

Syncope

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Grand Rounds
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Objective

- What is Syncope?
- Types of Syncope?
- Do they require hospital admission?
- When to call the Cardiologist?
- Testing options?



Syncope - What is it?

- Symptom that presents with an abrupt, transient, complete loss of consciousness
- associated with inability to maintain postural tone
- rapid and spontaneous recovery
- Presumed mechanism is cerebral hypoperfusion.
- There should not be clinical features of other non-syncope causes of loss of consciousness
 - seizure, antecedent head trauma, or apparent loss of consciousness (i.e., pseudosyncope)

Prevalence and Prognosis

- Prevalence depends upon clinical setting and age.
- Incidence increases with age >5% over the age of 70.
- Prognosis is excellent if there is no structural heart disease.
- Recurrence is 1/3 in 3 years and recurrence is dependent upon how many episodes you have had and NOT age, sex or tilt table results.
- 30% with minor trauma
- 5% major trauma
- 30% trauma if carotid disease and elderly

Pathophysiology

- The pathophysiology of syncope involves interaction between the circulatory system and the ANS.
- Syncope and near syncope is caused by brain hypoxia, usually as a result from decreased cerebral perfusion pressure.
- Cerebral autoregulation is able to maintain normal perfusion over a range of CPP or MAP (60 to 160 mmHg)
- On standing gravity begins to pool blood to the distensible veins and results in decreased venous return and reduced cardiac output.
- Vasodepressor and cardio inhibitory mechanisms

Types of Syncope

Neural or Reflex is the most common ~ 60-70%

Several subtypes:

- Vasovagal
- Situational
- Carotid sinus hypersensitivity
- Post exertional

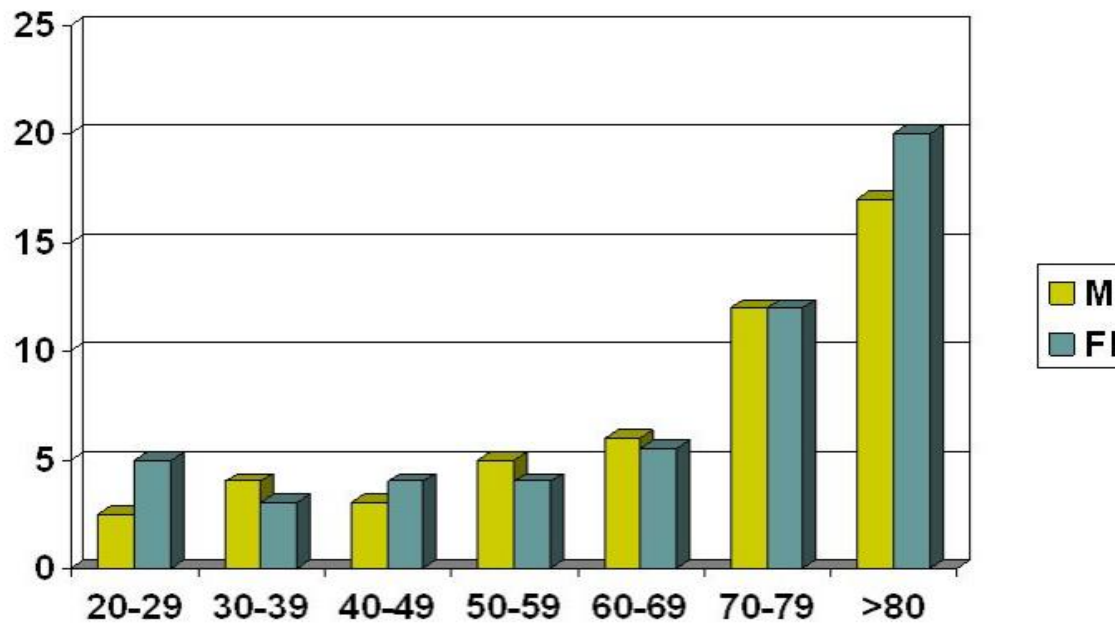
Types of Syncope

Cardiac Induced is the most dangerous form ~ 10-20%

Orthostatic Hypotension is the least common ~ 10%

- multiple system atrophy (MSA), familial dysautonomia, primary autonomic failure

INCIDENCE OF SYNCOPE IN FRAMINGHAM STUDY



Pathophysiology

- Involves interaction between the circulatory system and the ANS.
- Syncope and near syncope is caused by brain hypoxia
 - usually as a result from decreased cerebral perfusion pressure
- Cerebral autoregulation is able to maintain normal perfusion over a range of CCP or MAP (60-160 mmHg)
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- Vasodepressor and cardio inhibitory mechanisms

