IMPROVING THE EVALUATION AND MANAGEMENT OF SYNCOPE

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DISCLOSURES

- No disclosures relevant to this topic

History & Exam

Testing

Treatment

DEFINITION: KEY ELEMENTS

GLOBAL CEREBRAL HYPOPERFUSION

TRANSIENT LOSS OF CONSCIOUSNESS AND POSTURAL TONE

RAPID AND BRIEF

SPONTANEOUS RECOVERY

VARIABLE PRODROMAL SYMPTOMS

CASE#1

History	26yo female with no significant PMH presents with first syncope in setting of heated argument with parents
Prodrome	none
Witnesses	arm shaking for ~2-3 min, urinary incontinence
Upon waking	confused, disoriented for >1 hour
Workup	not orthostatic, laboratories and ECG normal Exam with horizontal nystagmus, tongue bleeding

*WHAT DO YOU DO NEXT?

1. No further testing, discharge home
2. Echocardiogram
3. Head CT/MRI
4. Stress test
5. Start fludrocortisone

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Likely first time seizure

WEED OUT IMPOSTERS

Hypoglycemia

Hypoxia

Sleep Disorders: narcolepsy

Drop Attack: loss of postural tone without LOC

Coma: LOC without spontaneous recovery

Seizure: no cerebral hypoperfusion

TIA/stroke: may have vagal component early on

CASE#2

HISTORY	26yo female with no significant PMH presents with first syncope in setting of heated argument with parents
PRODROME	Lightheaded, no palpitations/chest pain/dyspnea
WITNESSES	Some arm twitching, looked pale
UPON WAKING	Nauseated , fatigued, better after 15 minutes
WORKUP	Not orthostatic, normal exam/laboratories/ECG

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Vasovagal/neurocardiogenic syncope

NMS VS SEIZURE

	NMS	Seizure
Occurs supine	Uncommon	***Common***
Typical prodrome- warm, clammy	***Common***	Uncommon- occasional aura
Pallor	***Common***	Uncommon
Tongue biting	Uncommon- at the tip	Common- on the sides
Eye deviation	Fixed/upward	Lateral deviation
Incontinence	Uncommon	***Common***
Muscle movement/tone	Pleomorphic/flaccid	Rhythmic and generalized/tonic
Duration of LOC	< 1 minute	***Often several minutes***
Postictal symptoms	Brief fatigue, nausea, clammy	Confusion

Adapted from Sheldon Cardiol Clin 2015 and ESC 2009 guidelines

HISTORY

A detailed history is the FIRST and MOST important tool in diagnosis

- Severity of injury sustained during syncope does ***NOT*** correlate with etiology of syncope
  - Manifestation of activity around time of syncope

HISTORY

Circumstances	• Time of day, relation to eating, emotional or painful stimulus, location, atmosphere, going to bathroom
Position	• Standing vs supine, change in posture
Activity	• During or after exercise, arm movement, quick head turning
Prodrome	• Aura, nausea, diaphoresis, palpitations
Recovery	• Rapid recovery or prolonged symptoms

EGSYS SCORE

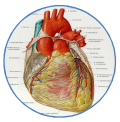
Predictors of cardiac cause of syncope

Variable	OR (95% CI)	Score
Palpitations	64.8 (8.9 to 469.8)	4
Heart disease or abnormal ECG	11.8 (7.7 to 42.3)	3
Syncope during exertion	17.0 (4.1 to 72.2)	3
Syncope while supine	7.6 (1.7 to 33.0)	2
Precipitating factors	0.3 (0.1 to 0.8)	-1
Autonomic prodrome	0.4 (0.2 to 0.9)	-1

Score >3 Suggestive of cardiac cause of syncope

Adapted from Del Rosso Heart 2008

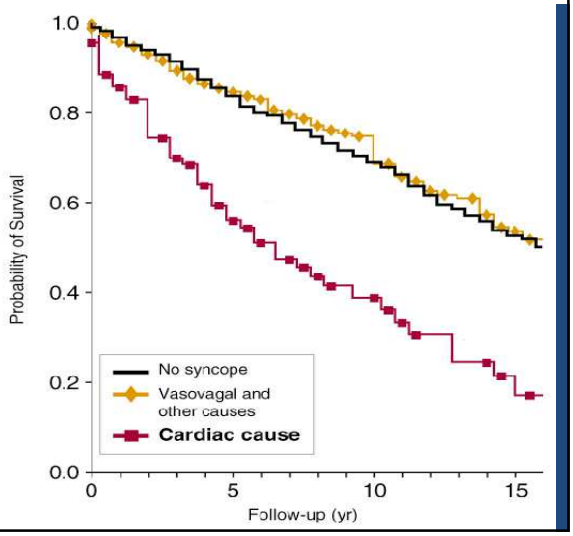
Excellent Review: Albassam JAMA 2019:321



CARDIAC CAUSE OF SYNCOPE

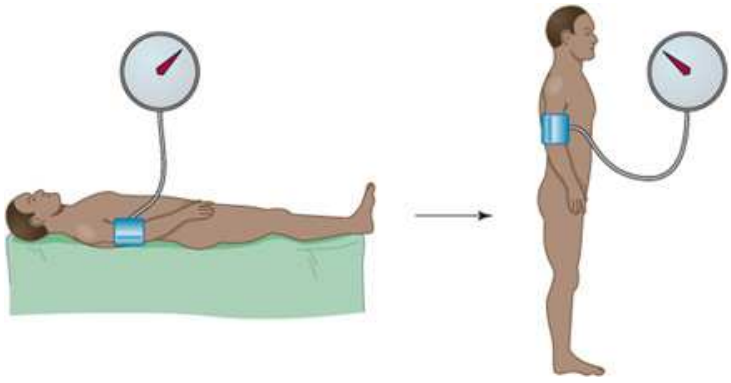
- Etiology of syncope has a significant impact on mortality
  - Cardiac vs non-cardiac syncope
- Appropriate, timely therapy has great potential to prevent morbidity and mortality

Soteriades *N Engl J Med.* 2002



EXAM

- Orthostatic vital signs
- Tongue biting or focal neurologic deficit
- Murmurs- examine in 2 positions
  - Sitting up and leaning forward
  - Left lateral recumbent
  - PMI-point of maximal impulse- diffuse or laterally displaced?
- Injury pattern- able to brace their fall?- indicates prodrome
- Peripheral edema- symmetric or asymmetric?



May take up to 3 minutes for BP drop

HOW TO PERFORM ORTHOSTATICS

Diagnostic:

- Symptoms reproduced
- Fall in SBP >20 mmHg or DBP >10 mmHg
- Decrease in SBP to <90 mmHg

Suggestive:

- No symptoms
- Fall in SBP >20 mmHg or DBP >10 mmHg
- Decrease in SBP to <90 mmHg
- Symptoms from history are consistent with orthostatic hypotension

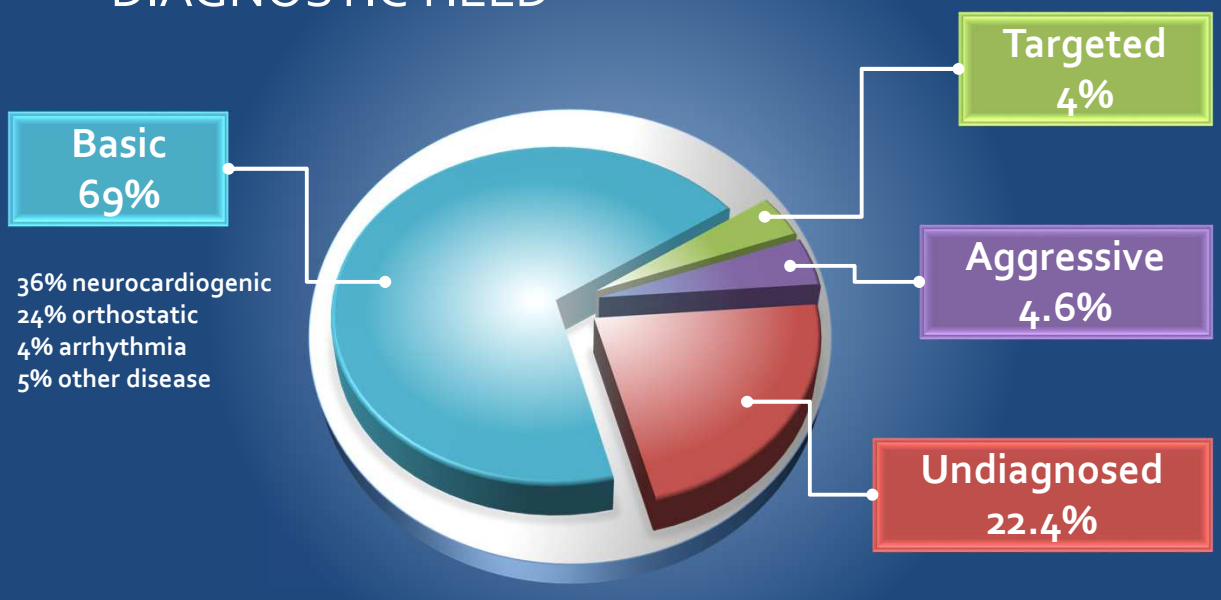
ESC Syncope guidelines Eur Heart J. 2018;1183

