Syncope

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www.brain101.info
Syncope

- Definition
- Epidemiology
- Etiology
- Diagnosis & Evaluation Options
- Specific Conditions
Syncope - Definitions

- ACP 1997 - Transient loss of consciousness (LOC) with loss of postural tone, from which recovery is spontaneous.
- ACEP 2001 - Sudden, transient LOC with inability to maintain tone & is distinct from seizures, coma, vertigo, hypoglycemia and other states of altered consciousness.
- ESC 2001 - Transient, self limited LOC with a relatively rapid onset and usually leading to fainting; the subsequent recovery is spontaneous, complete, and usually prompt.
- AFP 2005 - Transient loss of consciousness, usually accompanied by falling, and with spontaneous recovery.
Syncope:
A Symptom…Not a Diagnosis

- Self-limited loss of consciousness and postural tone
- Relatively rapid onset
- Variable warning symptoms
- Spontaneous complete recovery
The Significance of Syncope

The only difference between syncope and sudden death is that in one you wake up.¹

The Significance of Syncope

1 More than 1 million patients in the U.S.  
2 More than 500,000 new patients per year  
3 1–6% of admissions  
4 3% of emergency room visits per year  

1 National Disease and Therapeutic Index on Syncope and Collapse, ICD-9-CM 780.2, IMS America, 1997  
Syncope Reported Frequency

- Individuals <18 yrs: 15%
- Military Population 17-46 yrs: 20-25%
- Individuals 40-59 yrs*: 16-19%
- Individuals >70 yrs*: 23%

*during a 10-year period

The Significance of Syncope

- 500,000 new syncope patients each year
- 170,000 have recurrent syncope
- 70,000 have recurrent, infrequent, unexplained syncope

5 National Disease and Therapeutic Index, IMS America, Syncope and Collapse #780.2; Jan 1997-Dec 1997.
Some causes of syncope are potentially fatal
Cardiac causes of syncope have the highest mortality rates (5 year mortality - 50 %, 1 year mortality - 30 %)

Impact of Syncope

Anxiety/Depression: 73% ¹
Alter Daily Activities: 71% ²
Restricted Driving: 60% ²
Change Employment: 37% ²

Syncope - Mechanism

- Global cerebral hypoperfusion
- Interruption of sympathetic outflow
- Increased vagal tone
- Other mechanisms - edema, cerebral autoregulation, central serotonin pathways.

The trigger for the switch in autonomic response remains one of the unresolved mysteries in cardiovascular physiology*

Hainsworth. Syncope: what is the trigger? Heart 2003; 89: 123-124
Syncope - Etiology

- Reflex mediated - 40%
- Unexplained - 25%
- Cardiac - 15%
- Others - 20%
  - Orthostatic Hypotension
  - Cerebrovascular / Neurologic
  - Psychiatric
  - Hypoglycemia
  - Medications
Syncope - Etiology

Reflex (Neurally) Mediated
- Vasovagal (common faint)
- Carotid Sinus
- Neuralgia
- Situational
  - Cough
  - Post-micturition
  - 24%

Orthostatic
- Drug Induced
- ANS Failure
  - Primary
  - Secondary
  - 11%

Cardiac Arrhythmia
- Brady
  - Sick sinus
  - AV block
- Tachy
  - VT
  - SVT
- Long QT Syndrome
  - 14%

Structural Cardio-Pulmonary
- Aortic Stenosis
- HOCM
- Pulmonary Hypertension
  - 4%

Non-Cardiovascular
- Psychogenic
- Metabolic e.g. hyper-ventilation
- Neurological
  - 12%

Unknown Cause = 34%

DG Benditt, UM Cardiac Arrhythmia Center
# Causes of Syncope

<table>
<thead>
<tr>
<th>Cause</th>
<th>Prevalence (Mean) %</th>
<th>Prevalence (Range) %</th>
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</thead>
<tbody>
<tr>
<td>Reflex-mediated:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Vasovagal</td>
<td>18</td>
<td>8-37</td>
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<tr>
<td>• Situational</td>
<td>5</td>
<td>1-8</td>
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<tr>
<td>Carotid Sinus</td>
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<td>0-4</td>
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<tr>
<td>Orthostatic hypotension</td>
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<td>4-10</td>
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<tr>
<td>Medications</td>
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<tr>
<td>Psychiatric</td>
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<td>1-7</td>
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<tr>
<td>Neurological</td>
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<td>3-32</td>
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<tr>
<td>Organic Heart Disease</td>
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<tr>
<td>Cardiac Arrhythmias</td>
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<td>4-38</td>
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<tr>
<td>Unknown</td>
<td>34</td>
<td>13-41</td>
</tr>
</tbody>
</table>

