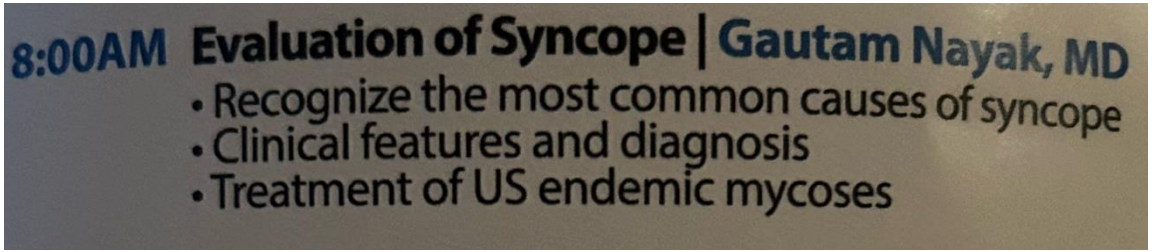
Syncope

Diagnosis and Management

Gautam Nayak, MD FACC
Cardiologist, Kittitas Valley Healthcare
Cardiologist, Mission Cardiology
Associate Clinical Professor of Medicine, University of Washington School of Medicine
Co-Founder and CEO, Coltrain

Some Goals

- Understand the various ways syncope presents.
- Feel confident in letting the history and some basic tests guide your path to a diagnosis.
- Initiate further testing and treatment as appropriate.
- Strategies on when to refer



Definition

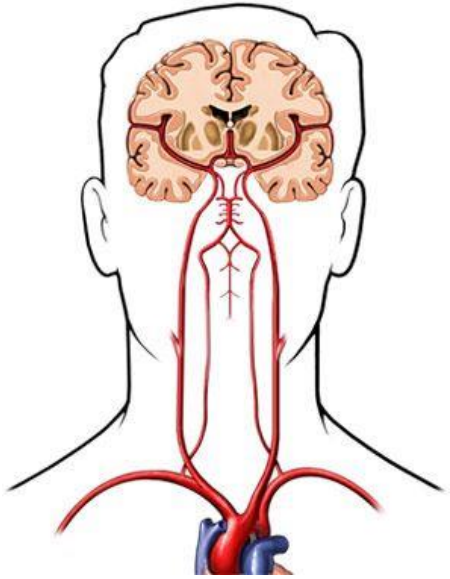
A symptom

Abrupt and transient complete **loss of consciousness**

Inability to maintain postural tone

Rapid and spontaneous recovery

Cause: cerebral hypoperfusion



*Other causes that **don't** involve cerebral hypoperfusion: seizures, trauma, metabolic, SAH, subclavian steal syndrome.

Incidence

Trimodal distribution: age 20, 60, 80

Women > Men

By the age of 60, 42% of women and 32% of men would have experienced at least one episode of vasovagal syncope in their lifetime.

Cardiovascular disease increases the incidence

Pre-Syncope:

Symptoms before syncope occurs (lightheaded, tunneled vision, nausea, flushing)

If pre-syncope progresses to syncope, then these symptoms are called the prodrome.

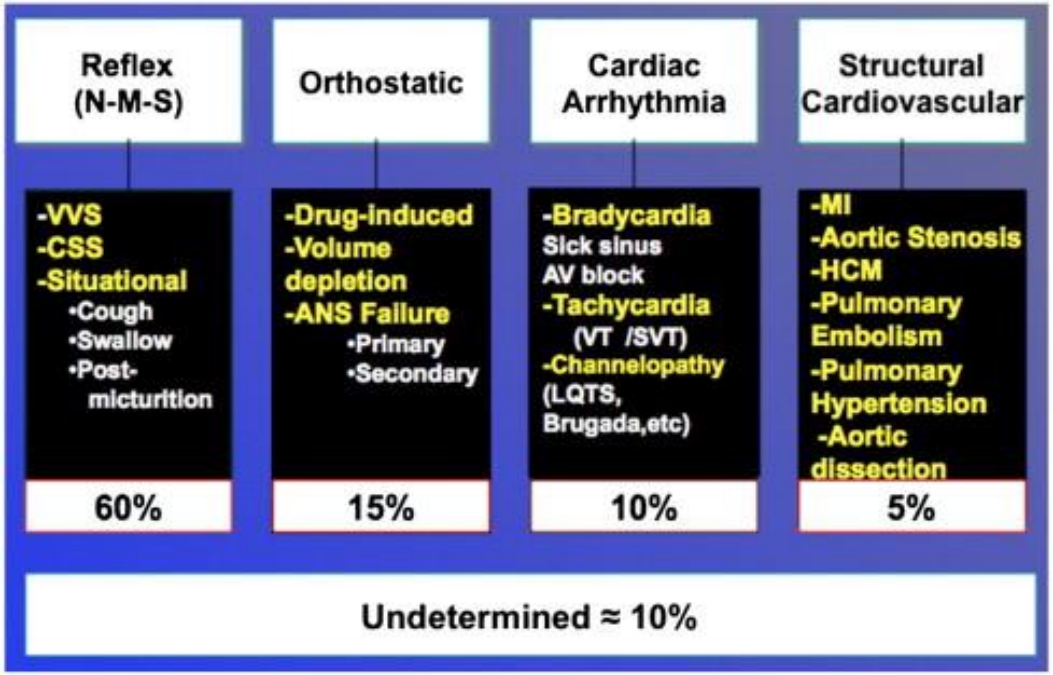
Shen et al, JACC, 2017: 70: e39-110

Classification

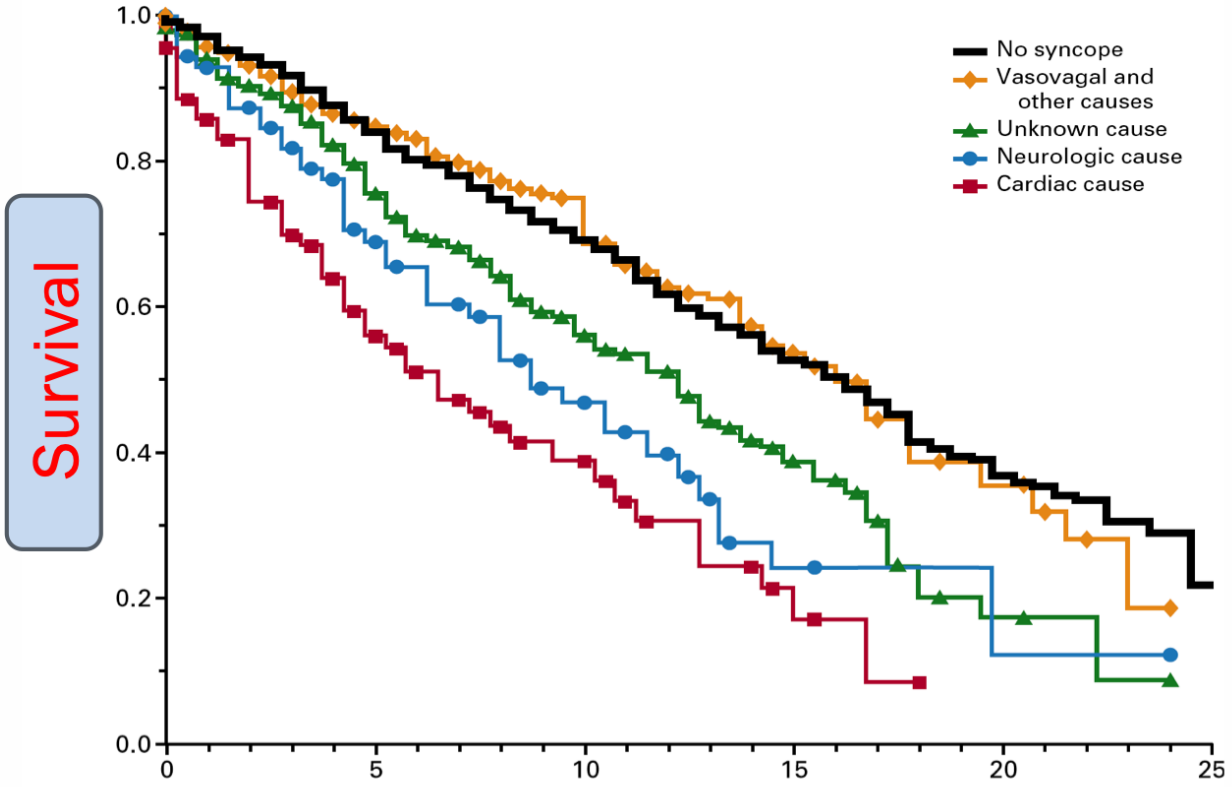
Neurally Mediated Reflex Syncope

Orthostatic Hypotension

Cardiovascular



Prognosis of Syncope



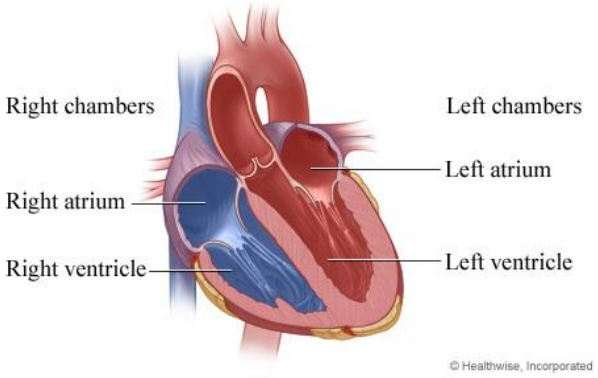
The prognosis of vasovagal syncope is the same as the general population survival

