



# SYNCOPE

Know and understand:

- Factors that may lead to syncope in older adults
- Elements of evaluation (history, physical examination, testing) of older adults with syncope
- Treatment options for syncope

- Natural History: Diagnosis and Prognosis
- Pathophysiology
- Evaluation: History, Physical Exam, Diagnostic Testing
- Treatment

- A symptom complex composed of a sudden and transient loss of consciousness resulting from a temporary interruption of global cerebral perfusion
- A common reason for evaluation in both outpatient clinics and emergency departments, and for hospital admission
- Annually it accounts for approximately 3% of emergency department visits and 2%–6% of hospital admissions

- Incidence of syncope increases with age
  - Incidence doubles in those  $\geq 70$  years old, and the rate among those  $\geq 80$  years old is three to four times that seen among younger people
  - Approximately 80% of patients hospitalized for syncope are  $\geq 65$  years old
- Potential causes range from those that are benign and self-limiting to those that are life threatening
  - In older adults, the cause of syncope can often be multifactorial, adding to the diagnostic difficulty

Decreases in Cardiac Output or Peripheral Vascular Resistance, or both



Decreased Systemic Blood Pressure and Reduced Cerebral Perfusion



Syncope

Causes may be benign or life-threatening

Causes may be multifactorial

Must consider adverse effects of drugs as a cause

# COMMON CAUSES OF SYNCOPE

- Neurally mediated
- Cardiac rhythm disturbances
- Decreased intravascular volume due to blood loss or dehydration
- Alterations in the peripheral vasculature due to arterial vasodilation or increased venous pooling
- Medication related

Epileptic seizure, a common cause of transient loss of consciousness, is no longer categorized as a cause of syncope, because seizure is not mediated by a decrease in cerebral perfusion

It is important to differentiate a seizure from syncope as a cause of transient loss of consciousness

# DISTINGUISHING CHARACTERISTICS OF SEIZURE AND SYNCOPE

Phase	Sign/Symptom	Seizure	Cardiac Syncope Due to	
			Arrhythmia	Vasovagal Syncope
<b>Before</b>	Position	Any	Any	Upright; aborted by lying flat
	Warning/prodrome	None	<5 seconds	Seconds to minutes
	Precipitant	Usually absent	Absent	Present
	Palpitations	Absent	Sometimes	Absent
<b>During</b>	Nausea/diaphoresis	Rare	Absent	Common
	Visual changes	None	None	Common
	Tone	Rigid	Flaccid	Motionless, relaxed
	Pulse	Rapid	Absent or faint	Slow, faint
	Color	Pale or normal	Blue, ashen	Pale
	Incontinence	Common	Rare	Very rare
	Eye findings	Tonic eye deviation	Variable pupils	Dilated, reactive pupils
	Oral frothing	Common	Absent	Absent
<b>After</b>	Type of recovery	Slow, incomplete	Rapid, complete	Fatigue common
	Mental status	Disorientation	No retrograde amnesia	No retrograde amnesia
	Nausea/diaphoresis	Rare	Absent	Common
	Focal neurologic findings	Common	Absent	Absent

Characteristics most distinctive in determining the cause of syncope are highlighted in bold.



- Depends on the underlying cause
- Cardiac causes have the worst prognosis
  - 1-year mortality is 18%–33%, with deaths chiefly due to underlying disease, not syncope
- Noncardiac causes
  - 1-year mortality of approximately 6%

- **Neurally mediated or vasovagal syncope**
  - Vasovagal mechanisms as the cause of syncope in approximately 30%–50% of patients >65 years old
  - Some suggestion that vasovagal syncope in older adults is often associated with comorbid illness that can increase overall mortality
- **No cause** found In approximately 10%–20% of syncopal patients
  - Prognosis for these patients is no worse or better than that of the general population

# PATHOPHYSIOLOGY: MECHANISMS THAT PREVENT SYNCOPE

- The integrity of a number of control mechanisms is crucial for maintaining adequate perfusion of the brain and cerebral oxygen delivery after sudden changes in blood pressure
  - Carotid and aortic baroreceptors
  - Sympathetic renal stimulation of the renin-angiotensin system
  - Arteriolar autoregulation

- Reflex mechanisms are less responsive with aging
  - Decreased ability to increase heart rate in response to sympathetic stimulation
  - Increased sensitivity to effects of dehydration, diuretics and vasodilator medications
- Comorbidities that affect postural responses are common (eg, diabetes mellitus, Parkinson disease)
- Medications may further impair postural reflexes (eg, vasodilators, calcium channel blockers, ACE inhibitors,  $\alpha$ -blockers,  $\beta$ -blockers, tricyclic antidepressants)

- Age-related decline in adaptive reflexes, comorbid conditions, and medications can all have a role in older adults presenting with syncope
- Address these issues by:
  - Increasing caution with postural change, physical counter-pressure maneuvers (eg, leg crossing) and compression stockings to increase venous return
  - Adequate hydration
  - Simplification of the patient's medication regimen to eliminate excessive medications

# SYNCOPE EVALUATION: HISTORY (1 of 2)

An accurate recall of the syncopal event is frequently inadequate because of the high prevalence of cognitive dysfunction in the older population. The medical history, if possible, should be obtained from a witness to the event.