Causes of Syncope-like States

- Migraine*
- Acute hypoxemia*
- Hyperventilation*
- Somatization disorder (psychogenic syncope)
- Acute Intoxication (e.g., alcohol)
- Seizures
- Hypoglycemia
- Sleep disorders

* may cause ‘true’ syncope
**Syncope**

**Diagnostic Objectives**

- Distinguish ‘True’ Syncope from other ‘Loss of Consciousness’ spells:
  - Seizures
  - Psychiatric disturbances
- Establish the cause of syncope with sufficient certainty to:
  - Assess prognosis confidently
  - Initiate effective preventive treatment
Initial Evaluation (Clinic/Emergency Dept.)

- Detailed history
- Physical examination
- 12-lead ECG
- Echocardiogram (as available)
Syncope
Basic Diagnostic Steps

- Detailed History & Physical
  - Document details of events
  - Assess frequency, severity
  - Obtain careful family history

- Heart disease present?
  - Physical exam
  - ECG: long QT, WPW, conduction system disease
  - Echo: LV function, valve status, HOCM

- Follow a diagnostic plan...
Syncope
Evaluation and Differential Diagnosis

History – What to Look for

- Complete Description
  - From patient and observers
- Type of Onset
- Duration of Attacks
- Posture
- Associated Symptoms
- Sequelae
12-Lead ECG

- Normal or Abnormal?
  - Acute MI
  - Severe Sinus Bradycardia/pause
  - AV Block
  - Tachyarrhythmia (SVT, VT)
  - Preexcitation (WPW), Long QT, Brugada

- Short sampling window (approx. 12 sec)
Carotid Sinus Massage

- **Site:**
  - Carotid arterial pulse just below thyroid cartilage

- **Method:**
  - Right followed by left, pause between
  - Massage, NOT occlusion
  - Duration: 5-10 sec
  - Posture – supine & erect
Carotid Sinus Massage

- **Outcome:**
  - 3 sec asystole and/or 50 mmHg fall in systolic blood pressure with reproduction of symptoms = Carotid Sinus Syndrome (CSS)

- **Contraindications**
  - Carotid bruit, known significant carotid arterial disease, previous CVA, MI last 3 months

- **Risks**
  - 1 in 5000 massages complicated by TIA
Head-up Tilt Test (HUT)

- Unmasks VVS susceptibility
- Reproduces symptoms
- Patient learns VVS warning symptoms
- Physician is better able to give prognostic / treatment advice
Electroencephalogram

- Not a first line of testing
- Syncope from Seizures
  - Abnormal in the interval between two attacks – Epilepsy
  - Normal – Syncope
## Ambulatory ECG

<table>
<thead>
<tr>
<th>Method</th>
<th>Comments</th>
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<tbody>
<tr>
<td>Holter (24-48 hours)</td>
<td>Useful for infrequent events</td>
</tr>
<tr>
<td>Event Recorder</td>
<td>• Useful for infrequent events</td>
</tr>
<tr>
<td></td>
<td>• Limited value in sudden LOC</td>
</tr>
<tr>
<td>Loop Recorder</td>
<td>• Useful for infrequent events</td>
</tr>
<tr>
<td></td>
<td>• Implantable type more convenient (ILR)</td>
</tr>
<tr>
<td>Wireless (internet) Event Monitoring</td>
<td>Initiated</td>
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</table>
Reveal® Plus
Insertable Loop Recorder

Patient Activator  Reveal® Plus ILR  9790 Programmer
Conventional EP Testing in Syncope

- Limited utility in syncope evaluation

- Most useful in patients with structural heart disease
  - Heart disease ……50-80%
  - No Heart disease …18-50%