Vasovagal syncope would include carotid sinus hypersensitivity and situational syncope

1. Soteriades. NEJM. 2002;347:878

AMERICAN COLLEGE of CARDIOLOGY

Neurally Mediated *Reflex* Syncope



Vasovagal Syncope

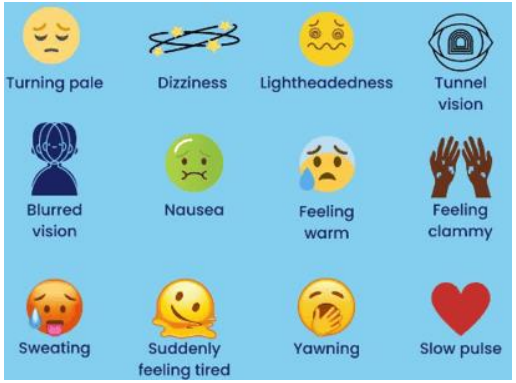
Often occurs in an upright posture (prolonged standing)

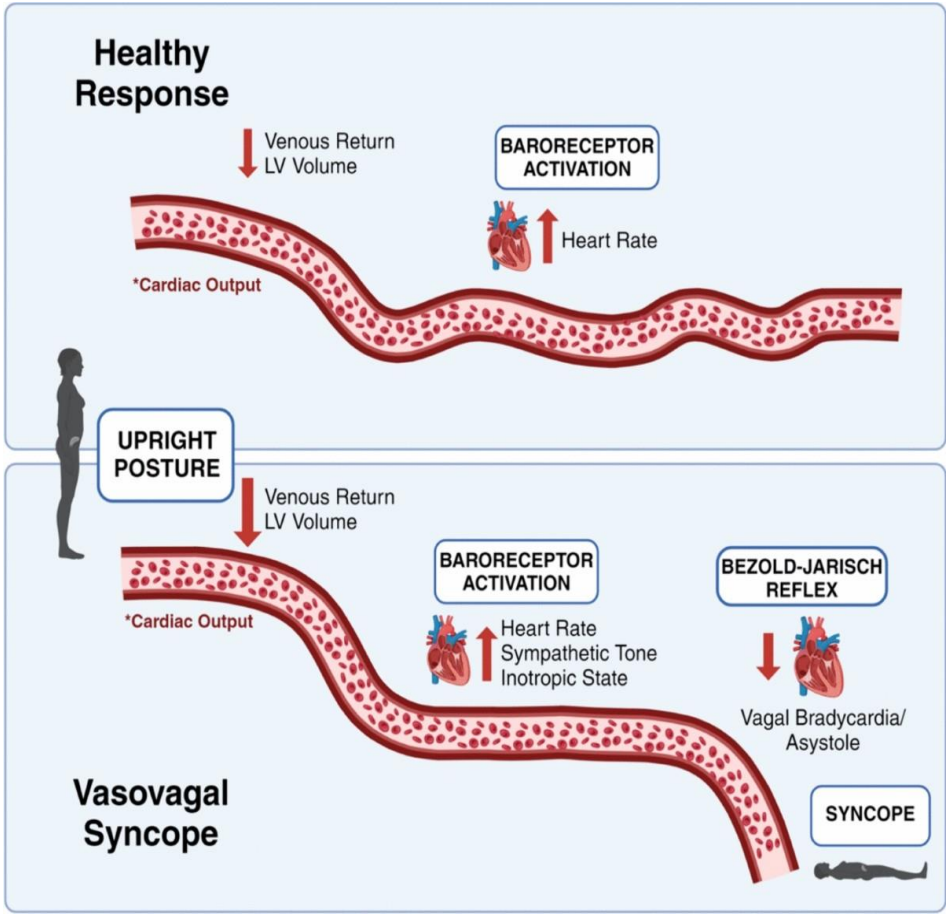
Exposure to emotional stress, pain, medical settings.

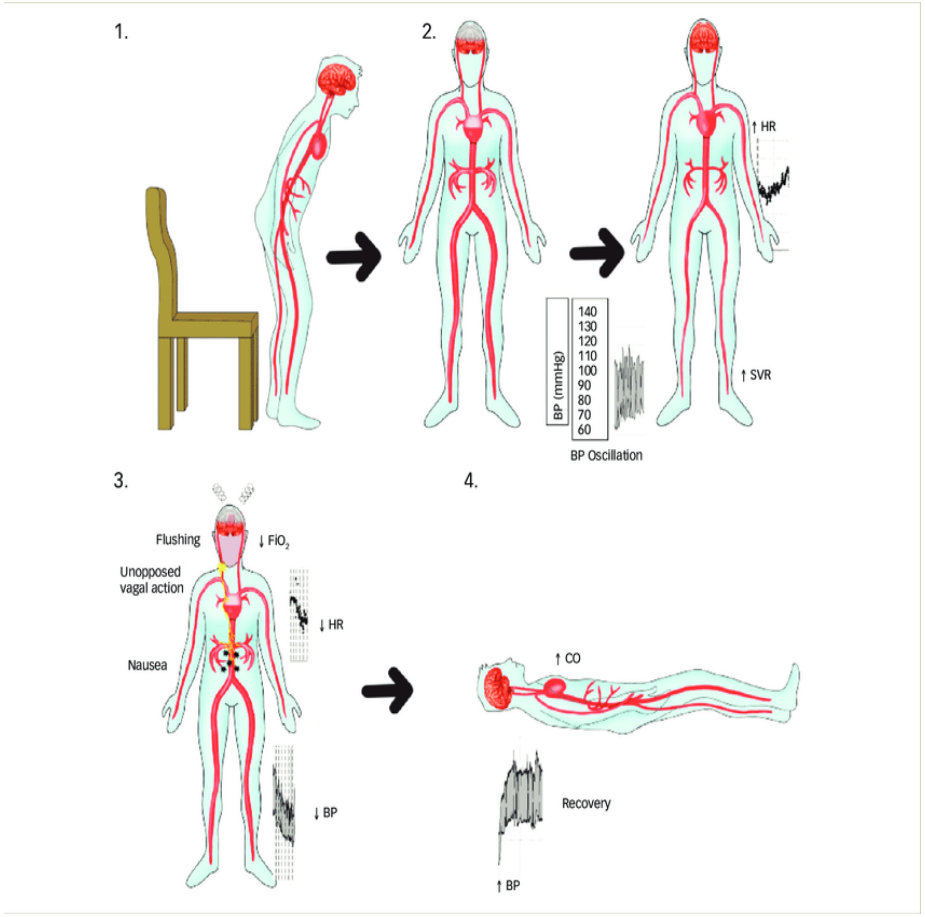
Features: diaphoresis, warmth, nausea, pallor, tunneled vision.

Hypotension +/- bradycardia → Syncope

Immediate recovery, followed by symptoms of fatigue.