Types of TRUE Syncope

1. Neurally mediated (the most common – 60-70% of cases)

- Vasovagal syncope
- Situational syncope
- Carotid artery hypersensitivity
- Postexertional syncope

2. Cardiac (the most worrisome – 10-15% of the cases)

3. Orthostatic hypotensive (least common – 10%)

(multiple system atrophy, familial dysautonomia, primary autonomic failure)

Neurally Mediated Syncope

- Autonomic reflexes that respond inappropriately, leading to vasodilation and bradycardia
- Premonitory symptoms are common
- These could include extreme lightheadedness; visual sensations, such as “tunnel vision” or “graying out”; and variable degrees of altered consciousness without complete loss of consciousness.
- However, this may not be the case in one-third of patients, especially in elderly patients, who may not recognize or remember the warning symptoms
- NEVER OCCURS IN THE SUPINE POSITION

Vasovagal Syncope

- VASOVAGAL MOST COMMON
- Triggers are emotional stress, dehydration, prolonged sitting or standing.
- Can occur in elderly
- Symptoms can persist after recovery
- If >60 seconds then one can see clonic movements and incontinence.
- MECHANISM: SMALL HEART AND HYPERCONTRACTILITY

Situational Syncope

- Laughing, micturition, defecation and weight lifting
- The reflex may be initiated by a receptor on the visceral wall (eg, the bladder wall) or by straining that reduces venous return.

Carotid artery hypersensitivity

- Still rare but occurs after age 50
- Spontaneous carotid sinus hypersensitivity syndrome
- Induced carotid sinus hypersensitivity syndrome
- Features
 - Vasodepressor: >50 mmHg drop in systolic blood pressure
 - Cardioinhibitory: >3 second pause
 - Combination

Post exertional Syncope

- Following exercise, venous return diminishes while catecholamine surge is still present.

Orthostatic Hypotension

- Orthostatic intolerance
 - A syndrome consisting of a constellation of symptoms
 - Frequent, recurrent, or persistent lightheadedness, palpitations, tremulousness, generalized weakness, blurred vision, exercise intolerance, and fatigue upon standing.
- Due to autonomic failure with inability to increase peripheral resistance or heart rate with orthostasis.
- Defined: >20 mmHg drop in systolic or 10 mmHg drop in diastolic blood pressure on standing
- Individuals with orthostatic intolerance have >1 of these symptoms associated with reduced ability to maintain upright posture.

- Orthostatic hypotension (OH) - A drop in systolic BP of >20 mmHg or diastolic BP of >10 mm Hg with assumption of an upright posture.
 - Initial (immediate) OH - within 15 s after standing, with presyncope or syncope.
 - Classic OH - within 3 min of assuming upright posture.
 - Delayed OH - takes >3 min of upright posture to develop. The fall in BP is usually gradual until reaching the threshold.
- Neurogenic OH - A subtype of OH that is due to dysfunction of the autonomic nervous system and not solely due to environmental triggers (e.g., dehydration or drugs).
 - due to lesions involving the central or peripheral autonomic nerves.

POTS

- Postural (orthostatic) tachycardia syndrome
- A clinical syndrome usually characterized by all of the following:
 1. Frequent symptoms that occur with standing (e.g., lightheadedness, palpitations, tremulousness, generalized weakness, blurred vision, exercise intolerance, and fatigue) and
 2. An increase in heart rate of >30 bpm during a positional change from supine to standing (or >40 bpm in those 12–19 y of age) and
 3. Absence of OH (>20 mm Hg reduction in systolic BP).
- Autonomic dysfunction affects peripheral vascular resistance, which fails to increase in response to orthostatic stress. This autonomic dysfunction does not affect the heart, which manifests a striking compensatory increase in rate

Predictors of recurrent syncope in older adults

- aortic stenosis
- impaired renal function
- atrioventricular (AV)
- Left bundle-branch block
- male sex
- chronic obstructive pulmonary disorder
- heart failure (HF), atrial fibrillation (AF)
- advancing age
- orthostatic medications, with a sharp increase in incidence after 70 years of age.