## Precipitants?

- Eating
- Urinating
- Coughing
- Medication
- Emotional stress
- Physical exertion
- Turning head

## Prodromal symptoms?

- Chest pain
- Palpitations
- Dyspnea
- Diaphoresis
- Presyncope
- Nausea
- Vomiting

# SYNCOPE EVALUATION: HISTORY (2 of 2)

- Medications?
  - How, when taken, what doses
  - Relationship to meals and activities
  - Recent changes
- Any witnesses?
  - Duration of event
  - Appearance of patient during event (flaccid tone and motionless or increased tone and motion?)
- Comorbid conditions?
  - For example, CAD, Diabetes Mellitus

# PREDICTING CARDIAC SYNCOPES: EGSYS SCORE

Variable	Score
Palpitations preceding syncope	4
Heart disease or abnormal ECG	3
Syncope during effort	3
Syncope while supine	2
Precipitating or predisposing factors, or both (warm or crowded place, prolonged orthostasis, fear, pain or emotional distress)	-1
Autonomic prodromes (nausea/vomiting)	-1



# CARDIAC SYNCOPE DUE TO ARRHYTHMIA

- **Prior to event**
  - Occurs in any position, <5 sec warning
  - No precipitant; palpitations are sometimes present
  - No nausea, diaphoresis or visual changes
- **During event**
  - Flaccid tone; pulse faint or absent
  - Blue, ashen skin
  - Incontinence (rare)
  - Variable pupils, no oral frothing
- **Recovery**
  - Rapid and complete

- **Prior to event**
  - Aborted if person lies flat
  - Seconds to minutes of warning
  - Precipitant present; nausea/diaphoresis common
  - Visual changes are common
- **During event**
  - Motionless; relaxed tone; slow, faint pulse
  - Pale color; dilated, reactive pupils
- **Recovery**
  - Fatigue, nausea, and diaphoresis common
  - No retrograde amnesia

- **Prior to event**
  - Occurs in any position
  - No warning or prodrome
- **During event**
  - Rigid tone; rapid pulse
  - Tonic eye deviation common
  - Frothing at mouth
- **Recovery**
  - Slow, incomplete
  - Disorientation; focal neurologic findings

# SYNCOPE EVALUATION: PHYSICAL EXAMINATION (1 of 2)

- Should focus on elements raised by the history
- **Pulse** in supine and standing positions
- **Orthostatic vital signs**: Measure BP in both arms, 1 min after standing and again after standing for 3 min
- **Carotid pulse examination**
  - Delayed upstroke and low volume may identify aortic stenosis
  - Perform carotid massage only with continuous ECG and resuscitation equipment available
    - Contraindicated with carotid bruit, CVD, recent MI

# SYNCOPE EVALUATION: PHYSICAL EXAMINATION (2 of 2)

- Cardiac examination for murmurs, extra heart sounds
- Stool for occult blood
- Neurologic examination for focal deficits

# SYNCOPE EVALUATION: DIAGNOSTIC TESTING (1 of 4 )

- The following slides review the various diagnostic tests that are available for the evaluation of syncope
- It is important to remember that not every test is required; the history and physical are used to determine appropriate testing
- In all patients, an assessment of orthostatic vital signs, gait, laboratory tests, and ECG are reasonable first steps

# SYNCOPE EVALUATION: DIAGNOSTIC TESTING (2 of 4)

- ECG for all syncopal older adults; assess for:
  - Acute or remote MI
  - Conduction abnormalities and pre-excitation, such as Wolff-Parkinson-White
  - Sinus bradycardia
  - Prolonged QT interval
- Ambulatory ECG
  - Among patients able and willing to operate loop recorders, diagnostic yield is ~25%
- Implantable loop recorders
  - Consider using in patients when an arrhythmic cause of syncope is suspected but not sufficiently proved

# SYNCOPE EVALUATION: DIAGNOSTIC TESTING (3 of 4)

- **Echocardiography and Exercise Stress Testing**
  - Most useful in confirming a specific diagnosis suspected by other assessment, otherwise it is low yield
- **Tilt-table testing**
  - Useful for patients suspected of having vasovagal syncope and those with unexplained syncope who are not suspected of having a cardiac cause.
- **Electrophysiologic studies**
  - Not recommended for patients with a normal ECG or those without a history of heart disease or symptoms of palpitations



# SYNCOPE EVALUATION: DIAGNOSTIC TESTING (4 of 4)

- Neurologic testing (CT or MRI, and/or EEG)
  - Not generally required
  - Appropriate in situations when focal neurologic signs or symptoms are present or when the history suggests seizure or trauma
  - Consider autonomic evaluation if signs and symptoms of autonomic dysfunction are present

