with rate below 55 beats/min
- Non-sinus rhythm: second degree heart block, complete heart block, sick sinus rhythm
- Features suggestive of Wolff-Parkinson-White (delta waves, shortened PR intervals, widened QRS intervals)
- Features suggestive of Brugada Syndrome
- QT prolongation +50 ms
- Features suggestive of hypertrophic cardiomyopathy (left ventricular hypertrophy, large amplitude QRS, ST abnormalities, narrow QRS in leads I, AVL, V5, V6)

**5 Heart Conditions and STEMI equivalents**

- HOCM
- WPW
- Long and Short QT syndromes
- Brugada Syndrome
- ASVD
- De Winter’s T waves
- Wellens’ Syndrome
- Right MI, Posterior MI
- Sinoatrial's
**STEMI Equivalents**

- Used to identify STEMI in setting of LBBB and/or pacemaker
- Criteria with Smith's modification:
  - ST elevation of ≥ 1 mm in lead with upward QRS complex = 3 points
  - ST depression ≥ 1 mm in lead V1, V2, V3 = 3 points
  - ST elevation or depression ≥ 2 mm in a lead with downward QRS complex = 2 points
- ≥ 3 points = 98% probability of STEMI

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**De Winter's T Waves**

- Concerning for proximal LAD occlusion
- On EKG:
  - Tall, symmetric T-Waves (dagger-like) in leads V1-V4 (anterior wall starts below isoelectric baseline)
  - Spiking ST-depression at 1 point in leads V1-V4, maybe V1 and II, without STE
  - May have STE in lead aVR >0.5mm and AVL

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**Wellens' Syndrome**

- Sign of high grade proximal LAD occlusion
- Wellens' syndrome is a pattern of deeply inverted or biphasic T waves in V1-4
- Type A - Biphasic, with initial positivity B terminal negativity (25% of cases)
- Type B - Deeply and symmetrically inverted (75% of cases)
- In general, this EKG is at very high risk for acute MI, fatal dysrhythmia within 4 weeks of discharge if symptomatic (i.e., chest pain, syncope)

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**RV infarction**

- ST elevation in V1 - the only standard ECG lead that looks directly at the right ventricle
- ST elevation in lead III > lead II - lead III is more "rightward facing" than lead II
- R-said EKG obtained by placing the V4 electrode in the 9th right intercostal space in the midclavicular line
- ST elevation in V4R has a sensitivity of 88%, specificity of 78%, and diagnostic accuracy of 83% in the diagnosis of RV MI
- RV MI - 40% of inferior MI (isolated RV is very uncommon)
- Why differentiate from inferior? Preload sensitive!
Posterior MI

- Demonstrative of distal left circumflex artery occlusion or right coronary artery (lateral or inferior infarction)
- Isolated posterior MI is less common (3-11%), typically accompanies anterior infarction.
- Posterior extension implies a much larger area of myocardial damage, with an increased risk of LV dysfunction and death.
- Isolated posterior MI = indication for emergent coronary reperfusion.

On EKG:
- Horizontal ST depression or flattening in V7-9
- Tall, broad T waves (> 30 ms) in V1-3
- Upright T waves in leads V1-3

Obtain posterior EKG with leads V7-9 to reveal the greater than 1 mm STE
Hypertrophic Cardiomyopathy

- One of the most common inherited cardiac disorders
- Most common cause of sudden cardiac death in young athletes
- Mutations in multiple genes coding for sarcomere proteins, nearly 100 mutations identified
- Most commonly causes left ventricular hypertrophy without stimulus (i.e., HCM or aortic stenosis) asymmetrical along the anterior interventricular septum
- This hypertrophy can cause a dynamic left ventricular outflow tract obstruction and abnormal left ventricular architecture thus abnormal transmural electrical impulses (arrhythmia)

On EKG:
- DN and non-specific T wave and ST segment abnormalities
- Asymmetrical, deep, narrow, Q waves in lateral and inferior lead

Wolf Parkinson White Syndrome

- An accessory pathway connecting the atria to the ventricle, usually accessory pathway is FAST but may present as atrial fibrillation
- Distinguishes patients to tachyarrhythmias, most commonly WPW but may present as atrial fibrillation
- Presentation depends on impulse travel direction, orthodromic vs. antidromic

On EKG:
- Short PR interval (<120 ms)
- “Delta” wave
- Precedent T wave inversion (V1-3)

Brugada Syndrome

- Sodium channelopathy more common in southeast Asian populations
- History of unexplained death or sudden cardiac death at young age in family
- Diagnostic only if EEG and/or AH or clinical criteria (syncope or etc.)

On EKG:
- Type 1: “Coved” = 3mm ST elevation with negative deflected T in precordial leads V1-V3
- Type 2: “Saddle-back” = 2mm ST elevation in precordial leads V1-V3 with saddle shaped pattern in inferior segment

Short vs. Long QT Syndrome

- Short QT Syndrome
  - Genetically inherited cardiac channelopathy
  - On EKG:
    - QT <300 ms and QTc <400 ms
    - Fused T waves
    - Short or absent ST segments

- Long QT Syndrome
  - Congenital or acquired
  - Consider electrolyte abnormalities, intracardiac causes, medications or central causes
  - Use QTc for extremes of heart rate
  - On EKG:
    - QTc 440 ms

Arrhythmogenic Right Ventricular Dysplasia

- Fibro-fatty infiltration of myocardium that replaces conductive tissue with fibrous infiltrates
- More common in men, Italian and Greek descent
- May present with new onset CHF

On EKG:
- Epsilon wave
- T-wave inversion in precordial leads V1-V3 (more common)
- Prolonged S-wave upstroke in V1-V3
- QRS widening
EKG Practice