Misfiring of an primitive hemorrhage reflex (Bezold-Jarisch Reflex) activating the vagus nerve

Vasovagal Response: Evolutionary Principles

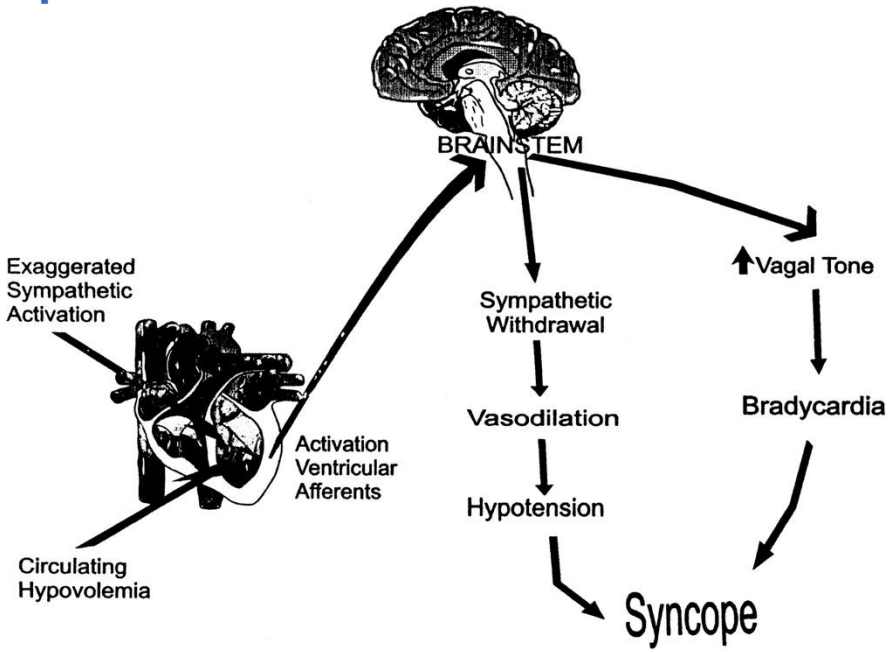
This is a primitive protective reflex from the **brainstem:**

“The ventricle is empty but contracting strongly”

- Massive hemorrhage
- Severe volume depletion
- Impending CV collapse

“We are about to lose circulatory stability”

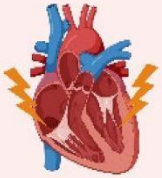
- Lowering HR reduces myocardial oxygen demand.
- Vasodilation redistributes centrally to critical organs.
- Horizontal position restores cerebral perfusion



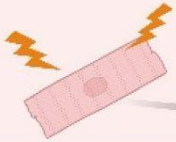
The brain thinks the body is under catastrophic circulatory threat and activates a shutdown reflex.

Neurally Mediated Reflex Syncope

Gravitational stress or intense emotion → ↑ Sympathetic tone → ↑ HR and contractility → Bradycardia and vasodilation → ↓ venous return and ↓ cardiac output → **Cerebral hypoperfusion leading to vasovagal syncope**

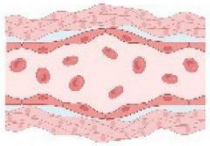


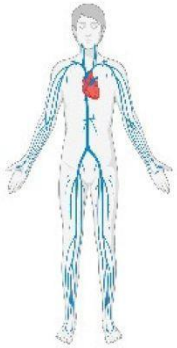
Stimulation of cardiac C fibers



Stimulation of vasodepressor region

Bezold Jarisch Reflex





Vasovagal Syncope: Treatment

Education and Lifestyle Modifications

- Increase sodium and fluids
- Physical Counterpressure Maneuvers

Increase Volume: Fludricortisone

Vasoconstrictors: Midodrine

Blunt Baroreceptors: Beta Blockers

Ongoing investigation: SSRIs

Vit D Deficiency: 5x more likely to have recurrent vasovagal syncope

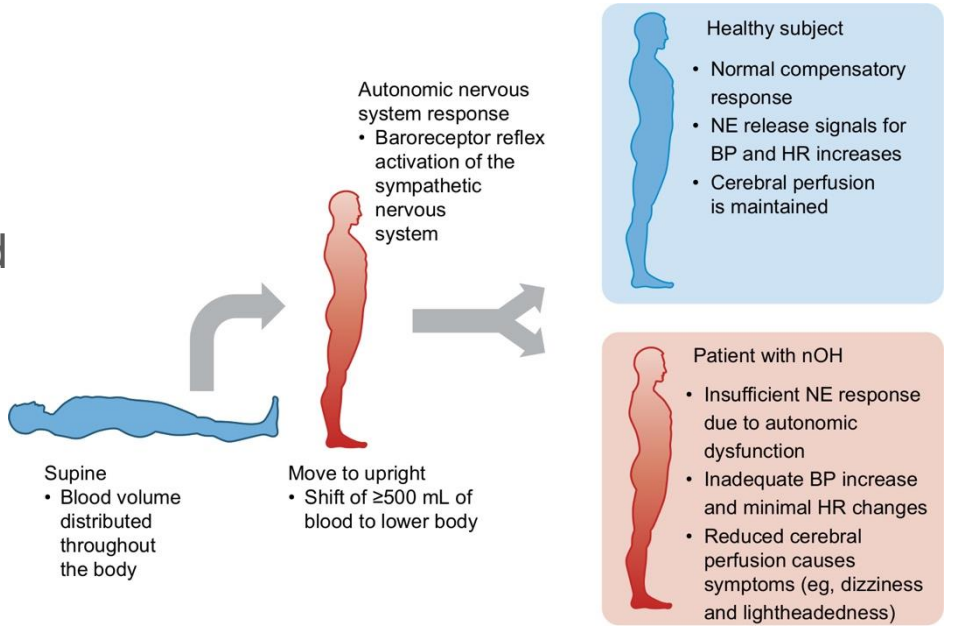
The prodrome usually lasts long enough for patients to get into a safe position.

Shen et al, JACC, 2017: 70: e39-110

Orthostatic Hypotension

Orthostatic shift of **500-800mL** of blood from the chest to the lower extremities and splanchnic circulation upon standing.

Diagnostic Criteria: >20mmHg drop in SBP from supine to standing at 1, 3, 5 minutes.



Causes:

- Neurogenic
- Dehydration / Volume depletion
- Drug-induced

Shen et al, JACC, 2017: 70: e39-110

Neurogenic Orthostatic Hypotension

Parkinson's Disease

- Rigidity, resting tremor, shuffling gait

Lewy Body Dementia

- Parkinson's with dementia, severe cognitive impairment

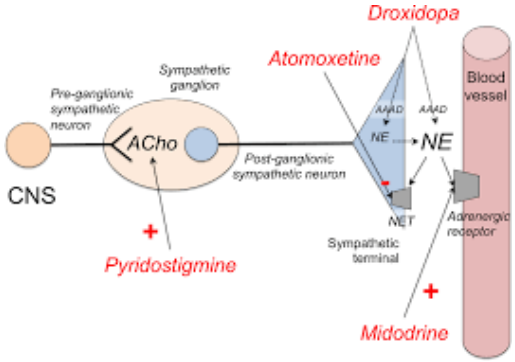
Pure Autonomic Failure

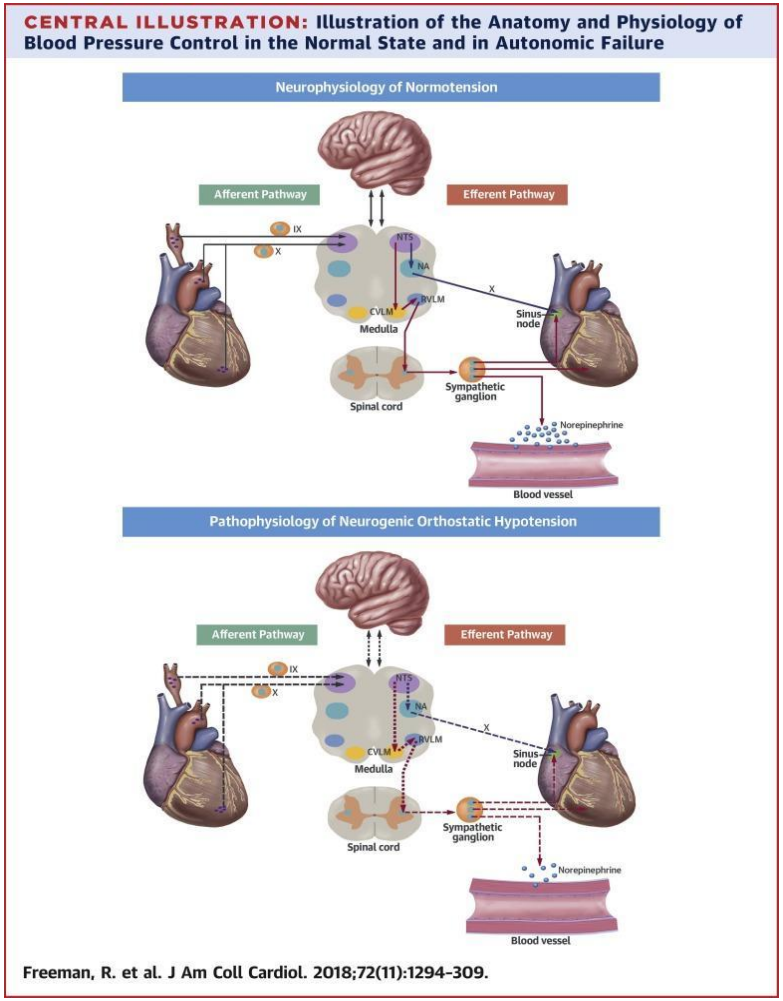
- Slow HR, urinary incontinence, difficulty sweating