# SYNCOPE: IS HOSPITAL ADMISSION REQUIRED?

- Older patients are frequently hospitalized for evaluation because they are presumed to be higher risk
- However, patients with no risk factors for adverse events were found to have no significant increase in adverse events regardless of age
- A specialized syncope observational unit in the hospital setting has been shown to reduce hospital admission and expedite the diagnosis of potential causes of syncope

# RISK FACTORS FOR ADVERSE PROGNOSIS IN SYNCOPE

<b>Symptoms</b>	<b>Chest pain</b> <b>Shortness of breath</b> <b>Palpitations or rapid heart beat</b> <b>GI bleeding</b>
Cardiac history	Coronary artery disease Heart failure Hypertrophic cardiomyopathy Pacemaker or defibrillator Antiarrhythmic medications Ventricular tachycardia or ventricular fibrillation
Syncope characteristics	Syncope during exercise >1 episode within 6 months
Family history	First-degree relative with sudden death, hypertrophic cardiomyopathy, Brugada syndrome, or long QT syndrome
Physical examination	Tachypnea Hypoxia (O <sub>2</sub> saturation <90%) Sinus heart rate <50 beats/min or >100 beats/min Systolic blood pressure <90 mmHg Heart murmur Volume depletion Neurologic deficits
ECG abnormalities	Q waves Ischemic ST segment or T wave changes Ventricular or supraventricular arrhythmias, including rapid atrial fibrillation Second- or third-degree AV block Corrected QT interval >500 ms
Laboratory abnormalities	Hematocrit <30% Occult blood in feces

*Note: Patients with no risk factors for adverse prognosis can likely be safely dismissed from the emergency department without hospitalization.*

- Goals of treatment: Improve quality of life and prevent physical injuries
- Focus on treating the underlying disorder
- In older patients, treatment of multiple possible causes is often necessary
- Discontinuation of medications that increase the risk of syncope is always an early step

## Reflex Syncope and Postural Hypotension

- Nonpharmacologic measures with physical counter-pressure maneuvers
  - Leg crossing, arm tensing, hand grip, and buttock clenching are able to induce a significant blood pressure increase during the phase of impending reflex syncope so that the patient can avoid or delay losing consciousness
- Compression stockings and abdominal binders
- Smaller and frequent meals can be effective in patients with postprandial hypotension

## Medical Management

- Midodrine
  - Increases vasoconstriction, may help with persistent postural hypotension
  - Use with caution: May cause supine hypertension in those with decreased vascular compliance
  - Monitor blood pressure response and symptoms
- Pyridostigmine
  - Increases both standing blood pressure and peripheral resistance, attenuating orthostatic blood pressure
  - Adverse effects: Abdominal cramps, diarrhea, urinary urgency
  - Use with close supervision, older adults may not tolerate adverse effects

## Medical Management

- Volume expansion with added salt
- Fludrocortisone
  - Both increase renal sodium retention and intravascular volume
  - May be effective in patients with persistent postural hypotension

## Role of Pacemakers

- Mainstay of treatment for sinus node dysfunction or high grade AV block
- Permanent pacing is indicated when syncope or near syncope is correlated with bradycardia after ensuring that medications that may be causing bradycardia have been discontinued, if indicated after a risk vs benefit analysis
- Not recommended in patients with unexplained syncope or falls without documentation of bradycardia



- In older adults the cause of syncope is often multifactorial
- Important to review the medication list of each patient, as medications are commonly implicated as a source of syncope
- Most diagnostic procedures for syncope are expensive and have a low yield unless findings from the history and physical suggest a particular cause

# SUMMARY (2 of 2)

- Important to rule out cardiac conditions as a cause of syncope as they are associated with worse prognosis
- Treatment of syncope focuses on treating the underlying disorder

*Recommendations for **Syncope**, based on the American Board of Internal Medicine Foundation's Choosing Wisely® Campaign:*

- Do not perform imaging of the carotid arteries for simple syncope without other neurologic symptoms.
- In the evaluation of simple syncope and a normal neurologic examination, do not obtain brain imaging studies (CT or MRI).

- A 68-year-old man lost consciousness twice in the last several months. His daughter witnessed the second event.
  - Patient was washing dishes when he suddenly felt lightheaded, looked pale and sweaty, and collapsed to the floor.
  - He felt lightheaded for about 30 sec before losing consciousness.
  - He awoke within 30 sec and was back to baseline within 1 minute of the event.
- History: hyperlipidemia, stroke, Parkinson disease, diabetes mellitus
- Examination
  - Blood pressure 129/78 mmHg
  - Normal cardiopulmonary and neurologic findings
  - ECG: no significant abnormalities

Which one of the following would most likely establish the diagnosis?

- A. Electroencephalography
- B. Tilt table test
- C. Magnetic resonance imaging of the brain
- D. Doppler ultrasonography of the carotid artery
- E. 24-Hour Holter monitoring

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