Outline

- Definition
- Pathophysiology
- Clinical classification and diagnostic evaluation
- Approach to evaluation
- Management
- Vasovagal syncope
- Postprandial hypotension
Syncope: definition

- Sudden transient loss of consciousness
  - Associated with loss of postural tone
  - Recover spontaneously
80% of the syncope patients are > 65 years old

Result in hospital admission (1-6%) and performance of many diagnostic tests

Association with falling in the elderly and significance injury after falling 26%

Increase mortality: esp cardiac causes

Injury from syncope > other falls

Syncope in the elderly, Siriraj Med J 2006; 58
In the elderly: Non cardiac cause 38%
- orthostatic hypotension
- vasovagal syncope
- drug induced syncope

<table>
<thead>
<tr>
<th>Cause</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac Ischemia</td>
<td>0.1</td>
</tr>
<tr>
<td>Miscellaneous cardiac</td>
<td>1.9</td>
</tr>
<tr>
<td>Orthostatic hypotension</td>
<td>10.8</td>
</tr>
<tr>
<td>Vasovagal response</td>
<td>8.5</td>
</tr>
<tr>
<td>Drug-induced</td>
<td>3.9</td>
</tr>
<tr>
<td>Situational</td>
<td>2.4</td>
</tr>
<tr>
<td>Cerebrovascular event</td>
<td>2.2</td>
</tr>
<tr>
<td>Seizure</td>
<td>1.7</td>
</tr>
<tr>
<td>Postprandial hypotension</td>
<td>0.4</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>7.9</td>
</tr>
<tr>
<td>Unknown etiology</td>
<td>39.2</td>
</tr>
</tbody>
</table>
Pathophysiology

- Sudden decrease in cerebral blood flow (RAS, both hemispheres)
- **Multiple comorbid conditions** with age-related physiologic derangement
- HT, atherosclerotic vascular diseases -- > \(\downarrow\) baseline cerebral blood flow
- Taking multiple medications --> alter vascular tone or volume

*The investigation of syncope, Seizure (2004) 13, 537-548*
Pathophysiology

- Age-related cardiovascular changes
- Decrease ability to maintain extracellular volume
Age-Related Cardiovascular changes

- Baroreflex sensitivity diminishes with aging
  - Reduction in vascular response to hypotensive stimuli
  - A blunt of beta-adrenergic-mediated vasodilation
  - Unable to maintain cerebral blood flow by ↑ HR and vascular tone in Hypotension

- Increase in plasma norepinephrine
  - Impaired end – organ responsiveness to adrenergic stimulation with normal central and afferent components of the baroreflex circuit
Age-Related Cardiovascular changes

- More sensitive to effects of vasodilators and hypotensive drugs
- Exaggerated hypotension from volume loss, hemorrhage, upright posture

- Systolic HT ( > 30% of age over 75 yr)
  - diminish baroreflex sensitivity
  - Decrease vascular and ventricular compliance
  - Increase threshold for cerebral autoregulation
  - Decrease cerebral blood flow

*The investigation of syncope, Seizure (2004) 13, 537-548*
Decreased Ability to Maintain Extracellular Volume

- Impairment of sodium conservation when restricted salt intake
- Decrease basal plasma renin and aldosterone

\[ \Downarrow \]

- Increase susceptibility to orthostatic hypotension and syncope
Clinical classification

- Neurally mediated syndromes
  - Vasovagal syncope
  - Situational syncope
  - Carotid sinus syncope
  - Neuralgias
  - High altitude
  - Others (exercise, selected drugs)

- Orthostatic hypotension: autonomic dysfunction
  - Primary: multiple systems atrophy
  - Secondary: diabetes, amyloid, drugs

*The investigation of syncope, Seizure (2004) 13, 537-548*
Clinical classification

- **Cardiac syncope**: Decreased cardiac output
  - Obstruction to flow
    - LV
    - RV
  - Other heart disease
    - Pump failure
    - Tamponade, aortic dissection
  - Arrhythmias
    - Bradyarrhythmias
    - Tachyarrhythmias