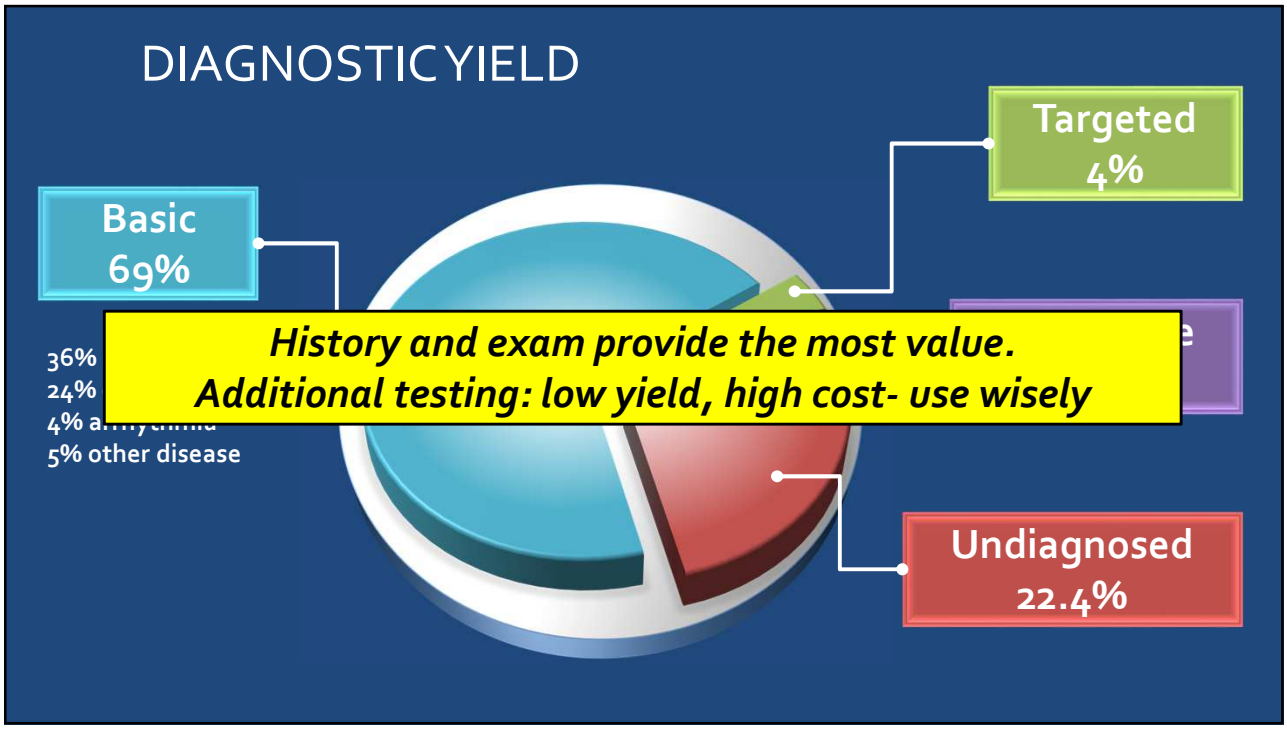
DIAGNOSTIC YIELD

- 650 consecutive patients presenting to ER with syncope as chief complaint followed for up to 18 months
- History and physical exam including CSM, ECG, basic labs
- Targeted tests (e.g. echo, CTA) when clinically suspected
- If syncope still unexplained, then more aggressive workup
  - Holter, event monitor, Tilt table test, SAECG, EP study

Sarasin AM J Med 2001

DIAGNOSTIC YIELD

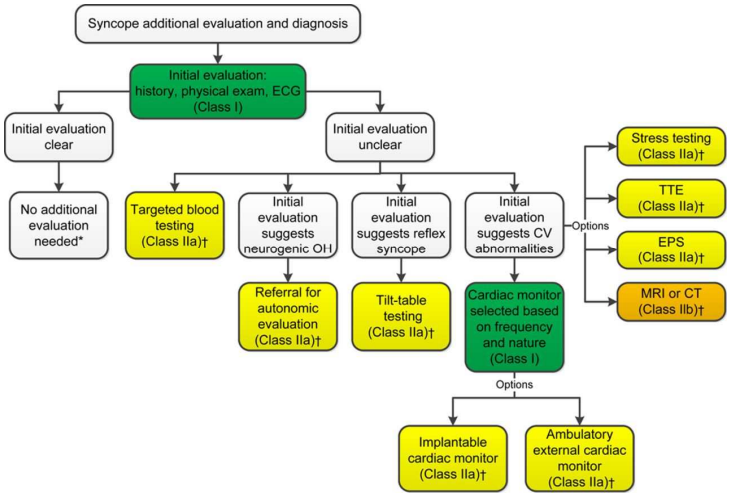




WHEN TO DO ANCILLARY TESTING

TESTING ALGORITHM

- Selective testing based on key elements of history, exam, and ECG



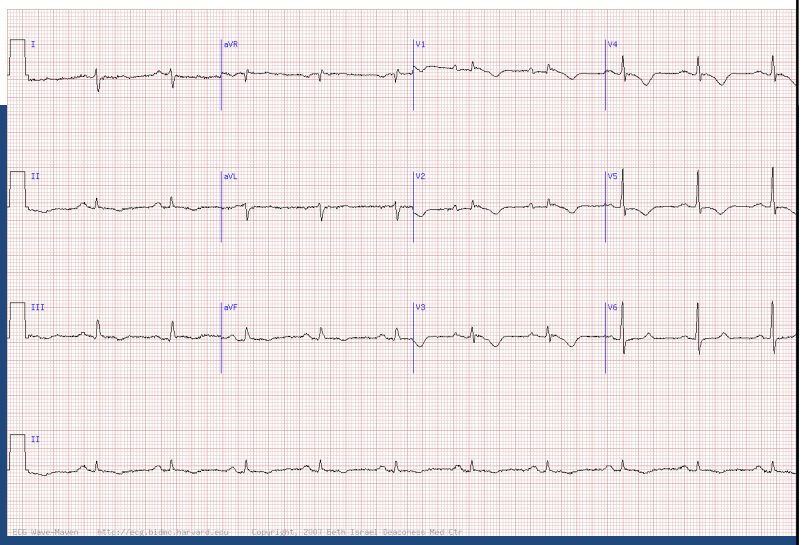
Syncope guidelines Circulation. 2017;136 Figure 3

CASE#3

HISTORY	26yo female with no significant PMH presents with first syncope in setting of daily run
PRODROME	Lightheaded/palpitations briefly
WITNESSES	Some arm twitching, blue lips
UPON WAKING	Felt well, confused, ready to run again
WORKUP	Not orthostatic, normal exam/laboratories Abnormal ECG

CASE#3 ECG

- Right axis deviation
- iRBBB
- Inverted T waves in precordium
- ? Epsilon wave



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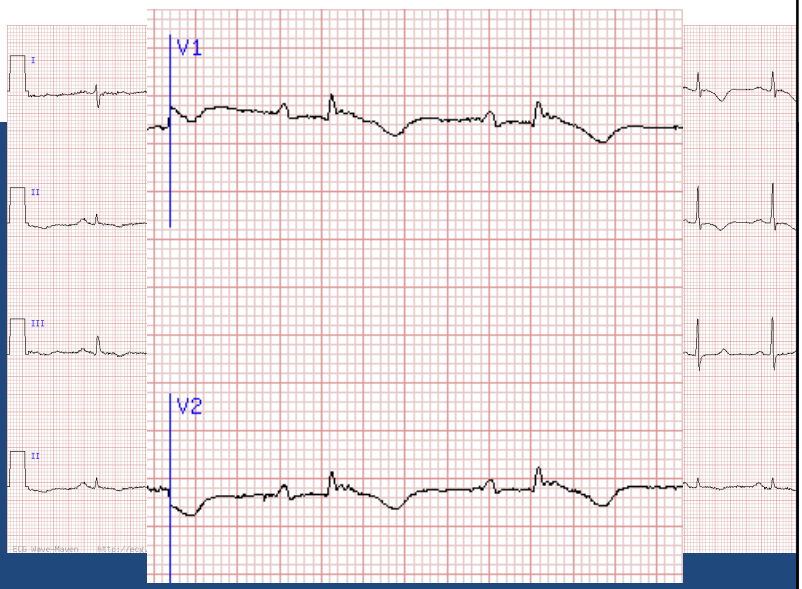

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Arrhythmogenic right ventricular cardiomyopathy with probable ventricular tachycardia

CASE#4

History	46yo male with syncope while rushing up stairs History of hypertension on lisinopril 10mg daily
Prodrome	Brief lightheaded, no palpitations/chest pain/dyspnea
Witnesses	None
Upon waking	Confused for 5 minutes, no incontinence
Workup	BP 110/70, HR 80, creatinine 0.9, BUN 20 Mildly orthostatic, normal exam/ECG

*WHAT DO YOU DO NEXT?