Post Chemotherapy

Peripheral autonomic neuropathies

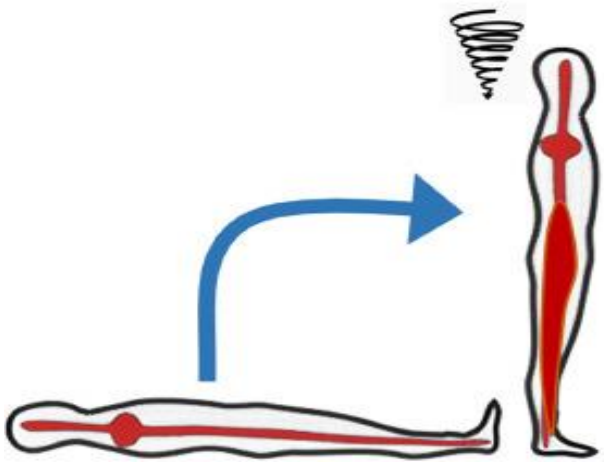
- Diabetes
- Amyloidosis





Freeman, R. et al. J Am Coll Cardiol. 2018;72(11):1294-309.

Orthostatic Hypotension: Treatment



Gravitational venous pooling
500 – 1000 mL
splanchnic circulation >> lower limbs

- Abdominal binder
- Compression Stockings
- Leg muscle pump

↓ Venous Return
↓ Cardiac Output

Baroreflex Response:
↑ Sympathetic tone
↓ Parasympathetic tone

- Norepinephrine "enhancers"
  - Pyridostigmine
  - Atomoxetine

Cardiac output recovery
Vasoconstriction
Restoration of blood pressure

- Norepinephrine "replacers"
  - Midodrine
  - Droxidopa

* **In volume depletion:** fluids (2-3L), salt liberalization (5-8 g/day), small frequent meals

Brignole, Eur Heart Journal, 2018: 1883-1948

Orthostatic Hypotension: Offending Medications

Nitrates (venodilators)

Antihypertensives - all can cause, but pay close attention to:

- Alpha blockers (also for BPH)
- **Diuretics**
- Beta blockers

PDE5 inhibitors

Dopamine antagonists (Levodopa, Carbidopa)

Tricyclic Antidepressants

*Think about a patient's living situation: more common in nursing homes.

POTS: Postural Orthostatic Tachycardia Syndrome

Dizziness or lightheadedness

Palpitations

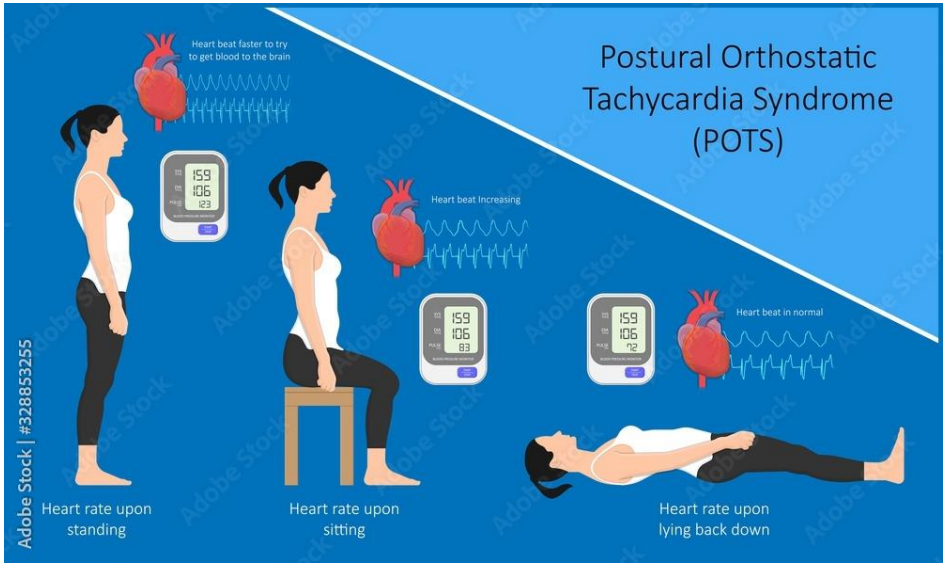
Weakness and fatigue

Exercise intolerance

Chest pain and dyspnea

Fainting

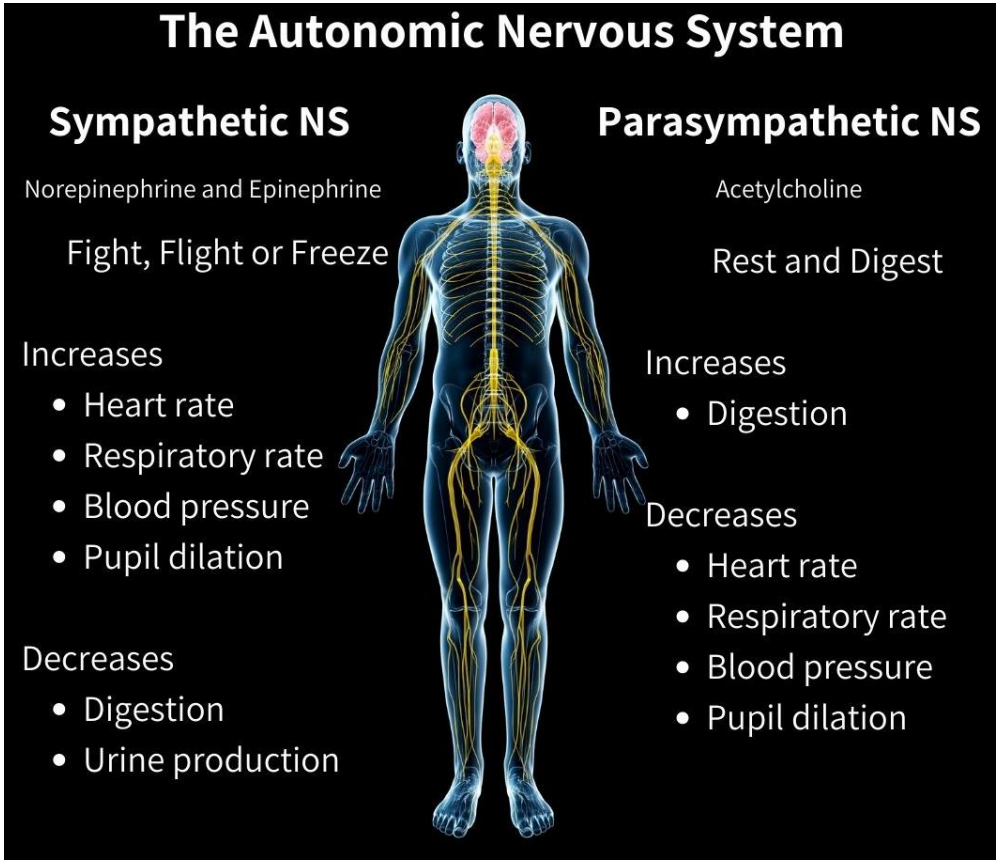
Poor sleep

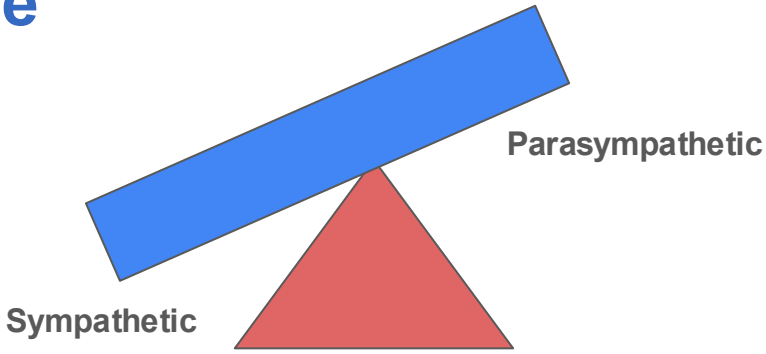


Commonly Follows:
Viral infections
Trauma
Pregnancy
Prolonged Bed Rest

General Treatment Considerations:
Increased salt (5-8g /day)
Increased fluid (>3L/day)
Compression Garments
Recumbent Exercise

POTS: A Dysautonomia Syndrome





POTS: Regulatory Injury

Treatment: It depends…

Hyperadrenergic: **beta blockers,** ivabridine

Neuropathic: **midodrine**, pyridostigmine

Hypovolumic: **fludrocortisone**, IV fluids

Brignole, Eur Heart Journal, 2018: 1883-1948

How Worried Should I Be? Take a Great History!