- Relatively ineffective for assessing bradyarrhythmias

Diagnostic Limitations

- Difficult to correlate spontaneous events and laboratory findings
- Often must settle for an attributable cause
- Unknowns remain 20-30% 

Challenges of Syncope

- **Cost**
  - Cost/year
  - Cost/diagnosis

- **Quality of Life Implications**
  - Work/financial
  - Mobility (automobiles)
  - Psychological

- **Diagnosis & Treatment**
  - Diagnostic yield and repeatability of tests
  - Frequency and clustering of events
  - Difficulty in managing/treating/controlling future events
  - Appropriate risk stratification
  - Complex Etiology
Unexplained Syncope Diagnosis

History and Physical Exam
Surface ECG

Neurological Testing
- Head CT Scan
- Carotid Doppler
- MRI
- Skull Films
- Brain Scan
- EEG

CV Syncope Workup
- Holter
- ELR or ILR
- Tilt Table
- Echo
- EPS

Psychological Evaluation

ENT Evaluation

Endocrine Evaluation

Other CV Testing
- Angiogram
- Exercise Test
- SAECG

Typical Cardiovascular Diagnostic Pathway

**Syncope**

History and Physical, ECG

- **Known SHD**
  - Echo
    - EPS
      - Tilt/ILR
      - Treat

- **No SHD**
  - > 30 days; > 2 Events
    - Tilt
    - ILR
  - < 30 days
    - Tilt
    - Holter/ ELR
    - ILR

Adapted from:
Specific Conditions
Neurally-Mediated Reflex Syncope (NMS)

- Vasovagal syncope (VVS)
- Carotid sinus syndrome (CSS)
- Situational syncope
  - post-micturition
  - cough
  - swallow
  - defecation
  - blood drawing
  - etc.
NM Reflex Syncope: Pathophysiology

- Multiple triggers
- Variable contribution of vasodilatation and bradycardia
Neurally Mediated Physiologic Reflex Mechanism with two Components:

- Cardioinhibitory (↓ HR)
- Vasodepressor (↓ BP)

Both components are usually present
Diagnosing VVS

- Patient history and physical exam
- Positive tilt table test
  - Overnight fast
  - ECG
  - Blood pressure
  - Supine and upright
  - Tilt to 60-80 degrees
  - Isoproterenol
  - Re-tilt

DG Benditt, Tilt Table Testing, 1996.
Management Strategies for VVS

- Optimal management strategies for VVS are a source of debate
  - Patient education, reassurance, instruction
  - Fluids, salt, diet
  - Tilt Training
  - Support hose
- Drug therapies
- Pacing
  - Class II indication for VVS patients with positive HUT and cardioinhibitory or mixed reflex
VVS: Treatment Overview

- **Education**
  - symptom recognition
  - reassurance
  - situation avoidance

- **Tilt-Training**
  - prescribed upright posture

- **Pharmacologic Agents**
  - salt/volume management
  - beta-adrenergic blockers
  - SSRIs
  - vasoconstrictors (e.g., midodrine)

- **Cardiac Pacemakers**
VVS: Tilt-Training

- **Objectives**
  - Enhance Orthostatic Tolerance
  - Diminish Excessive Autonomic Reflex Activity
  - Reduce Syncope Susceptibility / Recurrences

- **Technique**
  - Prescribed Periods of Upright Posture
  - Progressive Increased Duration
VVS: Pharmacologic Rx

- **Salt /Volume**
  - Salt tablets, ‘sport’ drinks, fludrocortisone

- **Beta-adrenergic blockers**
  - 1 positive controlled trial (atenolol),
  - 1 on-going RCT (POST)

- **Disopyramide**

- **SSRIs**
  - 1 controlled trial

- **Vasoconstrictors (e.g., midodrine)**
  - 1 negative controlled trial (etilephrine)
Recent clinical studies demonstrated benefits of pacing in select VVS patients:

- VPS I
- VASIS
- SYDIT
- VPS II – Phase I
- ROME VVS Trial
VVS Pacing Trials Conclusions

DDD pacing reduces the risk of syncope in patients with recurrent, refractory, highly-symptomatic, cardioinhibitory vasovagal syncope.
Carotid Sinus Syndrome (CSS)