1. Hydrate and discharge home
2. Echocardiogram
3. Head CT/MRI
4. Stress test
5. Start fludrocortisone

*WHAT DO YOU DO NEXT?

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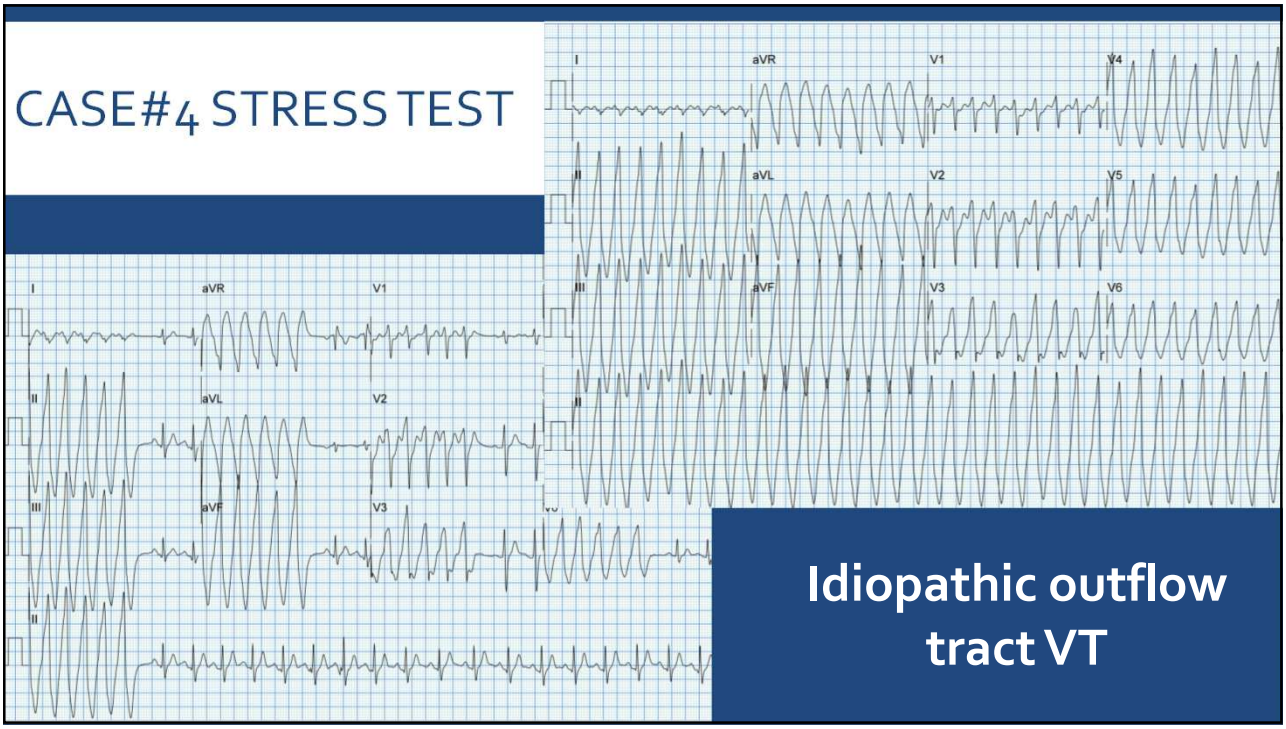
Exertional syncope is a RED FLAG!

CASE#4 STRESS TEST



Idiopathic outflow tract VT

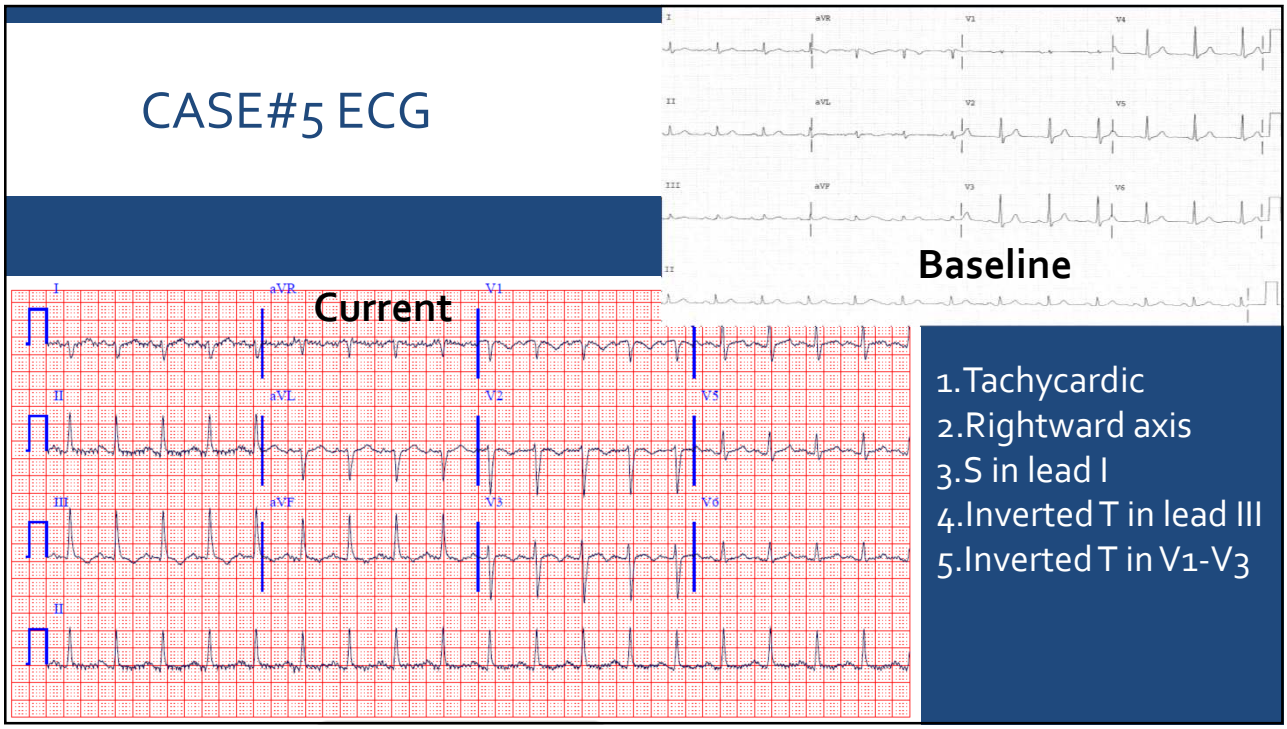
CARDIAC TESTING

- Echocardiogram (IIa, LOC-B)
  - Part of extended workup when cardiac etiology is suspected
  - Cheap, simple, and reliable method for evaluating structural heart disease
- Exercise stress testing (IIa, LOC-C)
  - Stress testing is most valuable in patients who have experienced episodes of syncope ***during or shortly after exertion***

CASE#5

HISTORY	83yo M with CKD III, remote renal cell cancer Syncope during daily walk, road trip 2 weeks ago
PRODROME	None
WITNESSES	None
UPON WAKING	Mild dyspnea, nausea and chest pain
WORKUP	SBP 100->80, HR 110bpm, JVP 16, 2/6 systolic murmur 1+ LLE, bilateral carotid bruits, crt 2.2, Hb 11

CASE#5 ECG



1. Tachycardic
2. Rightward axis
3. S in lead I
4. Inverted T in lead III
5. Inverted T in V1-V3

*WHAT DO YOU DO NEXT?