- **Neurologic disease**
  - Migraines
  - TIAs
  - Seizures
  - Intermittent obstructive hydrocephalus

- **Metabolic syncope**
  - Hypoglycemia
  - Hypocalcemia

- **Psychogenic syncope**
  - Panic disorder
  - Conversion

*The investigation of syncope, Seizure (2004) 13, 537-548*
Diagnostic evaluation

1. Determining whether the patient had syncope
2. Risk stratification
3. Selective use of diagnostic tests
Determining whether the patient had syncope

- History taking: separate syncope from other symptoms
- Syncope VS Seizure

<table>
<thead>
<tr>
<th>Question</th>
<th>Points (if yes)</th>
</tr>
</thead>
<tbody>
<tr>
<td>At times do you wake with a cut tongue after your spells?</td>
<td>2</td>
</tr>
<tr>
<td>At times do you have a sense of déjà vu or jamais vu before your spells?</td>
<td>1</td>
</tr>
<tr>
<td>At times is emotional stress associated with losing consciousness?</td>
<td>1</td>
</tr>
<tr>
<td>Has anyone ever noted your head turning during a spell?</td>
<td>1</td>
</tr>
<tr>
<td>Has anyone ever noted that you are unresponsive, have unusual posturing or jerking limbs during your spells, or have no memory of your spells afterwards? (score as yes for any positive response)</td>
<td>1</td>
</tr>
<tr>
<td>Has anyone ever noted that you are confused after a spell?</td>
<td>1</td>
</tr>
<tr>
<td>Have you ever had lightheaded spells?</td>
<td>-2</td>
</tr>
<tr>
<td>At times do you sweat before your spells?</td>
<td>-2</td>
</tr>
<tr>
<td>Is prolonged sitting or standing associated with your spells?</td>
<td>-2</td>
</tr>
</tbody>
</table>

The patient has seizures if the point score is ≥ 1, and syncope if the point score is <1.

<table>
<thead>
<tr>
<th></th>
<th>Vasovagal syncope</th>
<th>Seizure</th>
<th>Cardiac syncope</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Trigger</strong></td>
<td>Common (upright, bathroom, blood, needles)</td>
<td>Rare (flashing lights, hyperventilation)</td>
<td>Rare, exertional (consider left ventricular outflow obstruction)</td>
</tr>
<tr>
<td><strong>Prodrome</strong></td>
<td>Almost always (presyncope)</td>
<td>Common (aura)</td>
<td>Uncommon or brief</td>
</tr>
<tr>
<td><strong>Onset</strong></td>
<td>Gradual (often minutes)</td>
<td>Usually sudden</td>
<td>Usually sudden</td>
</tr>
<tr>
<td><strong>Duration</strong></td>
<td>1–30 s</td>
<td>1–3 min</td>
<td>Variable</td>
</tr>
<tr>
<td><strong>Convulsive jerks</strong></td>
<td>Common (brief)</td>
<td>Common (prolonged)</td>
<td>Common (brief)</td>
</tr>
<tr>
<td><strong>Incontinence</strong></td>
<td>Uncommon</td>
<td>Common</td>
<td>Uncommon</td>
</tr>
<tr>
<td><strong>Lateral tongue bite</strong></td>
<td>Very rare</td>
<td>Common</td>
<td>Very rare</td>
</tr>
<tr>
<td><strong>Colour</strong></td>
<td>Very pale, cold skin</td>
<td>Pale or flushed (partial seizure); blue (tonic-clonic seizure)</td>
<td>Very pale, cold skin</td>
</tr>
<tr>
<td><strong>Post-ictal confusion</strong></td>
<td>Rare (wakes on floor)</td>
<td>Common (wakes in ambulance)</td>
<td>Rare (wakes on the floor)</td>
</tr>
<tr>
<td><strong>Recovery</strong></td>
<td>Quickly orientated</td>
<td>Slow (confused)</td>
<td>Quickly orientated</td>
</tr>
<tr>
<td></td>
<td>Fatigue (minutes-hours)</td>
<td>Fatigue (minutes-hours)</td>
<td>No fatigue</td>
</tr>
</tbody>
</table>
Risk stratification