Cardiac Syncope

- There are three forms of cardiac syncope:
 - Structural heart disease with cardiac obstruction
 - Ventricular tachycardia (structural heart disease or primary electrical disease)
 - Bradyarrhythmias (degenerative conduction disease, drug effect, structural heart disease)

Historial features associated with increased likelihood of cardiac cause

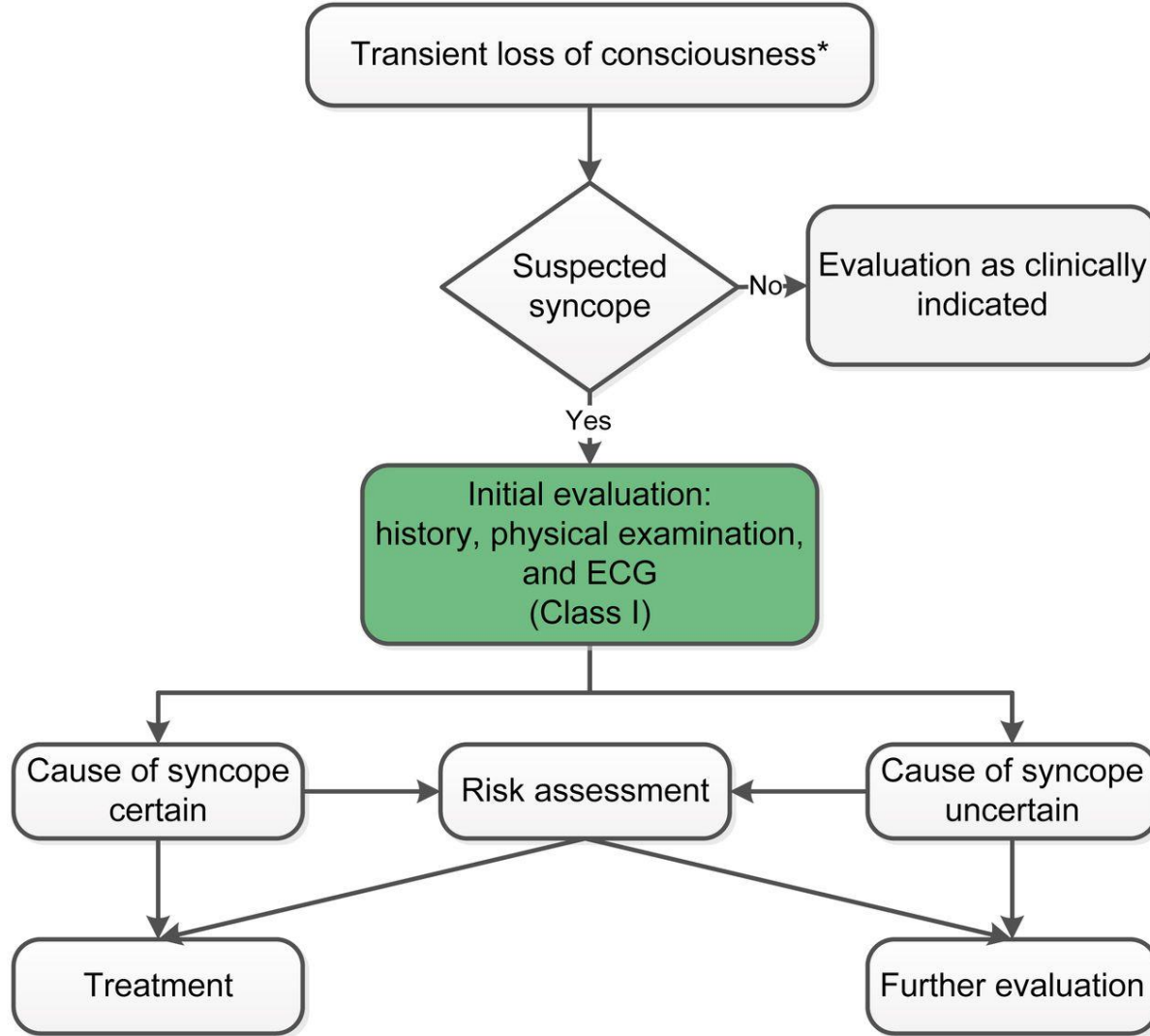
- Age >60
- Male
- H/o congenital heart disease
- Syncope with exertion
- Abnormal cardiac examination
- Presence of known IHD with previous arrhythmia or low EF.
- Abnormal cardiac testing/implantable devices

CONTEXT	CARDIAC	NONCARDIAC
Older age (>60 years), male gender	✓	
Known heart disease (e.g., ischemic, previous arrhythmia)	✓	
Family history of sudden death	✓	
Prodrome (e.g., nausea, vomiting, warmth)		✓
Specific triggers (e.g., dehydration, pain, distress)		✓
Situational triggers (defecation, deglutition, coughing)		✓

Don't Miss!