1. Diagnosis of orthostatic hypotension is clear, no further testing necessary, hydrate with IV fluids
2. Admit to hospital and observe overnight
3. Additional labs: troponin, BNP, D-dimer
4. Cardiology consult for urgent coronary catheterization
5. Obtain head CT and carotid ultrasound

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ADDITIONAL LABS

- TroponinT 0.18 ng/dL
  - >0.1ng/dL suggestive of acute MI
- D-Dimer 2000 ng/mL
  - <500ng/mL is normal
- Pro-NT BNP 655 pg/mL
  - 0-177 pg/mL is normal
  - <450 pg/mL 99% Neg pred value

*WHAT DO YOU DO NEXT?

1. Diagnosis of orthostatic hypotension is clear, no further testing necessary, hydrate with IV fluids
2. Echocardiogram
3. Chest CT angiogram
4. Cardiology consult for urgent coronary catheterization
5. Obtain head CT and carotid ultrasound

*WHAT DO YOU DO NEXT?

1. Diagnosis of orthostatic hypotension is clear, no further testing necessary, hydrate with IV fluids
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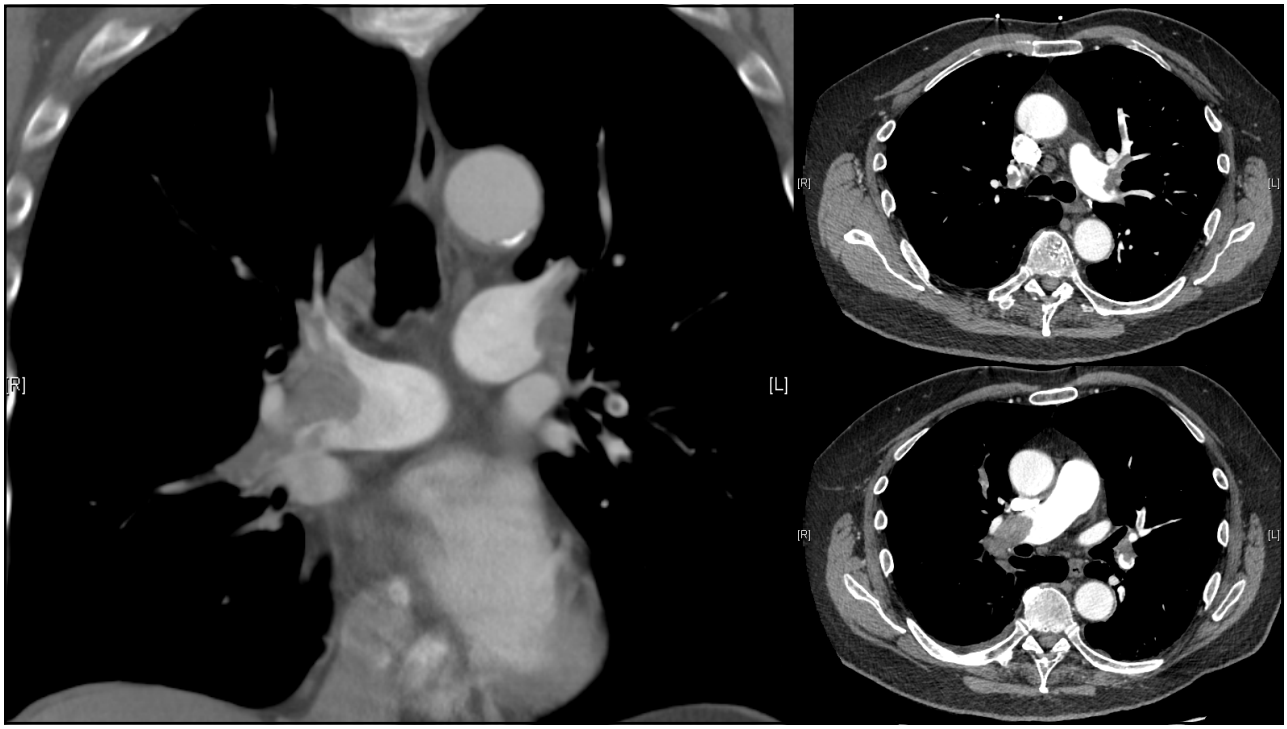


LABORATORIES

- Key elements of history helps to focus testing
- Combo of elevated high sensitivity Troponin and BNP may suggest a cardiac etiology

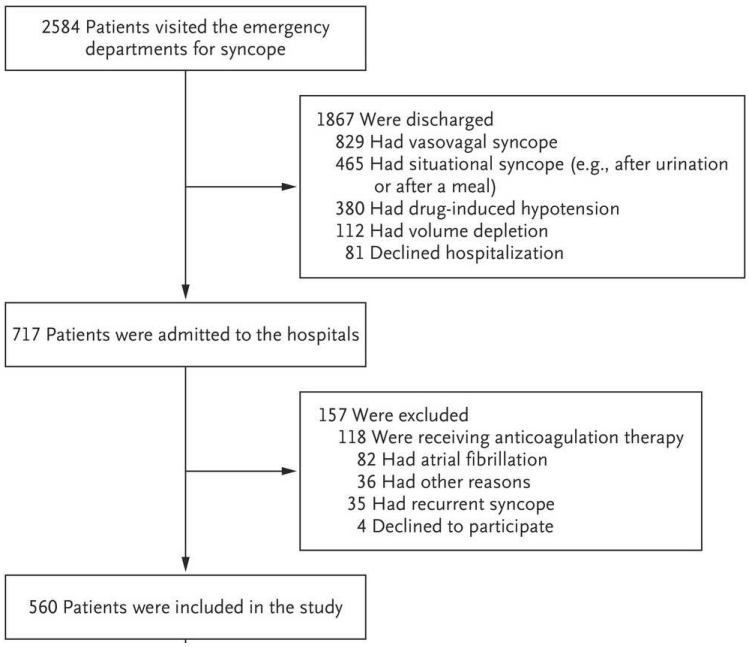
COR	LOE	Recommendations
IIa	B-NR	Targeted blood tests are reasonable in the evaluation of selected patients with syncope identified on the basis of clinical assessment from history, physical examination, and ECG.[82]
IIb	C-LD	Usefulness of brain natriuretic peptide and high-sensitivity troponin measurement is uncertain in patients for whom a cardiac cause of syncope is suspected.[83–86]
III: No Benefit	B-NR	Routine and comprehensive laboratory testing is not useful in the evaluation of patients with syncope.[87,88]

Du Fay de Lavallaz Circ. 2019;139

Syncope guidelines Circulation. 2017;136

WHAT IS THE ROLE OF D-DIMER TESTING

- Hospitalized for 1st episode of syncope
- All had detailed history and basic blood work including D-dimer
- CT angiogram or V/Q scan performed if:
  - Elevated D-dimer
  - High pre-test probability based on Wells score
- Bottom line: 1/6 (17%) pts presenting with syncope had a pulmonary embolus



Prandoni NEJM 2016;375:1524

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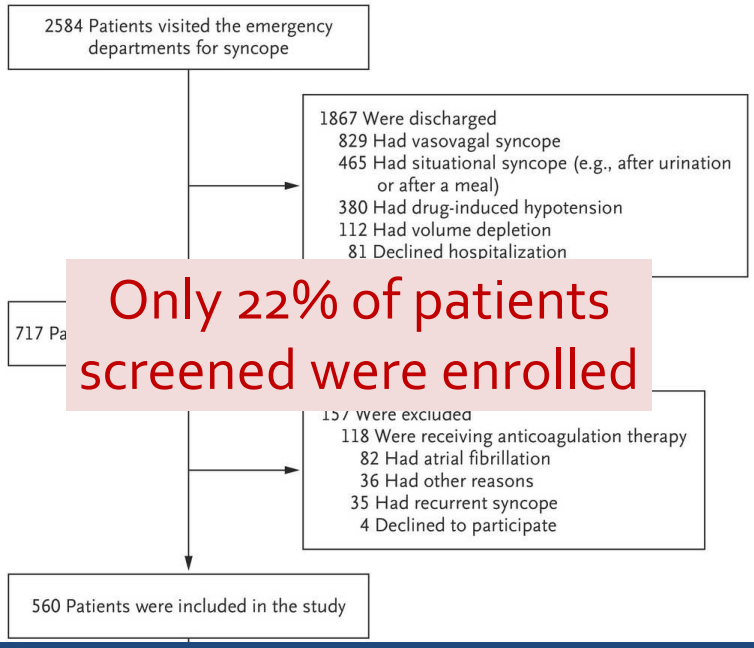