- Prediction of risk of sudden death and LR of cardiac syncope
  - Cause of syncope
  - Underlying cardiac disease
  - Abnormal on EKG
- Decision for admission/Use of invasive testing

- Aortic stenosis
- Pulmonary hypertension
- CHF
- VHD
- HOCM
- Organic heart disease
- AV block
- Old MI
- WPW syndrome

High risk subset

Syncope in the elderly, Wishwa N Kapoor, 957-967
<table>
<thead>
<tr>
<th>Specific entities</th>
<th>Symptoms of finding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vasovagal</td>
<td>After sudden unexpected pain, fear, sound, smell</td>
</tr>
<tr>
<td></td>
<td>Prolonged standing at attention</td>
</tr>
<tr>
<td></td>
<td>Well-trained athlete after exertion (without heart disease)</td>
</tr>
<tr>
<td>Situational syncope</td>
<td>During or immediately after micturation, defecation, cough, swallow</td>
</tr>
<tr>
<td>Neurally mediated syncope with neuralgia</td>
<td>Syncope with throat and facial pain (glossopharyngeal or trigeminal neuralgia)</td>
</tr>
<tr>
<td>Carotid sinus syncope</td>
<td>Head rotation, pressure on carotid sinus</td>
</tr>
<tr>
<td>Orthostatic hypotension</td>
<td>Immediately upon standing</td>
</tr>
<tr>
<td>Drug induced</td>
<td>Medication that may lead to long QT syndrome</td>
</tr>
<tr>
<td>Migraines</td>
<td>Associated with headache</td>
</tr>
<tr>
<td>Seizure</td>
<td>Associated with headache, confusion after spell &gt; 5 min</td>
</tr>
<tr>
<td>TIA, basilar migraine, subclavian steal</td>
<td>Vertigo, dysarthria, diplopia</td>
</tr>
<tr>
<td>Subclavian steal, aortic dissection</td>
<td>Different BP or pulse in 2 arms</td>
</tr>
<tr>
<td>Atrial myxoma or thrombus</td>
<td>Syncope and murmur with changing position</td>
</tr>
<tr>
<td>AS, Pulmonary HT, MS, HOCM, CAD</td>
<td>Syncope with exertion</td>
</tr>
<tr>
<td>Long QT syndrome, Brugada syndrome</td>
<td>Family history of sudden death</td>
</tr>
<tr>
<td>Psychiatric illness</td>
<td>Frequent syncope, somatic complaint, no heart disease</td>
</tr>
</tbody>
</table>
Diagnostic tests

- EKG
- Echocardiography
- Exercise stress test
Diagnostic tests: arrhythmia detection

- Prolonged Electrocardiographic monitoring
- Ambulatory monitor
- 4% of symptomatic patients have arrhythmias
- 17% of patients could be excluded for arrhythmia
- 80% no symptoms, found arrhythmia
- Extend duration to 72 hr → ↑ yield

- Loop monitoring
- Electrophysiologic study
  - Predictor of positive test
    - Organic heart disease
    - PVCs by EKG
    - Nonsustained VT by Holter
    - Sinus bradycardia
    - 1st degree AV block, BBB
  - Predictor of negative test
    - Absence of heart disease
    - LVEF > 40%
    - Normal EKG, Holter monitor
    - No injury during syncope
    - Multiple or prolong episodes (> 5min) of syncope

*Syncope in the elderly, Wishwa N. Kapoor, 957-967*
Diagnostic test

- **Tilt table testing**
  - Lying supine 30 min --> tilted to 60 – 80 for < 45 min
  - Report any symptoms , BP , HR
  - **Positive Test =** pre-syncopal or syncopal symptoms +
    - BP fall > 20 mmHg (: vasodepressor response)
    - Bradycardia HR fall >10% of baseline (: cardio-inhibitory response)
  - **Negative test =** hemodynamic change without symptoms
  - If non diagnostic test --> pharmacological provocation (nitrate, Isoproterenol)
  - Medication Increase the yield to 64% (50% drug-free HUTT)