Suggests Non-Cardiac:

Younger Age

No Known Cardiac Disease

Syncope occurs after change in position

Syncope *after* exercising

Prodromal vagal symptoms

Presence of triggers (pain, intense stimulus)

Prolonged history of similar syncope episodes

LOC < 2 minutes

Suggests Cardiac:

Older Age

Structural cardiac disease

No prodrome or trigger

Syncope *while* exercising

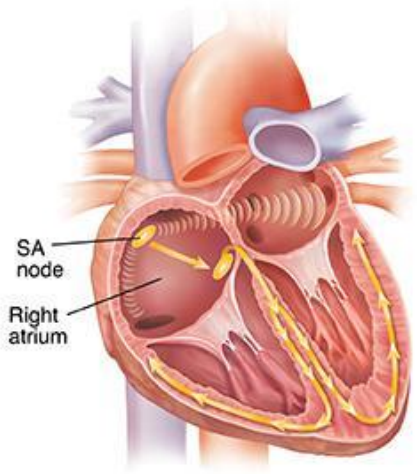
Syncope while supine

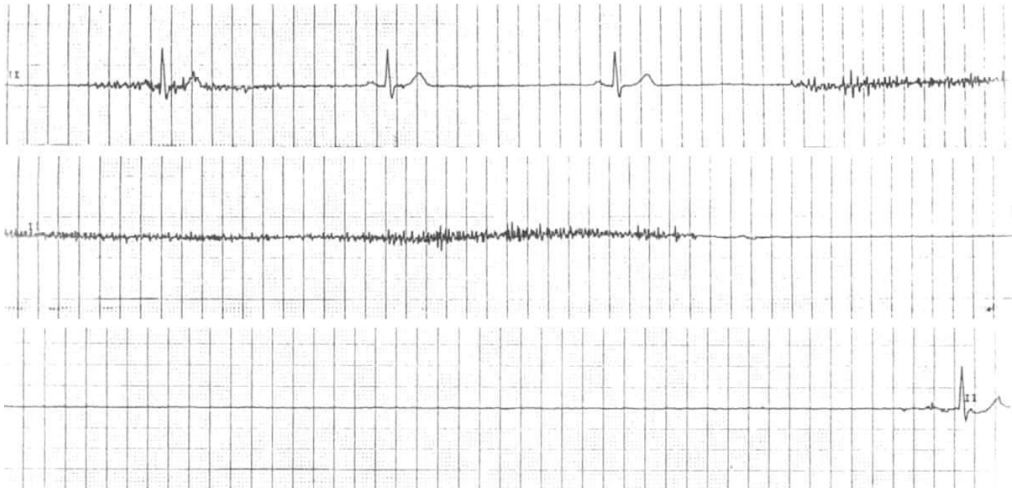
Rare episodes

Family history of sudden death

***Special Considerations!**

Cardiac: Bradycardia





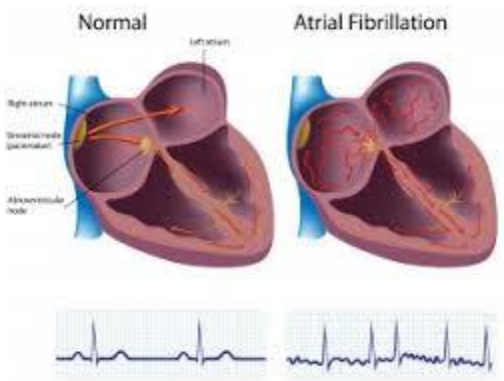
Sinus Node Dysfunction

High degree Heart Block

Trifascicular Block

Bundle Branch Block and Syncope

Cardiac: Supraventricular Tachycardia



Syncope with SVT is rare, except in atrial fibrillation

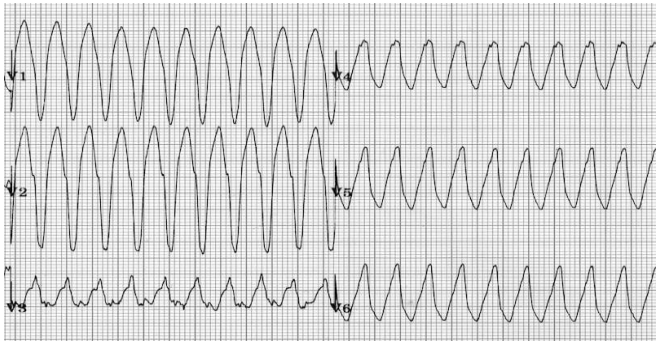
AF: not related to heart rate, but to **post-conversion pauses**.

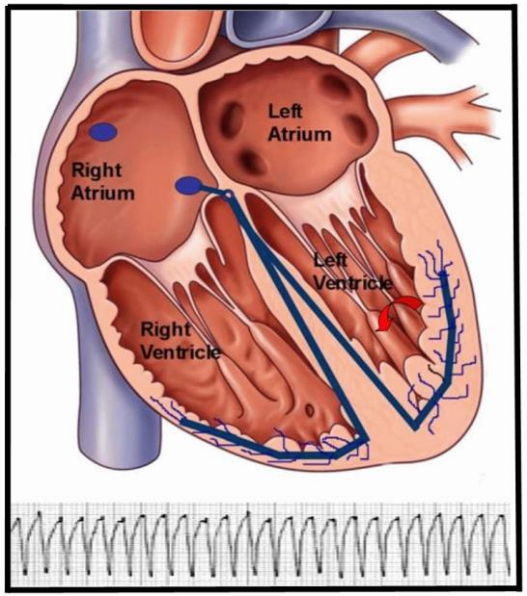
Rhythm control with ablation and/or anti-arrhythmics could help.

Most cases require a pacemaker (>3 second pauses **with symptoms**)

Shen et al, JACC, 2017: 70: e39-110

Cardiac: Ventricular Arrhythmias

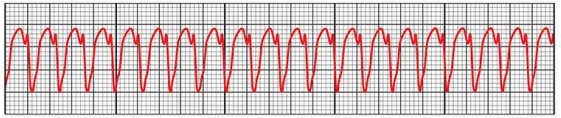


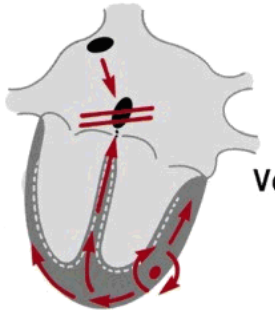


Monomorphic ventricular tachycardia

Normal ECG

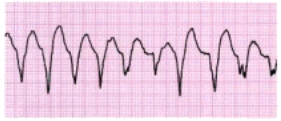
Monomorphic ventricular tachycardia

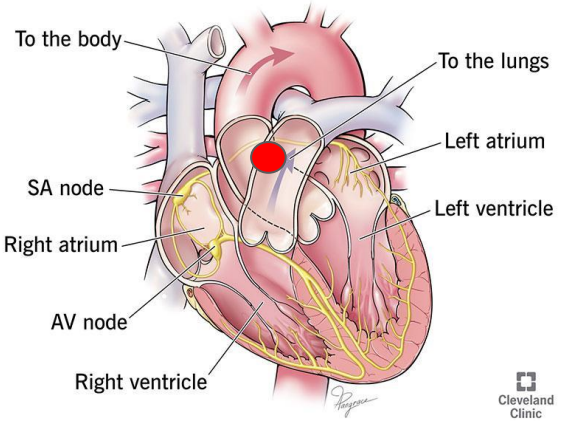




Normal Sinus Rhythm

Ventricular Tachycardia





Cardiac: Ventricular Arrhythmias

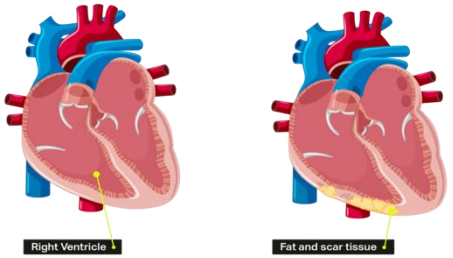
Most often in the setting of structural heart disease

- Cardiomyopathy (EF < 35% typically)
- Post-MI
- Infiltrative conditions (sarcoidosis, ARVC)
- Hypertrophic cardiomyopathy