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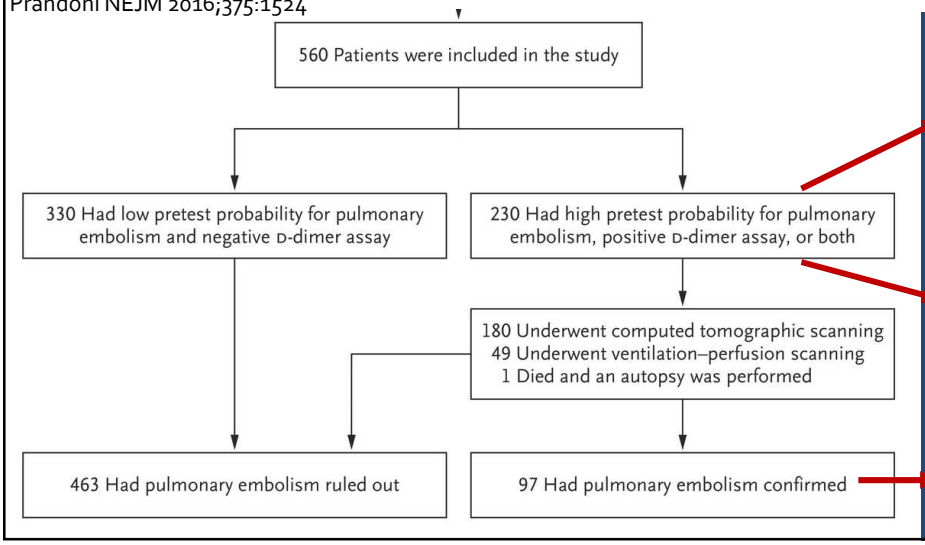


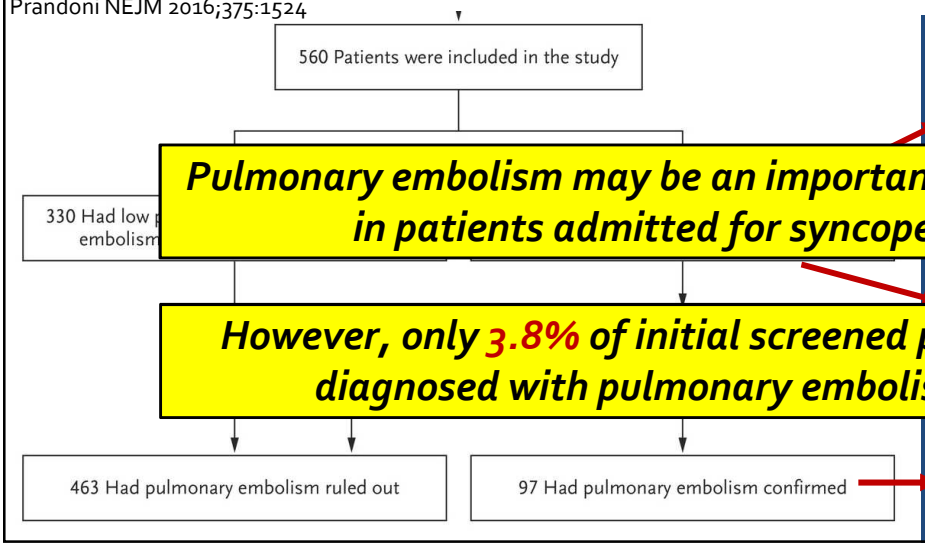
227 pts had positive D-Dimer

95 pts had high pretest probability

61 pts had main stem or lobar artery PE

WHAT IS THE ROLE OF D-DIMER TESTING

Prandoni NEJM 2016;375:1524



227 pts had positive D-Dimer

Pulmonary embolism may be an important etiology in patients admitted for syncope

95 pts had high pretest probability

However, only 3.8% of initial screened patients diagnosed with pulmonary embolism

61 pts had main stem or lobar artery PE

WHAT IS THE ROLE OF D-DIMER TESTING

- PE noted in 45 of 355 pts with potential alternate explanations of syncope
  - 31 had proximal or lobar location of PE
- Of the 97 pts with PE, 24 had NO clinical manifestations
- 32% of pts had cancer, infection, immobility, or surgery

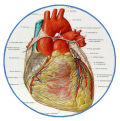
Mechanism of PE leading to syncope?
How often is PE an "incidental finding"?
How representative is this cohort?

Prandoni NEJM 2016;375:1524

SYNCOPE AND PE

- Meta-analysis to determine prevalence of PE in syncope
- Evaluated trials of patients presenting to ED or hospitalized due to syncope and etiologies including PE
  - Total of 12 studies
  - 7582 patients presented to ED or hospitalized
  - Pooled estimate of ED patients: ***0.8%*** (95% CI 0.5-1.3%)
  - Pooled estimate of hospitalized patients: ***1.0%*** (95% CI 0.5-1.9%)
- No *systematic* evaluation of PE in all patients

Ogab Am J Emerg Med 2018:36



STRUCTURAL HEART DISEASE

Any structural or physiologic abnormality that ***limits the augmentation of cardiac output during exertion*** may lead to global cerebral hypoperfusion

Since cardiopulmonary structures are connected in "series", any restriction in the circuit has the potential to obstruct flow

- ***Aortic stenosis and mitral stenosis*** are the most common
- Regurgitant valve lesions rarely cause syncope

CASE#6

HISTORY	69yo F with asx paroxysmal Afib, HTN on warfarin Second time unresponsive while watching TV in 2 months
PRODROME	"Vision blackening"
WITNESSES	Eyes rolled back, no jerking movement, <1 minute
UPON WAKING	Felt well
WORKUP	Not orthostatic, normal exam/laboratories ECG: sinus brady at 55bpm, otherwise normal

*WHAT TYPE OF CARDIAC MONITOR IS MOST APPROPRIATE?

1. 48hr Holter
2. Zio patch (2 weeks)
3. Looped event monitor or mobile telemetry (4 weeks)
4. Non-loop event monitor (4 weeks)
5. Implantable loop monitor
6. Kardia cell phone attachment

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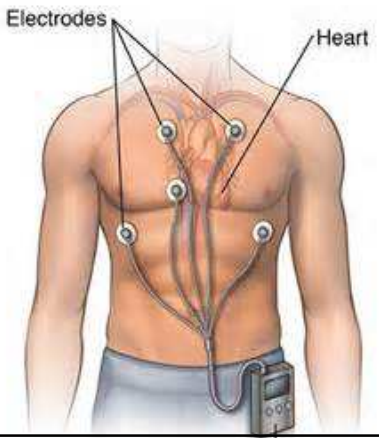
Type of monitor dictated by frequency of events

EVALUATION FOR ARRHYTHMIA

Method	Comment
ECG (12 seconds)	Low yield, but excellent screening test
Holter (24-48 hours)	Useful only for ***very frequent*** events
Event (loop) Recorder (7-30 days)	Useful for ***less frequent*** events
Implantable Loop Recorder (ILR)	For very ***infrequent*** events Battery life can last up to 3 years
Invasive Electrophysiologic study (EPS)	Mostly helpful in structural heart disease or abnormal EKG Tachyarrhythmias>>>bradyarrhythmias

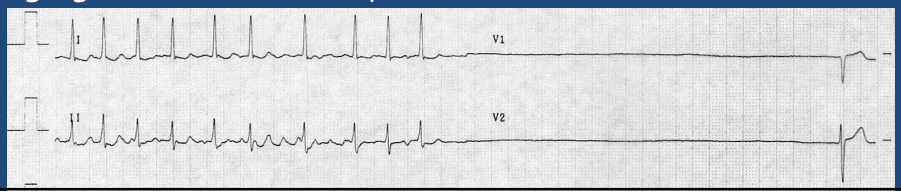
NON-loop monitors are NOT appropriate for syncope workup

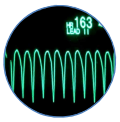
CARDIAC MONITORS



BRADYARRHYTHMIAS

- Most common type of arrhythmia associated with syncope
- Problem with impulse ***generation***
  - Sinus arrest, sinus exit block, conversion pause
- Problem with impulse ***conduction***
  - High grade or acute complete AV block