CONTEXT	CARDIAC	NONCARDIAC
Older age (>60 years), male gender	✓	
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Family history of sudden death	✓	
Prodrome (e.g., nausea, vomiting, warmth)		✓
Specific triggers (e.g., dehydration, pain, distress)		✓
Situational triggers (defecation, deglutition, coughing, laughing)		✓
Preceding palpitations	✓	
Syncope during exertion	✓	
Syncope while supine	✓	
Low number of episodes	✓	
Post event fatigue, somnolence		✓

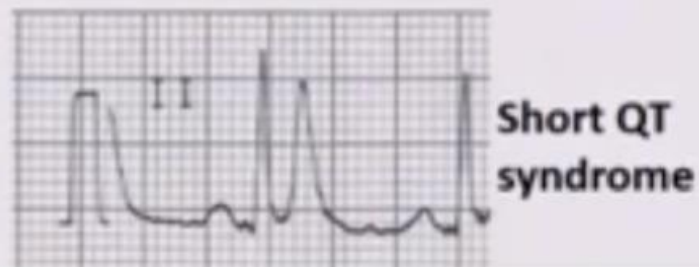
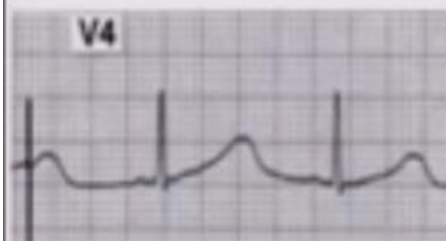
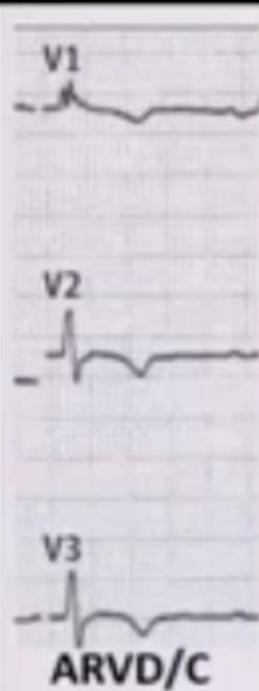
- The initial evaluation includes but is not limited to a thorough history, physical examination, and ECG.



HISTORY

- The history should aim to identify the prognosis, diagnosis, reversible or ameliorable factors, comorbidities and medication use.
- Cardiac syncope carries a significantly worse prognosis than does neurally mediated syncope.
- The diagnostic history focuses on the situations in which syncope occurs, prodromal symptoms that provide physiological insight, patient's self-report, bystander observations of the event and vital signs, and post-event symptoms.
- Video recordings are helpful when available.
- Time relationship to meals and physical activities and duration of the prodrome are helpful in differentiating neurally mediated syncope from cardiac syncope.

- Comorbidities and medication use are particularly important factors in older patients.
- A history of past medical conditions should be obtained, particularly with regard to the existence of preexisting cardiovascular disease.
- A family history should be obtained.unexplained death.
- The physical examination should include determination of orthostatic blood pressure and heart rate changes in lying and sitting positions, on immediate standing, and after 3 minutes of upright posture.
- Careful attention should be paid to heart rate and rhythm, as well the presence of murmurs, gallops, or rubs that would indicate the presence of structural heart disease.
- A basic neurological examination should be performed, looking for focal defects or other abnormalities that would suggest need for further neurological evaluation or referral.

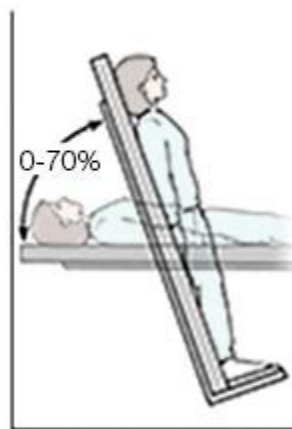


Who gets imaging?

Class IIa	B-NR	Transthoracic echocardiography can be useful in selected patients if structural heart disease suspected.
Class IIb	B-NR	CT or MRI may be useful in selected patients if cardiac etiology suspected.
Class III: No benefit	B-NR	Routine cardiac imaging is not useful unless cardiac etiology suspected based on initial evaluation, including history, physical exam, or ECG.