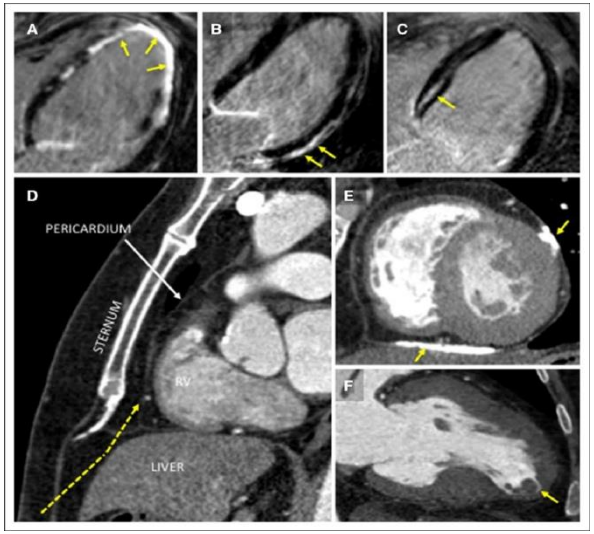
Outflow tract tachycardia

HR > 200 bpm is 4x as likely to cause syncope

Treatment: beta blockers, CCBs, anti-arrhythmics



Normal ARVC



Cardiac: Ventricular Arrhythmias

Channelopathies

Long QT syndrome

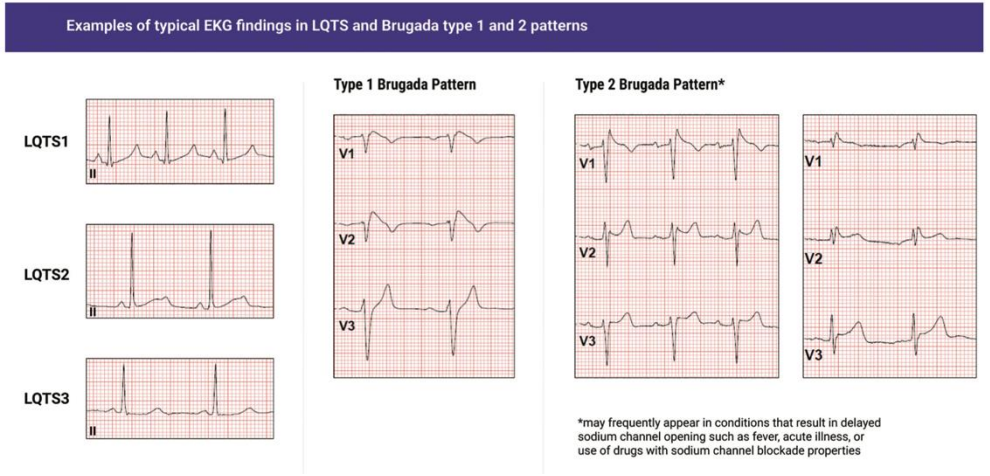
Brugada Syndrome

Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT)

Short QT Syndrome

Can find clues on the baseline ECG!

Character of syncope, family history are important.



Cardiac: Structural

Aortic Stenosis

Hypertrophic Cardiomyopathy

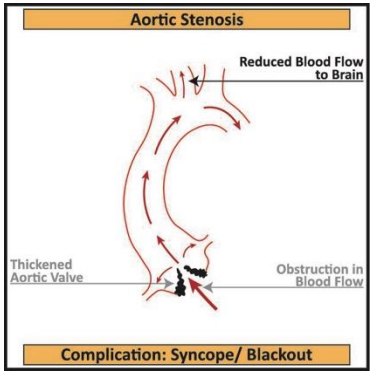
Pulmonary Embolism

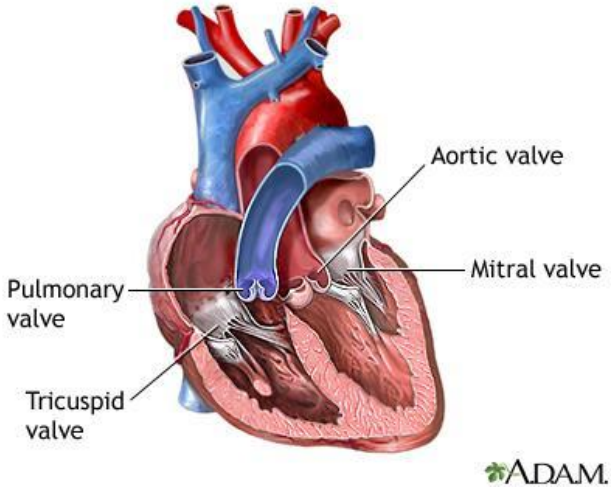
Severe Pulmonary Hypertension

Cardiac Masses

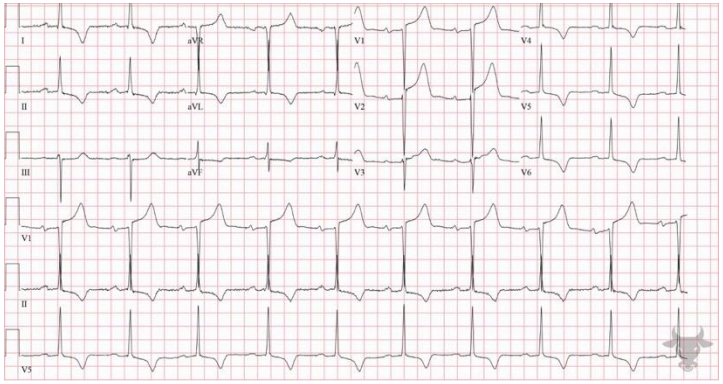
Cardiac Tamponade

Acute Aortic Dissection

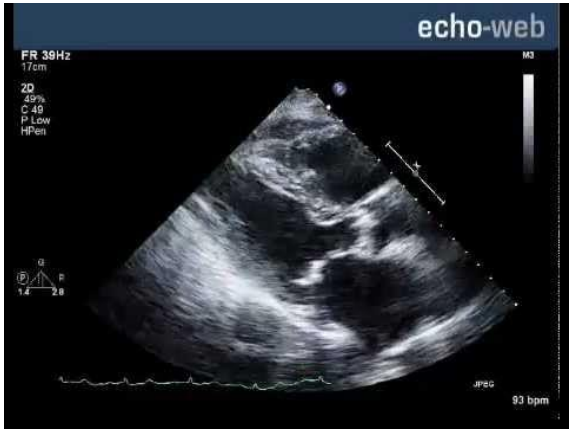




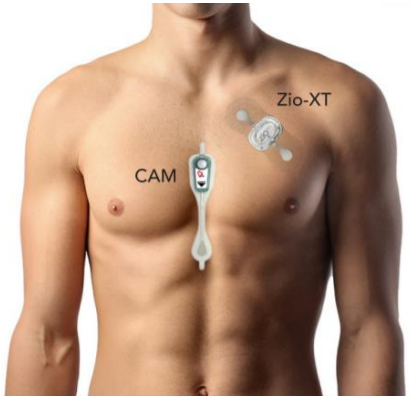
Initial Tests To Consider



Get an ECG



Consider an Echo



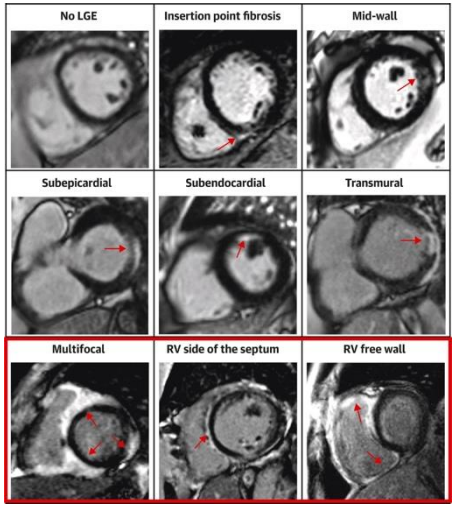
Consider Ambulatory Monitoring

Focus on History and Physical. ***The history is critical to the diagnosis.***

Basic testing can help refine the diagnosis.

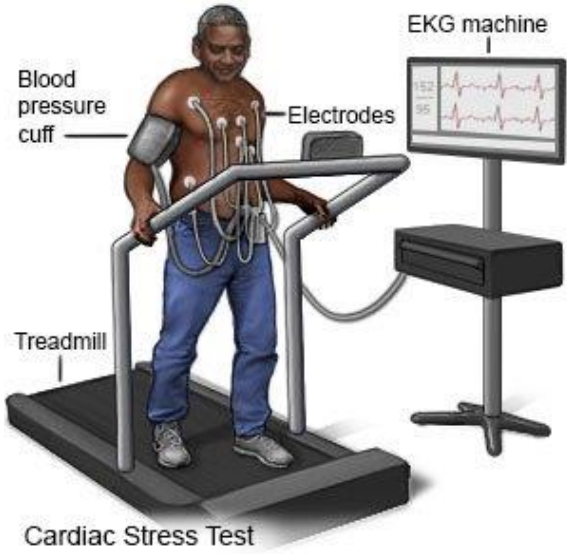
Advanced Testing: cardiac MRI, ILR, EP study, stress testing

Advanced Testing



Cardiac MRI

Implantable Loop Recorder



It's all about the History.