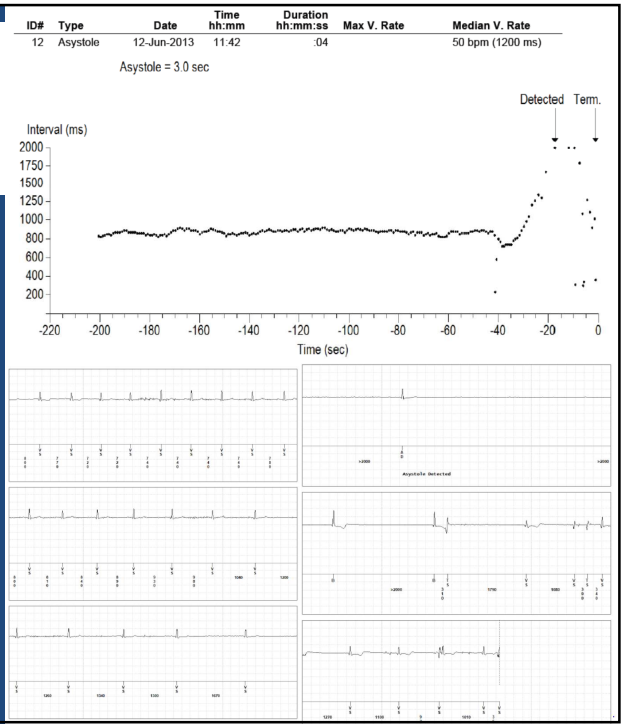


TACHYARRHYTHMIAS

- <u>Supraventricular Tachycardia</u>
  - AVNRT – AV nodal reentrant tachycardia more commonly associated with syncope
- <u>Ventricular Tachycardia</u>
  - Structural heart disease – i.e. prior myocardial infarction, hypertrophic cardiomyopathy
  - Inherited arrhythmia syndromes - i.e. Long QT syndrome
  - Drug/metabolic induced- i.e. Torsade de pointes, bidirectional VT (digoxin toxicity)
  - Pre-excited atrial fibrillation in WPW
  - Idiopathic VT- uncommon

IMPLANTABLE LOOP RECORDER

- Consider ILR if syncope is recurrent, rare, and workup including event monitor has not been diagnostic
- Simple brief surgical procedure
- Long term monitoring (3 years)
- Patient non-compliance eliminated
- Gold standard in recurrent unexplained syncope



IMPLANTABLE LOOP RECORDER

EaSyAS II trial

- 2 syncopal episodes within 2 years
- 246 pts randomized to ILR vs conventional management and syncope clinic (CONV)
- **50%** had ECG diagnosis of syncope with ILR with mean of 95 days
- **17%** had ECG diagnosis in CONV, mostly using tilt table testing
- ILR pts had less testing performed

Sulke Europace 2016;18:912

FRESH study

- 2 syncopal episodes within 1 year
- 78 pts randomized to ILR vs conventional management
- **46%** of ILR pts had diagnosis established within 14 month f/u
- **5%** of CONV pts had diagnosis established
- ILR pts had less testing performed

Podoleanu Arch Cardiovasc Dis 2014;107:546

IMPLANTABLE LOOP RECORDER

EaSyAS II trial	FRESH study
• 2 syncopal episodes within 2 years • 246 pts randomized to ILR vs	• 2 syncopal episodes within 1 year • 78 pts randomized to ILR vs
• **17%** had ECG diagnosis in CONV, mostly using tilt table testing • ILR pts had less testing performed	diagnosis established • ILR pts had less testing performed
Sulke Europace 2016;18:912	Podoleanu Arch Cardiovasc Dis 2014;107:546

ILRs most effective in establishing or refuting arrhythmic etiology of recurrent syncope. Perhaps cheaper as well?

IMPLANTABLE LOOP RECORDER

- ESC 2018: ILR can also be considered for
  - Suspected but unproven epilepsy (IIa)
  - Unexplained falls (IIb)

ESC Syncope guidelines Eur Heart J. 2018;1183

CASE#7

History	81yo F with CAD, Afib, diabetes, and CKD Unwitnessed fall resulting in right wrist fracture
Prodrome	No recollection, ? Loss of consciousness
Witnesses	None
Upon waking	Nausea and wrist pain
Workup	Mildly orthostatic, no head trauma, L carotid bruit R hand pain/weakness, no other deficit

*WHICH IS ***LEAST*** LIKELY TO BE USEFUL?

1. Echocardiogram
2. Head CT and carotid ultrasound
3. D-Dimer
4. Event monitor

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What is the value of neuroimaging in syncope?

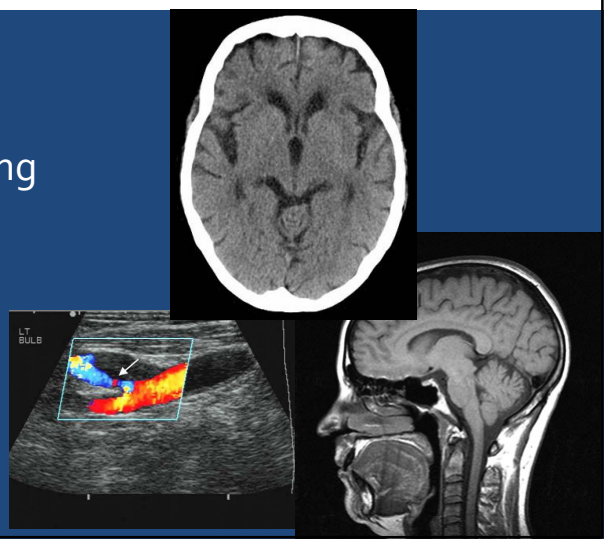
NEURO IMAGING

- 1114 pts presenting to the ED with syncope with or without mild head trauma
- Pts with focal neuro deficits, dizziness, N/V, or anticoagulant use were excluded
- Head CT was performed in 62.3% and Brain MRI in 10.2%
  - Total of 808 studies
- ***NONE*** of the neuro imaging studies revealed any clinically significant findings

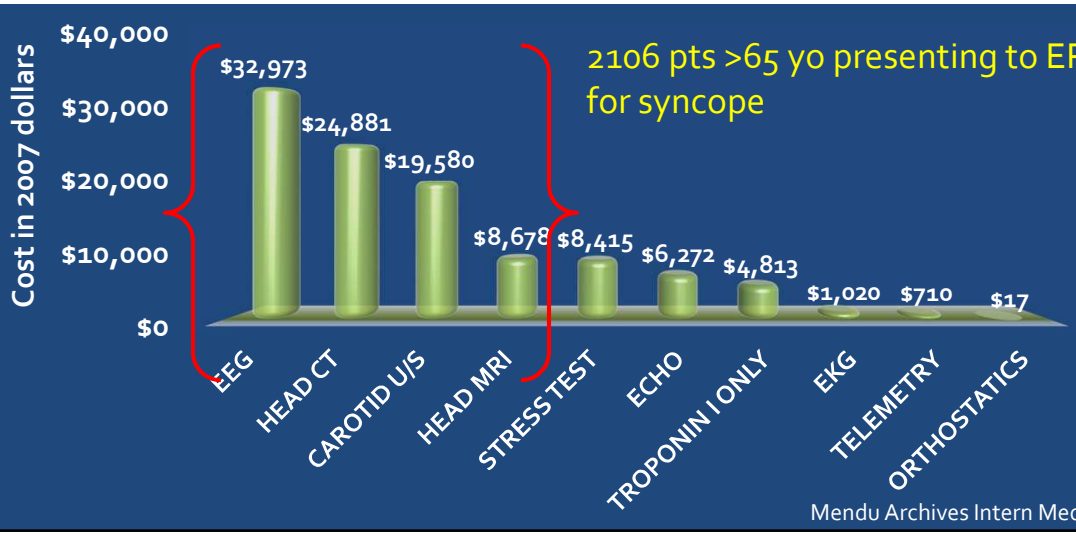
Idil Amer J Emer Med 2018

NEURO IMAGING

- If no focal neuro deficits, **brain imaging NOT necessary**
- Reasonable to order if suspecting
  - Seizure
  - Acute CVA
  - Head trauma
- Class III, LOE B: ***No Benefit***



TRUE COST OF TESTING



Mendu Archives Intern Med. 2009

CEREBROVASCULAR DISORDERS

- **Subclavian steal**: vigorous arm movement, shunts blood flow to arm through reversal of vertebral artery flow secondary to stenosis of subclavian artery- reproducible
- **TIA of vertibrobasilar system**: can cause LOC- often with vertigo and possible drop attacks
- **TIA of carotid artery**: rarely causes LOC unless concomitant severe stenosis causing global cerebral ischemia
  - Can sometimes have associated vasovagal syncope

ALL of these syndromes typically have associated focal exam findings

CASE#8

History	46yo M with recurrent syncope, 5 times over 10 years Associated with stressful/emotional events
Prodrome	Lightheaded, cold sweat
Witnesses	Looked "white as a ghost"
Upon waking	Nausea/vomiting, better after 30 minutes
Workup	BP 105/70, HR 58, not orthostatic Normal exam/labs/ECG

*WHICH THERAPY CAN PREVENT RECURRENT SYNCOPE IN THIS PATIENT?