Indications for Hospitalization

- Patients should be hospitalized if they have severe hypovolemia or bleeding, or if there is any suspicion of heart disease by history, examination, or electrocardiography, including:
 - History of heart failure, low ejection fraction, or coronary artery disease
 - An electrocardiogram suggestive of arrhythmia
 - Family history of sudden death
 - Lack of prodromes; occurrence of physical injury, exertional syncope, syncope in a supine position, or syncope associated with dyspnea or chest pain.





Scenario 1 – Mr. S

- 66 year old Gentleman
- History of uncontrolled hypertension and labile pressures
- Sporadic episodes of Near syncope
- Tilt table test

Time

Supine Baseline:

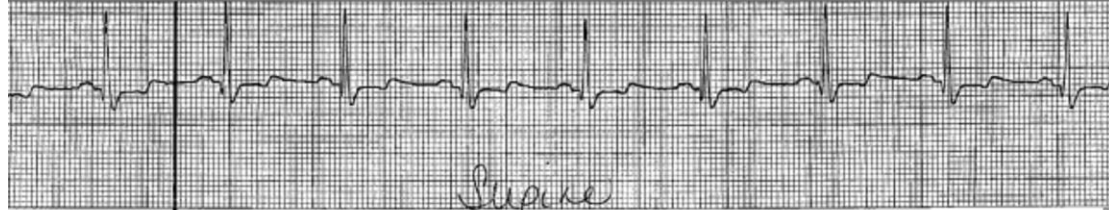
	Pulse	B.P	Symptoms/ Comments
2:00	67	179/94	Pt did not take any meds this AM
4:00	67	162/90	Pt does not take any BP meds
6:00	67	172/88	
8:00	66	162/86	
10:00	67	163/81	Denies symptoms

Upright:

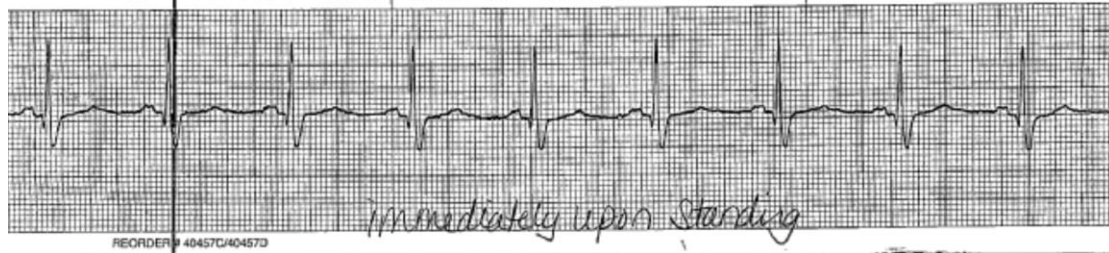
	Pulse	B.P	
2:00	70	85/65	Dizziness immediately upon standing
4:00	70	104/66	
6:00	72	102/79	Dizziness
8:00	70	89/69	
10:00	76	96/62	
12:00	76	86/59	
14:00	76	61/46	Dizziness continues
16:00	76	69/52	
18:00	78	69/52	
20:00	81	56/41	
22:00	80	58/47	Discomfort in back and chest
24:00	80	67/44	Dizziness
26:00	80	65/56	
28:00	81	69/54	
30:00	77	60/48	Dizziness
32:00	80	61/48	
34:00	80	69/59	
36:00	80	57/47	Nausea, cool, clammy
38:00	66	60/46	Pt stated to discontinue test due to
40:00			back pain

Return to Supine:

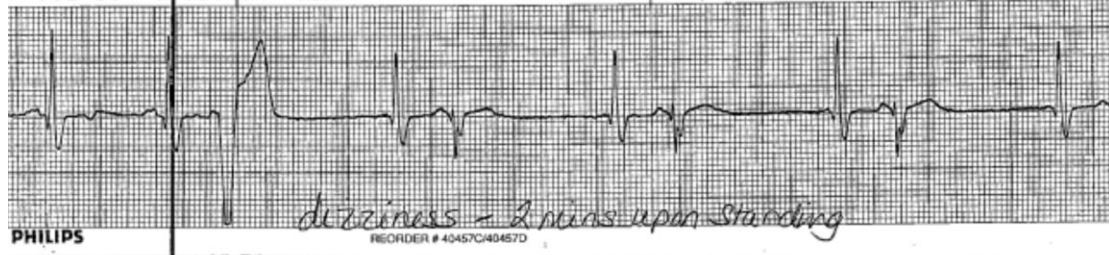
	Pulse	B.P	Symptoms/ Comments
Immediate:	67	122/71	Back feeling better
2:00	66	128/75	Dizziness gone
4:00	65	115/73	
6:00	66	120/74	Feeling better