Tilt table test

Indication
- Recurrent unexplained syncope (not suspected structural heart disease)
- Single episode if:
  - High risk setting
  - The result alter management --> permanent pacemaker therapy
    (in cardio-inhibitory vasovagal syncope)

Relative C/I
- proximal coronary artery disease
- Critical Mitral stenosis
- Severe Lt ventricular outflow obstruction
- Severe cerebrovascular disease

Approach to evaluation

History & Physical examination
- EKG

Further testing

Heart disease & EKG abnormalities

Cardiac assessment
- Higher LR of arrhythmic syncope

Stress test/echocardiogram

Multiple abnormalities

Empiric treatment of factors before invasive work up

Prolonged electrocardiographic monitoring
- EPS

Negative:
- most likely vasovagal syncope

Low likelihood for arrhythmia

Positive
- Ambulatory or Loop monitoring

Favorable
- Upright tilt test

Syncope in the elderly, Wishwa N. Kapoor, 957-967
## Management: Hospital Admission

### For Diagnostic Evaluation
- Structural heart diseases
  - CAD, CHF, congenital / valvular heart disease
  - Hx of ventricular arrhythmias
- Arrhythmias or ischemia
  - Palpitation, Chest pain, exertional syncope
- EKG abnormalities
- Neurologic diseases
  - New stroke or focal neurologic findings

### For Treatment
- Structural heart diseases
  - Acute MI, Pulmonary embolism
- Orthostatic hypotension
- Acute severe volume loss
- Mod to severe chronic
- Treatment of multiple coexist abnormalities
- Discontinuation of drug or drug adjustment
  - Anaphylaxis, bradyarrhythmias, drugs induced prolonged QT

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*Syncope in the elderly, Wishwa N. Kapoor, 957-967*
Neurally Mediated Syndromes

- “Neurocardiogenic syncope”
- Syncope from reflex mechanisms
- Inappropriate vasodilatation and/or bradycardia

- Vasovagal syncope
- Situational syncope
- Carotid sinus syncope

Syncope in the elderly, Wishwa N. Kapoor, 957-967
Neurally Mediated Syndromes

- Pathophysiology associated with:
  - Pain receptors
  - Mechanical stimuli receptors
  - Temperature receptors

- Afferent signals were triggered to various neurally mediated syncopal syndromes
  - Carotid sinus hypersensitivity
    - Carotid artery baroreceptors
  - Vasovagal syncope
    - Lt ventricular baroreceptors (mechanoreceptors)

*Syncope in the elderly, Wishwa N. Kapoor, 957-967*
Vasovagal syncope

- **Precipitating factors**
  - Emotional stress
  - Anxiety, metal anguish, trauma, physical pain, sight of blood
  - Accident, warm environment
  - Prolonged standing
  - **Vasomotor medication**

- **Manifestations**
  - Prodrome or aura
  - Loss of consciousness
  - Post syncopal phase
Menifestration

- Prodrome symptoms
  - Extreme fatigue, weakness
  - Diaphoresis
  - Nausea
  - Visual defects, visual and aural hallucinations
  - Dizziness, vertigo
  - Headache
  - Abdominal discomfort
  - Dysarthria
  - Paresthesias

- Prodrome duration
  - Few seconds to several min

- Syncope period: brief
  - Involuntary movements
  - Pallor, sweating, cold skin
  - Incontinence

- Recovery: Rapid with some protracted symptoms
  - Confusion, disorientation, nausea, headache, dizziness
  - General sense of ill health for hours
Pathophysiology