1. Physical counter pressure maneuvers
2. Salt and volume loading
3. midodrine
4. fludrocortisone
5. Fluoxetine
6. Metoprolol
7. Dual chamber pacemaker

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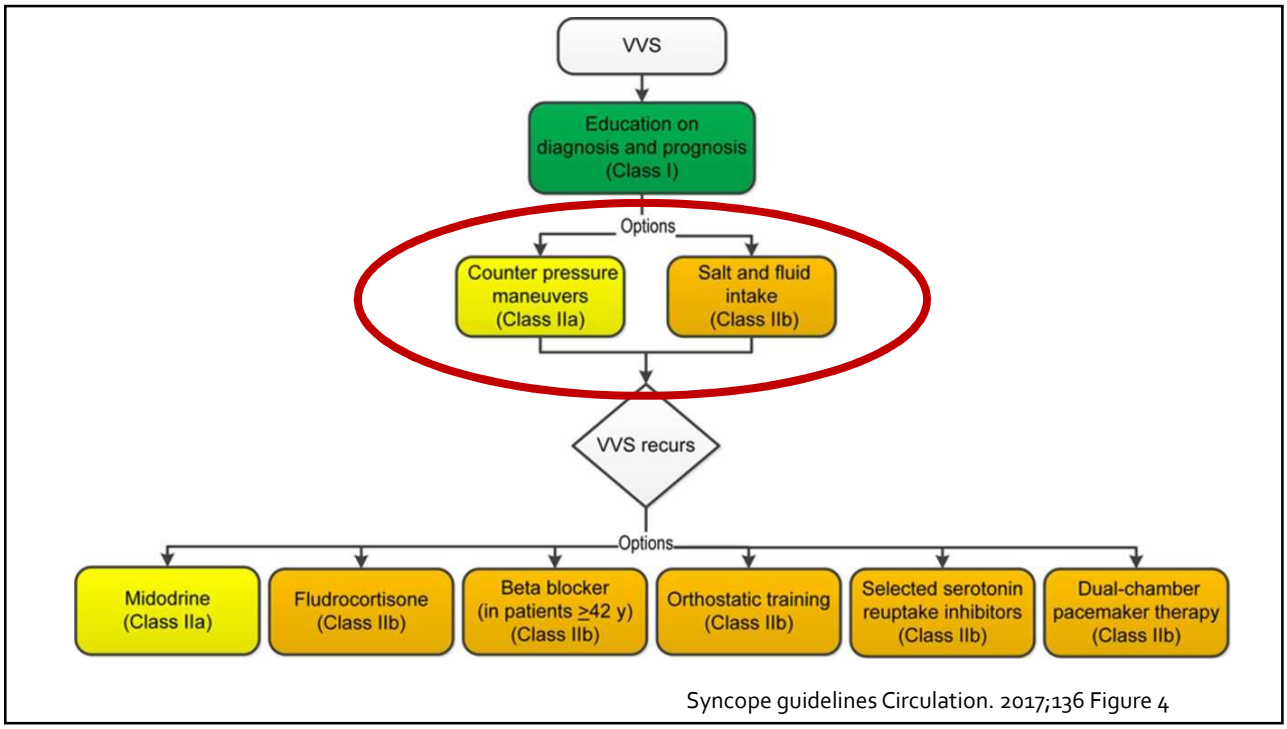
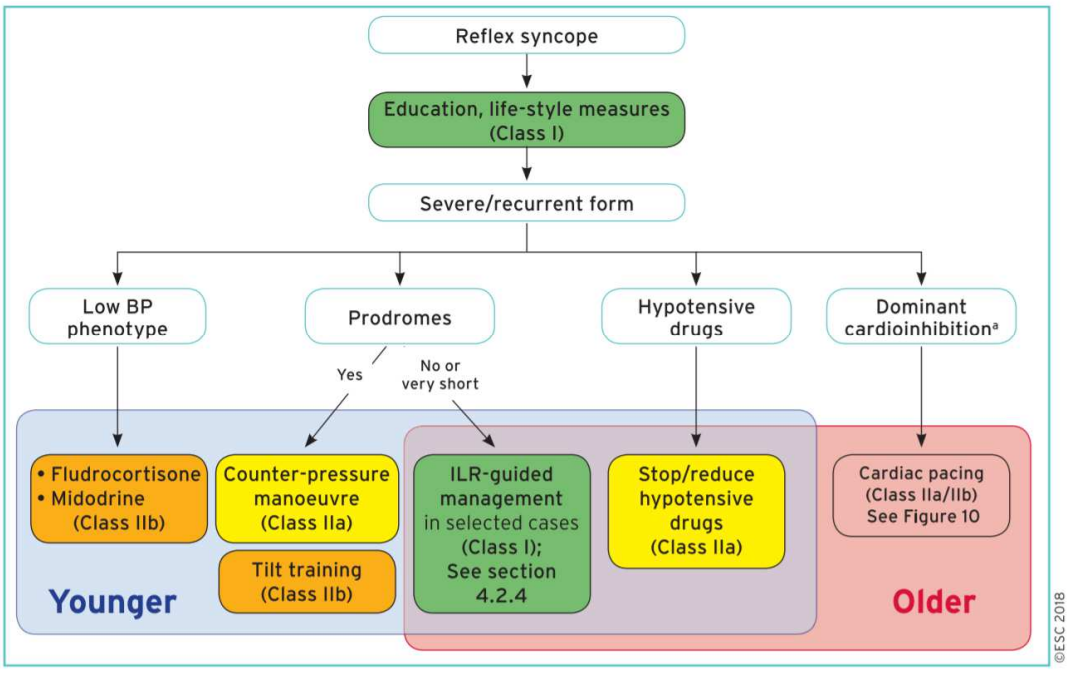
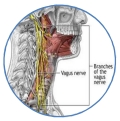


Syncope guidelines Circulation. 2017;136 Figure 4



ESC Syncope guidelines Eur Heart J. 2018;1183 Figure 9



NEURALLY MEDIATED SYNCOPE TREATMENT

- ***Lack*** of strong data for any treatment
- Acceptable to turn syncope into near syncope
- Trigger and prodrome recognition and prevention
- Cornerstone of therapy is salt and volume loading
  - Hydration with increased salt intake
- Physical counter pressure maneuvers
  - Arm tensing, hand grip, leg crossing

NMS: PACEMAKERS

- Several randomized trials with various methodological limitations
  - Many early studies were negative
- Pacemaker implantation most beneficial in patients with documented asystole >3 sec either by tilt table testing or ILR
- Algorithms that have shown benefit
  - Rate drop response/hysteresis
  - Closed loop system (CLS)- Biotronik
- 5 yr follow-up study: 66% RRR and 24% ARR in recurrent syncope