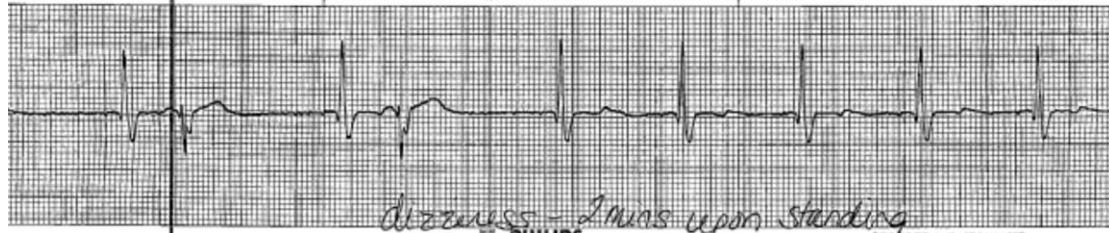
09 JUL 18 08:48:14 HR68 LEAD II AUTOGAIN DELAYED

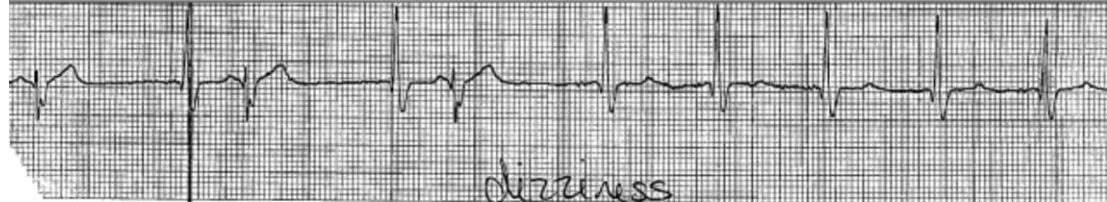


9 JUL 18 08:50:34 HR43 LEAD II AUTOGAIN DELAYED

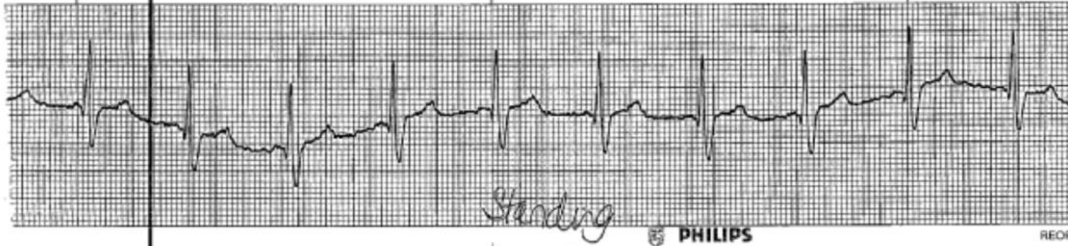


09 JUL 18 08:50:46 HR70 LEAD II AUTOGA





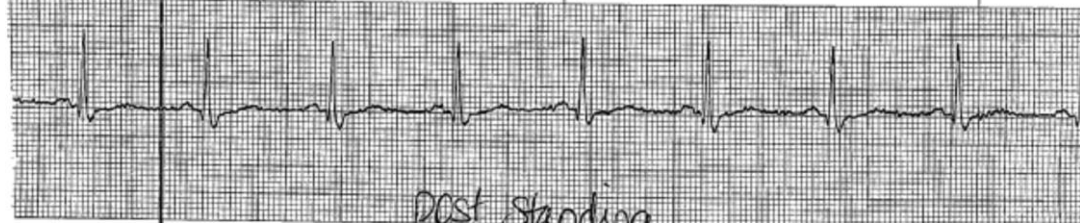
09 JUL 18 09:32:00 HR81 LEAD II AUTOGAIN DELAYED