- Upright position
- Baroreceptors at aortic arch, carotid sinus
- Mechanoreceptor at ventricle
- Bezold-Jarisch Reflex
  - Peripheral vasodilatation (Hypotension)
  - Bradycardia
<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1 Mixed</td>
<td>Ventricular rate during syncope ≥40 bpm or falls to &lt;40 bpm for &lt;10 s ± asystole for &lt;3 s. BP falls prior to heart rate.</td>
</tr>
<tr>
<td>Type 2A Cardioinhibitory</td>
<td>Ventricular rate during syncope &lt;40 bpm for &gt;20 sec or asystole for &gt;3 s. BP falls prior to heart rate.</td>
</tr>
<tr>
<td>Type 2B Cardioinhibitory</td>
<td>Ventricular rate at syncope &lt;40 bpm for &gt;20 s or asystole for &gt;3 s. BP falls to &lt;80 mmHg systolic at or after rapid fall in heart rate (as above).</td>
</tr>
<tr>
<td>Type 3 Pure vasodepressor</td>
<td>Heart rate does not fall more than 10% (20 bpm) from its peak during syncope. Fall in BP precipitates syncope.</td>
</tr>
</tbody>
</table>

Exceptions to this classification include:
- chronotropic incompetence, or absence of a rise in HR during head-up tilting, may be caused by SA disease
- excessive heart rate rise (>130 bpm) during tilt can be associated with type 1 – 3 owing to different pathophysiology
- carotid sinus hypersensitivity can be associated with any of subtypes
Treatment

- Education:
  - Avoiding the precipitate factors and culprit vasodilator medication
  - Elastic support hose
  - Relaxation techniques
  - Increase salt intake

- Training by repeating the Head-Up tilting (short intervals)
Treatment

- Medication: few controlled studies
  - Beta-blocker: Metoprolol 50-200 mg/day
    - Atenolol 25-200 mg/day
    - Propanolol 40-160 mg/day
  - Anticholinergic: transdermal scopolamine one patch q 2-3 days
    - Disopyramide 200-600 mg/day
    - Paroxetine 20-40 mg/day
    - Theophylline 6-12 mg/day
  - Fludocortisone acetate 0.1-1 mg/day
- Dual chamber atrioventricular pacing
- Medication failure

*Syncope in the elderly, Wishwa N. Kapoor*, 957-967
Post prandial hypotension
Postprandial hypotension

- Fall in SBP $\geq 20$ mmHg within 2 hours of the start of meal ingestion
- SBP < 90 mmHg if the pre-ingestion SBP > 100 mmHg
- Symptomatic postprandial hypotension
- Occur at any time from 15 to 75 min (30 – 60 min) after meal ingestion
Epidemiology

- Hypertension
- Primary autonomic failure
  - Multiple system atrophy
  - Diabetic peripheral and autonomic neuropathy
- Idiopathic parkinson’s disease
- Renal failure (on dialysis)
Epidemiology

- Hypertension
  - Young and old patients
  - Elderly hypertensive patients > age-matched normotensive patients
  - Asymptomatic cerebrovascular damage (MRI brain)

>500 elderly subjects with isolated systolic hypertension
2/3 postprandial fall in BP
1/4 BP fall >16 mmHg
Epidemiology

- Type of meal: carbohydrates, fats
- Amounts of food
- Time of day
- Temperature of the meal: warm meals
- Position of patients: sitting and standing
Clinical relevance and clinical features

- Fall
- Postprandial hypotension and orthostatic hypotension

Coexist

- Hypertension VS autonomic failure

Do not always occur together

- Unexplained syncope
  - none had orthostatic hypotension
  - $\frac{1}{2}$ had postprandial hypotension

Postprandial reduction in blood pressure

- Orthostatic fall on tilting

Clinics in geriatric medicine, volume 18, Number 2, May 2002
Clinical features

- Syncope
- Collapse
- Falls
- Angina
- Generalized weakness
- Dizziness
- Visual disturbance
- TIA
Pathophysiology

- Normal postprandial hemodynamic responses

![Diagram]