Initial testing refines the diagnosis.

Syncope and Driving

Syncope while driving: same evaluation as everyone else.

Vasovagal (neurally-mediated) most common cause.

Patients with vasovagal syncope less likely to have a MVA compared to cardiac causes (prodrome)

Patients with a history of syncope more likely to have a MVA

- Men > Women
- Age 18-35

Restrictions depend on the etiology, frequency, and whether driver is commercial.

- Usually 3-6 months, depending on etiology.
- If treated, can be shorter. If unknown, can be longer.
- Commercial drivers regulated by Federal Law.

Case 1

30yo female with recurrent syncope

Episodes occur while singing in her church choir.

Tunneled vision, nausea, sweating preceding episodes. Awakens upon going to ground. Feels “spent” for hours after.

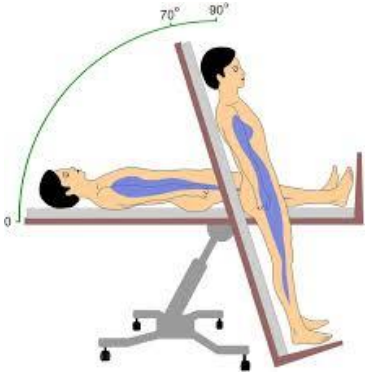
No medical history. No medications.

BP 110/60 HR 90. Normal exam. Normal ECG.

Initial thoughts?

Tilt Table Test

- Supine BP 120/70 mm Hg and HR 80 bpm.
- With passive tilt, BP over the first 5 min is 110-120/55-65 mm Hg and HRs are 80-100 bpm.
- At 8 min into the tilt, she reports nausea; 30 sec after that, she loses consciousness.
- Her BP reading obtained approximately 20 sec before fainting is 65/40 mm Hg and HR is 40 bpm.
- At the moment when she fainted, her HR was 30 bpm.
- Supine: prompt normalization of HR and BP.



Which one of the following is the best next step in her management?

1. Initiate midodrine
2. Implant a dual chamber pacemaker
3. Educate on mitigating triggers
4. Initiate Metoprolol.

Case 2

21yo woman presents to the ER following a sudden loss of consciousness while running from her apartment to a class on campus.

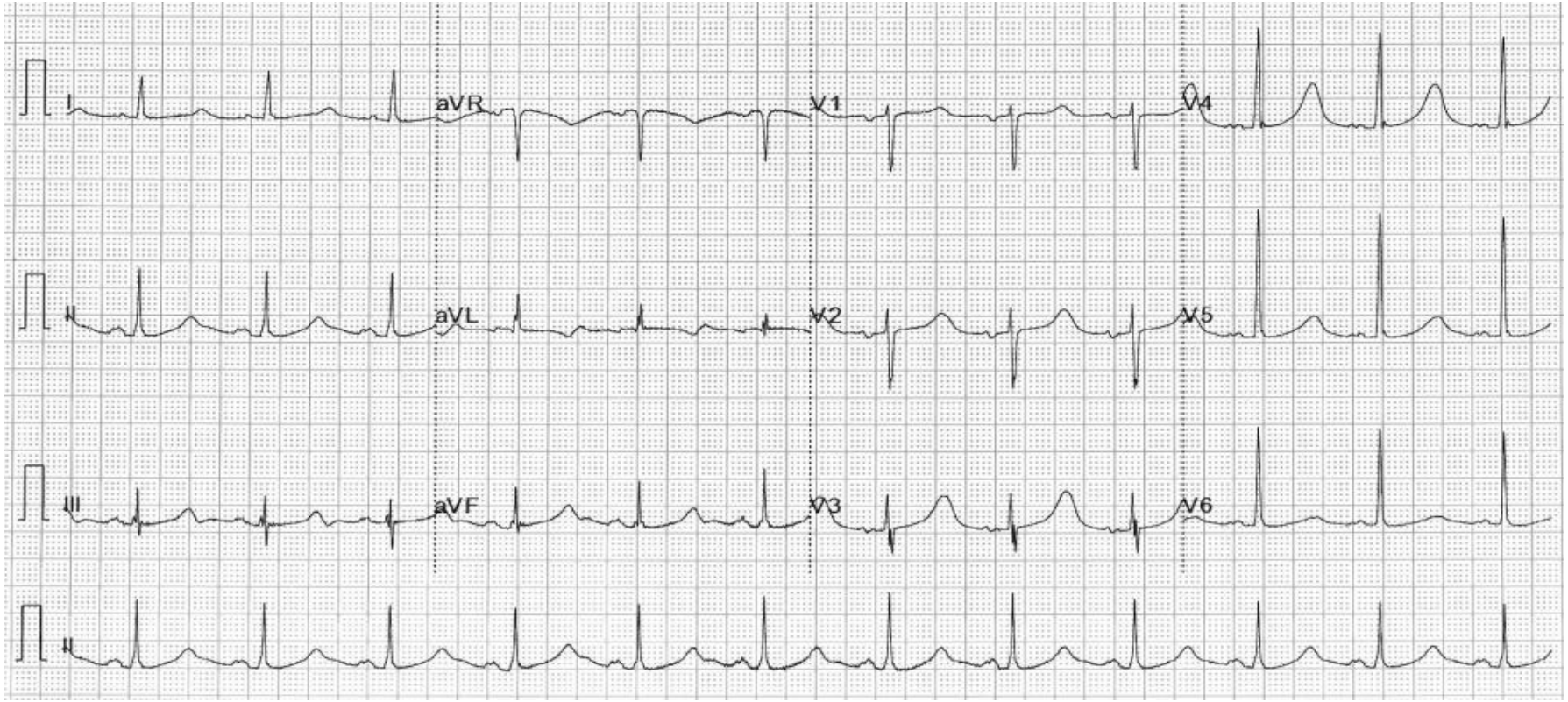
Hurt her left shoulder, left hip, left thigh. No witnesses. Awoke and was woozy.

No medications. No medical history.

Grandfather drowned when he was in his 20's. Father has seizures.

BP 120/70 HR 70. Normal exam. Normal labs.

One year ago, following an episode of pre-syncope, had a normal echo.



Next steps?

LQTS: Consider genetic testing

QTc: > 450ms in men; > 470ms in women.

Can lead to Torsades de Pointes.

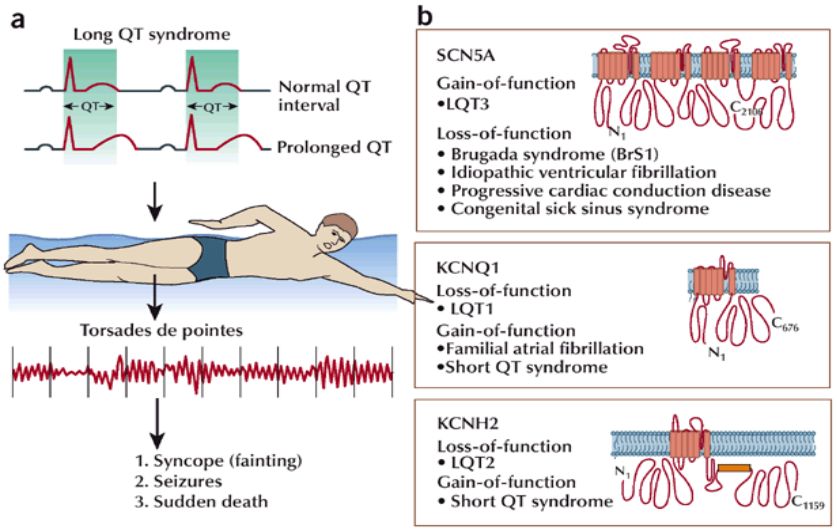
Triggers: competitive sports, sudden stressors, drugs.

Treatment:

Beta blockers are first line.

Avoid triggers.

Occasionally ICD.



Case 3

78yo man with PAF, T2D, and HTN presenting after a syncopal event.

Sudden onset, occurred while standing in his garage. Hit the front left side of his forehead. Happened 3 weeks ago, has otherwise felt well.

ER visit after event: no acute findings.