09 JUL 18 09:39:33 HR68 LEAD II AUTOGAIN DELAYED



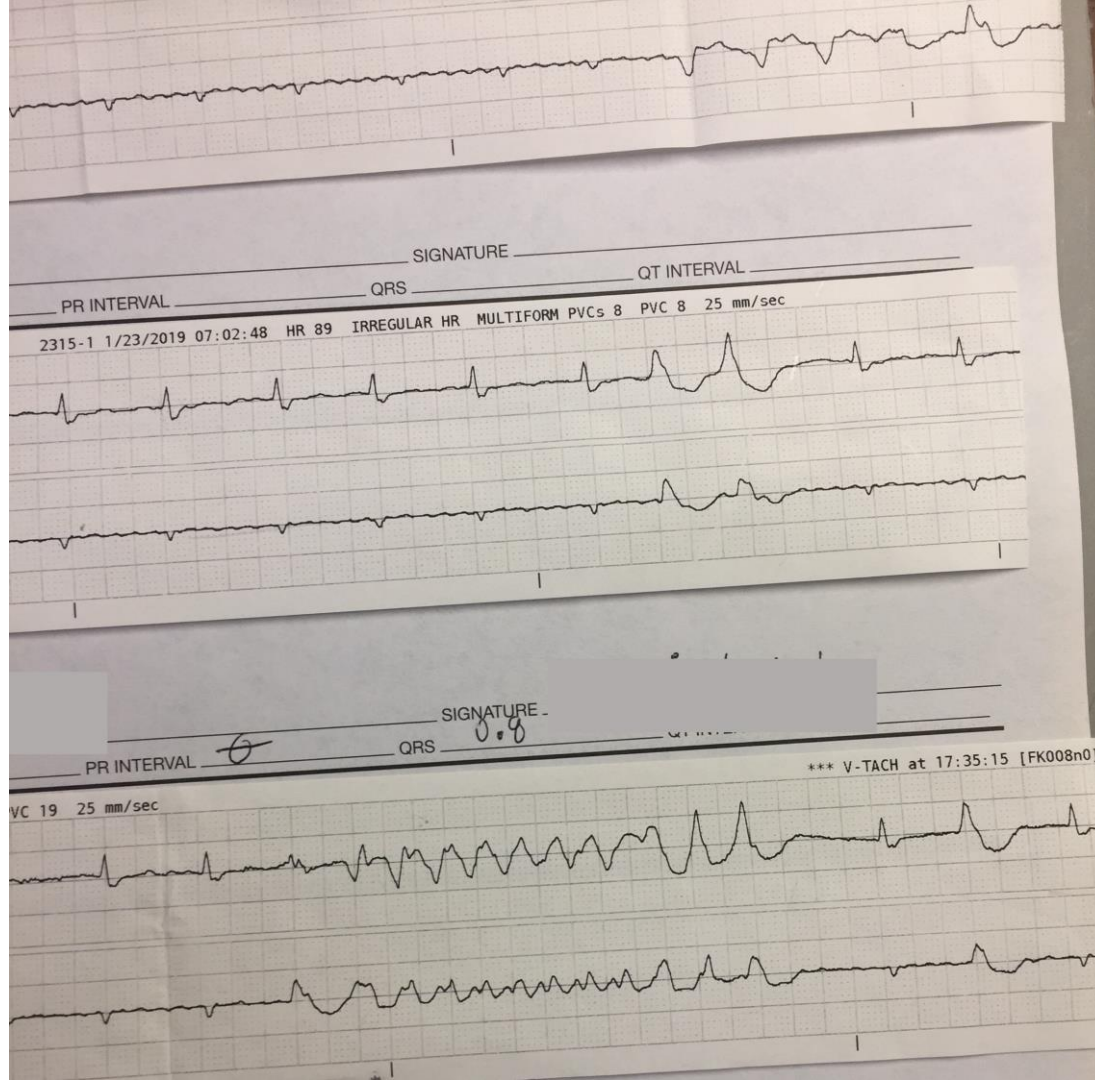
09 JUL 18 09:46:20 HR67 LEAD II AUTOGAIN DELAYED



Impression

- The head up tilt table test was indeterminate because patient developed nausea and pain leading to early termination of the test.
- However we did see a delayed vasodepressive/orthostatic response with greater than 20 mm drop in his systolic blood pressure leading to symptoms of dizziness.