- Splanchnic blood pooling
  - ↓ Total SVR
  - ↑ sympathetic nervous system activity
  - ↑ mesenteric a. resistance
    - ↓ flow
  - ↑ HR
  - ↑ Plasma NE
  - ↑ Muscle nerve activity
Abnormal postprandial hemodynamic responses

**Pathophysiology**

- Excessive splanchnic blood pooling
  - **↓** Total SVR
  - **Impaired** sympathetic nervous system activity
    - **↓** Plasma NE
    - **↓** Muscle nerve activity
  - **↑** mesenteric a. resistance
    - **↓** flow

*Blunt effect*
Pathophysiology

- Hydration status:
  - Diuretics used in heart failure with preserved systolic function
  - Renal failure during dialysis

- Vasoactive peptides
  - Vasoactive intestinal peptides
  - Somatostatin
  - Insulin

- Evidence on treatment
  - Somatostatin analogues
  - Caffeine
  - Oral glucose loading
Normal response to meal

Hypotensive mechanism
- Increase bowel blood volume
- Insulin release vasodilation
- GIT vasoactive peptides (mesenteric ± peripheral vasodilation)

Hypertensive mechanism
- Increase HR (baroreceptor)
- Vasoconstriction (sympathetic)

Stable blood pressure
Continue perfusion of vital organs

Abnormal response to meal

Hypotensive mechanism
- Increase bowel blood volume
- Insulin excess vasodilation
- GIT peptides excess vasodilation

Hypertensive mechanism
- failure to Increase HR (baroreceptor dysfunction)
- failure to Vasoconstriction (inadequate sympathetic response)

Reduction in blood pressure
Reduced perfusion of vital organs

Clinics in geriatric medicine, volume 18, Number 2, May 2002
Fig. 2. A mechanistic approach to the management of postprandial hypotension.

- Avoid prolonged standing/sitting postprandially
- Liberal salt intake
- Adequate fluid intake
- Stop diuretics
- Fludrocortisone
- Indomethicin

- Smaller meal sizes
- Lower carbohydrate content
- No periprandial alcoholic beverages
- Octreotide

- Resistance vessels

- Capacitance vessels

- Avoid hypotensive medicines at meal times
- Caffeine
- Alpha adrenergic agonists

**VASOMOTOR CENTRE**

**PRESSOR (LRN)**

**DEPRESSOR (NTS)**

**MEDULLA**

**Gut**
Table 2. Approach to the Treatment of Postprandial Hypotension

Inform patient about danger associated with meal-induced hypotension, particularly the risk for falling and syncope within 15 to 90 minutes after meals.

Discontinue therapy with any unnecessary medications that lower blood pressure. If the patient has sustained hypertension when blood pressure is measured before and after meals, institute treatment but monitor postprandial blood pressure to avoid hypotension.

Administer hypotensive medications between rather than during meals.

Have patient avoid sitting for prolonged periods or standing still after meals. Check blood pressure after a postprandial walk; if it returns to normal, patient should walk after meals. Otherwise, patient should lie semi-recumbent for 90 minutes after a meal.

Maintain patient’s intravascular volume.

- Advise a liberal salt intake when possible.
- Ensure adequate fluid intake.
- Discontinue use of diuretics.

Adjust the size and composition of patient’s meals.
- Encourage frequent small meals.
- Encourage limited carbohydrate content.

Advise avoiding meals during hemodialysis.

Advise avoiding alcoholic beverages before and after meals.

Institute therapy with pharmacologic agents.

- Caffeine, 250 mg (two cups of coffee), before a meal. Controversial, but might benefit some patients.
- Octreotide, 50 μg subcutaneously, administered 30 minutes before each meal.
- Fludrocortisone, 0.1 to 1.0 mg daily. Has not been studied for postprandial hypotension but is useful for orthostatic hypotension.
- Indomethacin, 25 to 50 mg orally, three times a day. 
  α1-Adrenergic agonists such as midodrine, 2.5 to 10.0 mg orally three times a day, or phenylephrine, 60 mg orally every 6 to 12 hours. Only empirical data are available for postprandial hypotension, but useful for orthostatic hypotension.