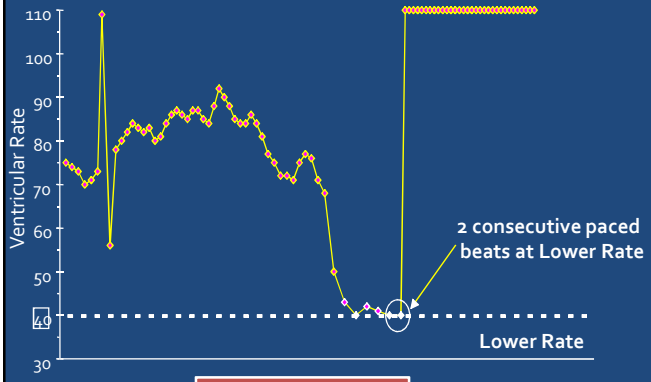
Russo Int J Cardiol 2018

Table 3
Trials that have assessed the role of pacing in reflex syncope

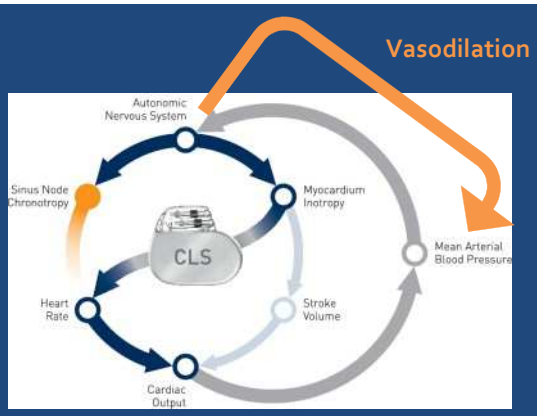
Trial	Inclusion Criteria	Design
VPS[45]	Positive TT with HR <60, 70, 80 bpm	PI vs conventional treatment
VASIS[46]	Positive TT with HR <40 bpm or asystole >3 s	PI vs conventional treatment
SYDIT[47]	Positive TT with HR <40 bpm or asystole >3 s	PI vs atenolol
VPSII[49]	Positive TT with BP × HR <6000	PI with randomization DDD vs ODO
SYNPACE[50]	Positive TT with HR <40 bpm or asystole >3 s	PI with randomization DDD vs ODO
INVASY[51]	Positive TT with cardioinhibitory or mixed response	PI with randomization DDD-CLS vs DDI
Flamang et al,[55] 2012	Asystole >10 s after intravenous ATP administration	PI with randomization DDD vs AAI
ISSUE 3[56]	Syncope with documented asystole >3 s or asymptomatic spontaneous asystole >6 s (usually documented by ILR)	PI with randomization DDD vs VVI 40 bpm

Moya Cardiol Clin 2015

PACEMAKERS: RATE DROP RESPONSE



Rate drop response

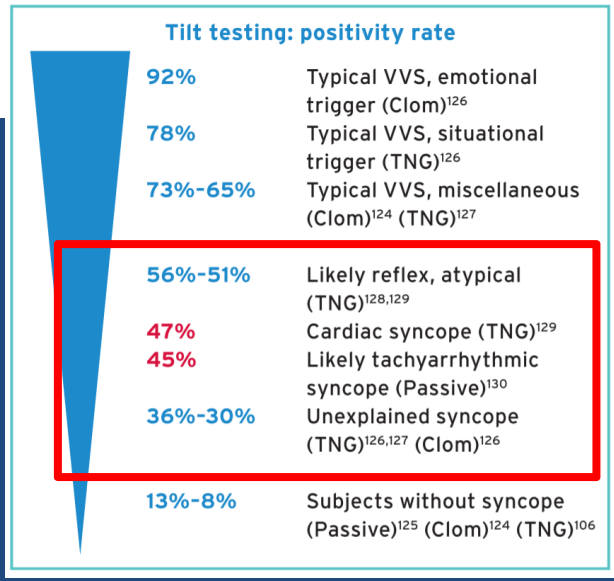


Biotronik closed loop system

Adapted from Benditt and Sutton "Syncope A diagnostic and Treatment Strategy"

TILT TABLE TESTING

- TTT provides little diagnostic value for whom it is most needed
- At most can suggest "hypotensive susceptibility"
- Can be helpful in pts with suspected diagnosis of POTS

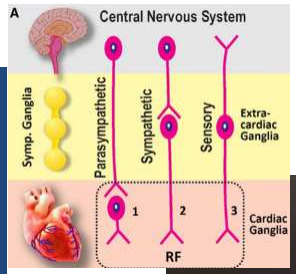


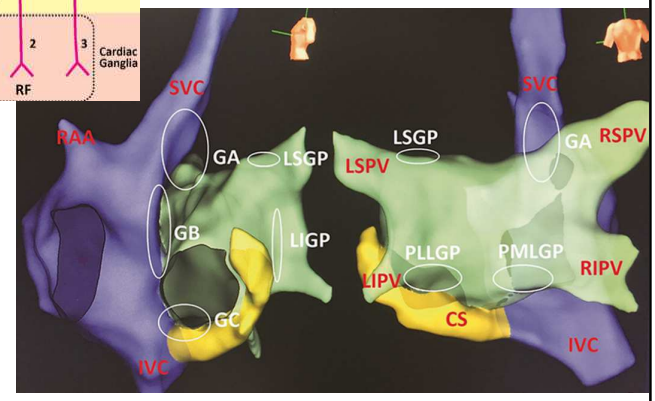
ESC Syncope guidelines Eur Heart J. 2018;1183 Figure 7

CARDIONEURO ABLATION (CNA)

- Targets cardiac parasympathetic ganglia from endocardial approach
- "Modification" of Afib ablation
- Early data is extremely promising
- Await long-term results

Cardiac Parasympathetic Ganglia





Pachon Circ Arrhythm Electrophy 2020; Aksu J Inter Cardiac Electrophy 2020

CASE#9

History	70yo M with diabetes, HTN, CKD, and CLL Syncope while having dinner, recent change in meds
Prodrome	Lightheaded, palpitatins
Witnesses	Eyes rolled back, fell to the side
Upon waking	Nausea, better after 10 minutes
Workup	110/80, HR 90, orthostatic, crt 1.5, Hg 10, WBC 25 Neurologically intact, ECG with RBBB which is chronic

*ADMIT OR NOT ADMIT?

1. Admit to hospital for expeditated workup, telemetry observation, and treatment
2. Follow-up with PCP within one week
3. Urgent outpatient cardiology consult within 3 days

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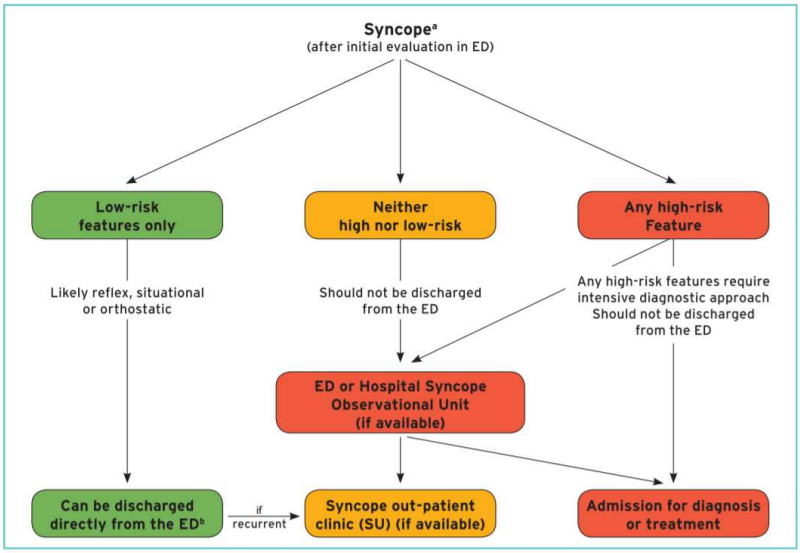
RISK ASSESSMENT

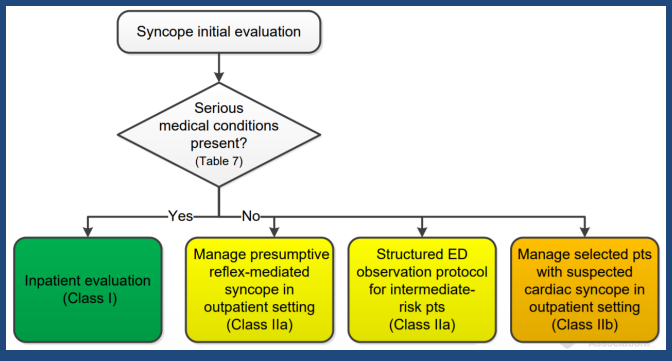
- Short and long term morbidity and mortality assessment
- Whom to admit and whom to discharge?
- Syncope risk scores no better than clinical judgement



ESC Syncope guidelines Eur Heart J. 2018;1183 Figure 6

RISK ASSESSMENT

- Serious comorbidities
- Age>65
- Exertional syncope
- Supine syncope
- Palpitations
- Abnormal ECG
- Abnormal vitals
- Abnormal exam



Syncope guidelines Circulation. 2017;136 Figure 2

SUMMARY

- Basic workup
  - Detailed history and exam, orthostatic vitals, ECG
  - Will provide the greatest diagnostic yield
- Targeted workup
  - Labs, echocardiogram, chest CT, etc. as warranted
  - Provides small additional yield
- Recurrent syncope
  - Frequency dictates which cardiac monitor to use
  - Implantable loop recorders: highest diagnostic yield of secondary testing
- Brain Imaging
  - **ONLY** if focal neuro deficits or head trauma