Case 2



Case 3 – 78 Female

Baseline: Supine for 10 mins

Time	Pulse	BP	Symptoms/Comments
2 mins	65	203/92	look all regularly scheduled meds headache, mild
4 mins	66	189/84	
6 mins	69	195/87	
8 mins	64	168/89	
10 mins	64	167/83	denies symptoms

Test: Upright for 46 minutes

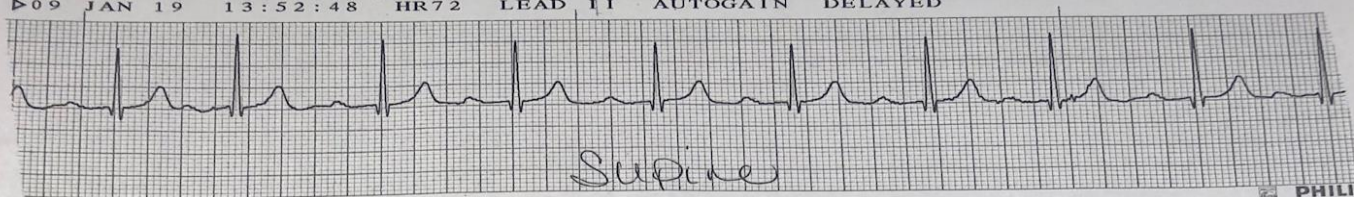
Time	Pulse	BP	Symptoms/Comments
2 mins	75	158/97	denies symptoms
4 mins	77	173/84	
6 mins	72	155/92	
8 mins	77	164/84	
10 mins	77	158/92	denies symptoms
12 mins	77	161/92	
14 mins	76	172/100	
16 mins	76	157/84	
18 mins	85	152/86	
20 mins	83	159/96	denies symptoms
22 mins	81	163/82	
24 mins	87	156/96	
26 mins	84	163/95	
28 mins	91	149/106	
30 mins	100	117/77	feeling sleepy
32 mins	104	122/88	feeling hot/anxious
34 mins	19	62/38	syncope episode
36 mins			
38 mins			
40 mins			
42 mins			
44 mins			
46 mins			
48 mins			
50 mins			

Returned to Supine Position for 10 minutes

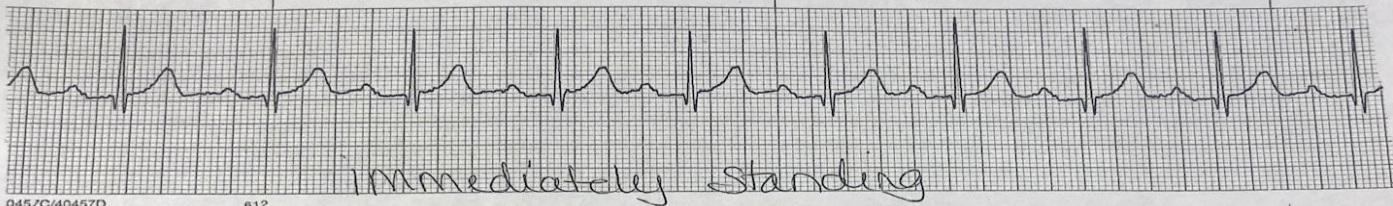
2 mins	68	82/58	coming around / lightheaded
4 mins	67	115/77	alert + oriented
6 mins	72	140/90	
8 mins	70	182/94	
10 mins			denies symptoms

Case 3

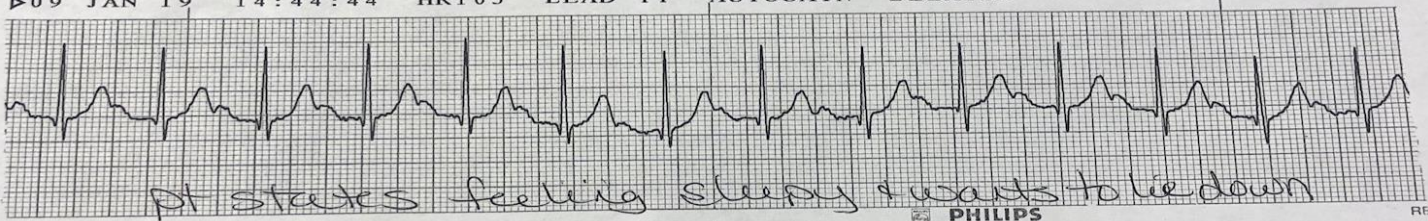
▷09 JAN 19 13:52:48 HR72 LEAD II AUTO GAIN DELAYED



▷09 JAN 19 14:07:03 HR76 LEAD II AUTO GAIN DELAYED



▷09 JAN 19 14:44:44 HR105 LEAD II AUTO GAIN DELAYED



▷09 JAN 19 14:47:06 HR77 LEAD II AUTO GAIN DELAYED



Case 3