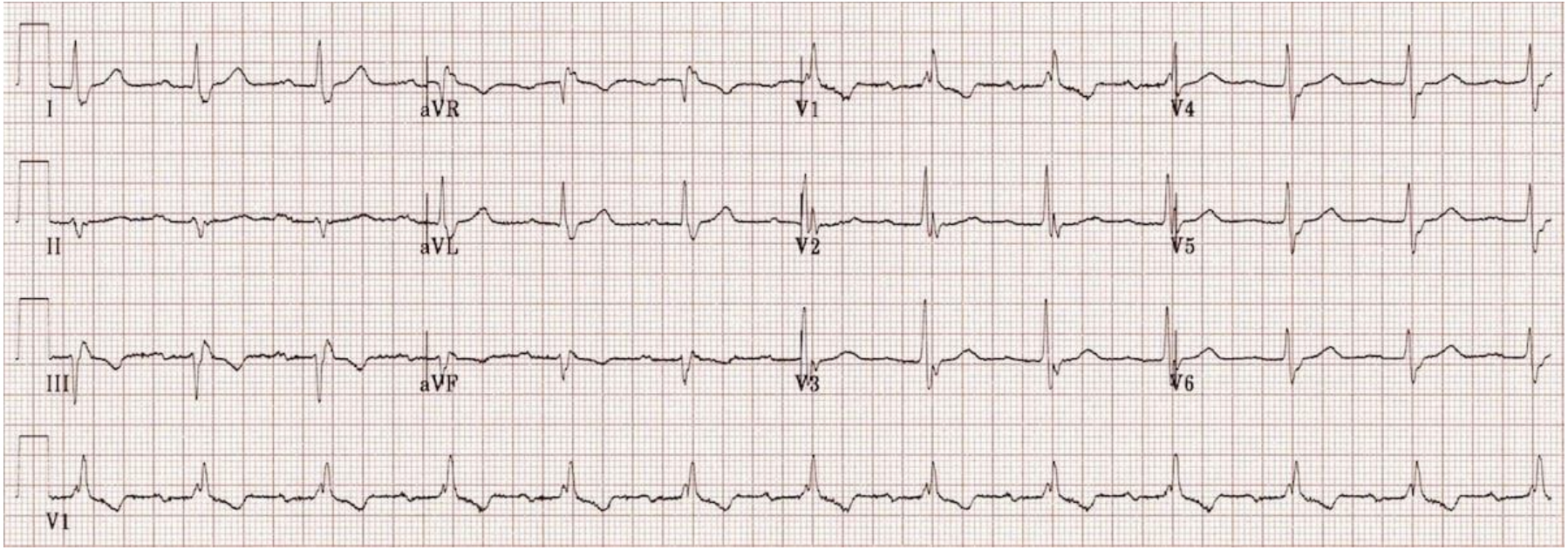
Medications: Amlodipine, Losartan, Eliquis

BP 138/80 HR 62 Exam unremarkable.

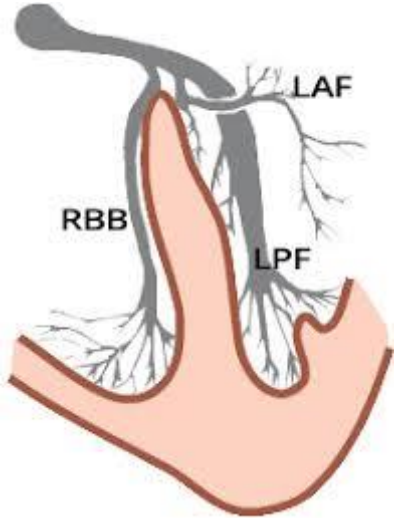
TTE: normal LV systolic function, normal valve function, mild biatrial enlargement.

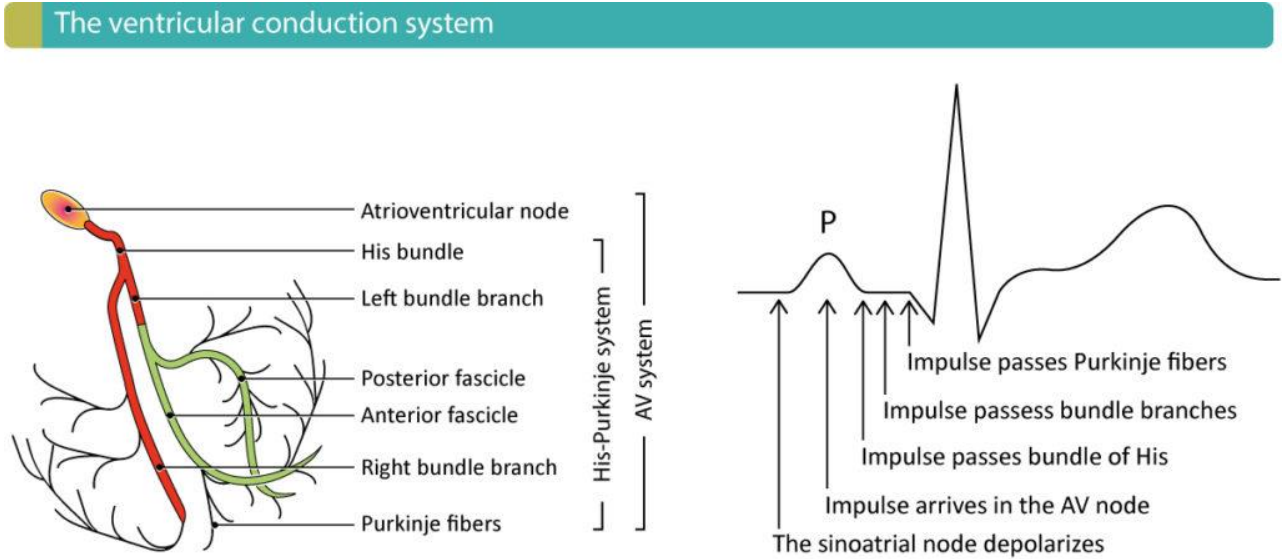
Labs normal in ER.

ECG in ER:



What is happening with a conduction delay?





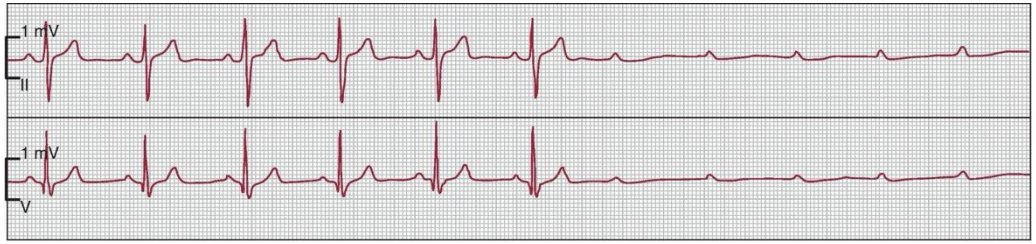
Temporal association between impulse transmission in the conduction system and the ECG waves

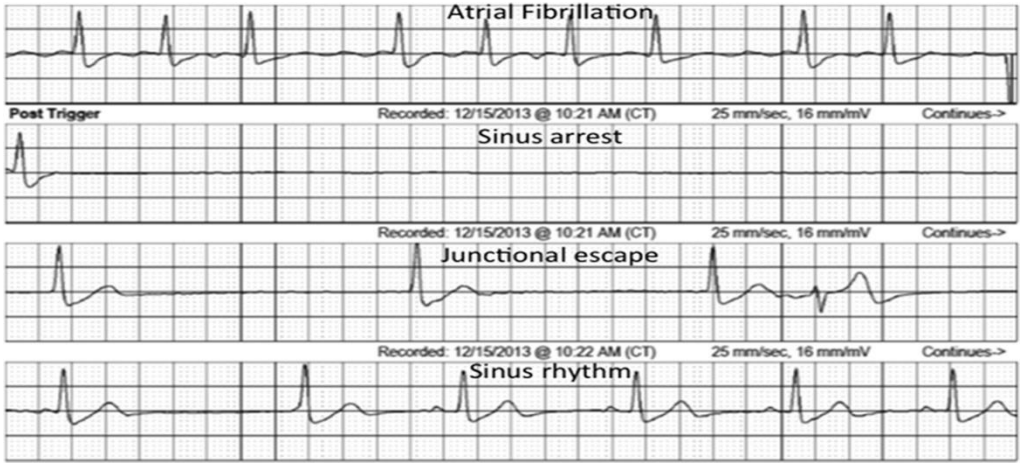
Next steps?

Zio showed transient Complete Heart Block (CHB).

Referral for pacemaker.

Note differences between transient CHB and Sick Sinus Syndrome (SSS)





Thank you!

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