- Syncope clearly associated with carotid sinus stimulation is rare (≤1% of syncope)

- CSS may be an important cause of unexplained syncope / falls in older individuals
Etiology of CSS

- Sensory nerve endings in the carotid sinus walls respond to deformation
- “Deafferentation” of neck muscles may contribute
- Increased afferent signals to brain stem
- Reflex increase in efferent vagal activity and diminution of sympathetic tone results in bradycardia and vasodilation
Carotid Sinus Hypersensitivity (CSH)

- Abnormal response to CSM
- Absence of symptoms attributable to CSS
- CSH reported frequent in ‘fallers’ (Kenny)

CSH ≠ CSS
CSS and Falls in the Elderly

- 30% of people >65 yrs of age fall each year\(^1\)
  - Total is 9,000,000 people in USA
  - Approximately 10% of falls in elderly persons are due to syncope\(^2\)
- 50% of fallers have documented recurrence\(^3\)
- Prevalence of CSS among frequent and unexplained fallers unknown but...
  - CSH present in 23% of >50 yrs fallers presenting at ER \(^3\)

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\(^3\) Richardson DA, Bexton RS, et al. Prevalence of cardioinhibitory carotid sinus hypersensitivity in patients 50 years or over presenting to the Accident and Emergency Department with “unexplained” or “recurrent” falls. *PACE* 1997
Role of Pacing in CSS -- Syncope Recurrence Rate

Class I indication for pacing (AHA and BPEG)
Limit pacing to CSS that is:
- Cardioinhibitory
- Mixed

DDD/DDI superior to VVI
(Mean follow-up = 6 months)

Principal Causes of Orthostatic Syncope

- **Drug-induced (very common)**
  - diuretics
  - vasodilators

- **Primary autonomic failure**
  - multiple system atrophy
  - Parkinsonism

- **Secondary autonomic failure**
  - diabetes
  - alcohol
  - amyloid

- **Alcohol**
  - orthostatic intolerance apart from neuropathy
Syncope Due to Arrhythmia or Structural CV Disease: General Rules

- Often life-threatening and/or exposes patient to high risk of injury
- May be warning of critical CV disease
  - Aortic stenosis, Myocardial ischemia, Pulmonary hypertension
- Assess culprit arrhythmia / structural abnormality aggressively
- Initiate treatment promptly
Principal Causes of Syncope due to Structural Cardiovascular Disease

- Acute MI / Ischemia
  - Acquired coronary artery disease
  - Congenital coronary artery anomalies
- HOCM
- Acute aortic dissection
- Pericardial disease / tamponade
- Pulmonary embolus / pulmonary hypertension
- Valvular abnormalities
  - Aortic stenosis, Atrial myxoma
Syncope Due to Cardiac Arrhythmias

- **Bradyarrhythmias**
  - Sinus arrest, exit block
  - High grade or acute complete AV block

- **Tachyarrhythmias**
  - Atrial fibrillation / flutter with rapid ventricular rate (e.g. WPW syndrome)
  - Paroxysmal SVT or VT
  - Torsades de pointes
Rhythms During Recurrent Syncope

- Normal Sinus Rhythm: 58%
- Bradycardia: 36%
- Tachyarrhythmia: 6%

Treatment of Syncope Due to Bradyarrhythmia

- Class I indication for pacing using dual-chamber system wherever adequate atrial rhythm is available
- Ventricular pacing in atrial fibrillation with slow ventricular response
Treatment of Syncope Due to Tachyarrhythmia

- **Atrial Tachyarrhythmias;**
  - AVRT due to accessory pathway – ablate pathway
  - AVNRT – ablate AV nodal slow pathway
  - Atrial fibrillation – Pacing, linear / focal ablation, ICD selected pts
  - Atrial flutter – Ablation of reentrant circuit

- **Ventricular Tachyarrhythmias;**
  - Ventricular tachycardia – ICD or ablation where appropriate
  - Torsades de Pointes – withdraw offending Rx or ICD (long-QT/Brugada)

- Drug therapy may be an alternative in many cases
Conclusion

Syncope is a common symptom, often with dramatic consequences, which deserves thorough investigation and appropriate treatment of its cause.
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Discord in the Evaluation of Syncope

Neurologist

Cardiologist