Syncope
What to do when the lights go out?

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Learning Objectives

• Evaluate patients who present with syncope to determine cardiac or non-cardiac causes.
• Prepare diagnostic plans for patients who present with neurocardiogenic forms of syncope, which may include conducting a differential diagnosis of syncope.
• Conduct appropriate tests, such as EKGs, exercise stress testing, tilt tests or blood screenings, to diagnose underlying conditions in patients whose EKGs and cardiac tests are normal.

What do you really want to know?

• Dangerous etiology vs. Benign etiology

“Those who suffer from frequent and strong faints without any manifest cause die suddenly”

Hippocrates (460 - 375 BC)

What do you really want to know?

Dangerous etiology vs. Benign etiology

Medical-legal peril vs. None

Admit vs. discharge to home?

What work-up is needed?
### Syncope

<table>
<thead>
<tr>
<th>Definition</th>
<th>- very useful</th>
</tr>
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<td>Pathophysiology</td>
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<td>- that are useful</td>
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<td>Tests/Clinical Policies</td>
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<td>- that are worrisome!</td>
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</table>

#### Conclusions and outpatient evaluation

This is a summary of the document content. The main points are:

**Definition:**

A brief loss of consciousness associated with an inability to maintain postural tone that spontaneously and completely resolves without medical intervention.

- **Brief:** not asleep/intoxicated, not post-ictal
- **Spontaneous:** no intervention needed, so rarely => "hypoglycemia"
- **Completely:** no neurologic deficit, return to baseline

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Syncope - Pathophysiology

- Global cerebral hypoperfusion

Forget the "TIA" diagnosis/eval

Syncope - Definition

- very useful

Syncope - Pathophysiology

- very useful

Syncope - H & P elements

- that are useful (RISK FACTORS)

Syncope - Tests/Clinical Policies

- that might be useful

Syncope - Clinical Decision Rule

- that might be useful

Syncope - EKG’s

- that are worrisome!

Conclusions and outpatient evaluation

Syncope - History

High - risk Lower - risk
Older age younger age
(+) CV diagnosis (-) CV diagnosis
(+) CHF (-) CHF
(+) Family Hx (-) Family Hx
No prodrome (+) prodrome
Supine position
Assoc. with exertion (-) exertion
(think structural outflow obstruction)

Syncope - Age does matter!!!

• Methods: prospective study, ED pts (+) syncope
• Results: 477 patients, 97% f/u @14 days
  – 80 (18%) patients with “serious event”

<table>
<thead>
<tr>
<th>AGE</th>
<th># (%) serious outcome</th>
<th>Odds Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 40</td>
<td>4/141 (3%)</td>
<td>1.0</td>
</tr>
<tr>
<td>40-59</td>
<td>15/112 (13%)</td>
<td>2.7</td>
</tr>
<tr>
<td>60-79</td>
<td>30/115 (26%)</td>
<td>3.8</td>
</tr>
<tr>
<td>80+</td>
<td>31/109 (28%)</td>
<td>3.9</td>
</tr>
</tbody>
</table>

Note: 63/80 events were noted in ED


Incidence Rates of Syncope According to Age and Sex

Syncope - Step 1: History

High - risk Lower - risk
Older age younger age
(+) CV diagnosis (-) CV diagnosis
(+) CHF (-) CHF
(+) Family Hx (-) Family Hx
No prodrome (+) prodrome
Supine position
Assoc. with exertion (-) exertion
(think structural outflow obstruction)
**CHF and Sudden Death**

**Methods:** Retrospective study, Kaiser data, 02-06
- 22,189 patients with 23,951 syncope episodes
- 307 deaths in 30 days

**Results:**
- CHF: (age 18-59) Hazard Ratio 14.3
- (age 60-79) 3.1
- (age 80+) 2.3
- Diabetes 1.5
- Seizure 1.6
- Dementia 1.4

30-day Death rate:
- 0.2% < 60yrs without CHF
- 2.5% all ages with CHF

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**Syncope - Step 1: History**

**High - risk**
- Older age
- (+) CV diagnosis
- (+) CHF

**Lower - risk**
- Younger age
- (-) CV diagnosis
- (-) CHF

- (+) Family Hx
- (-) Family Hx
- No prodrome
- (+) prodrome
- Supine position
- Assoc. with exertion (-) exertion

*(think structural outflow obstruction)*

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**Syncope - Step 2 in History**

The episode....

Ask observers
- Duration
- Interventions required
- Seizure activity, tongue biting, incontinence

**Syncope vs. Seizure**

- Tongue biting
- Head turning/posturing
- No memory of LOC
- LOC assoc. with stress
- Cyanosis observed
- Limb jerking observed
- Postictal confusion
- Postictal headache

- Presyncope/prodrome
- Warmth before spell
- Remembered LOC
- Prolonged sitting or standing
- Any chest pain
- Palpitations
- Dypsnea

**Syncope vs. Seizure**

**Suggests seizure**
- Tongue biting
- Head turning/posturing
- No memory of LOC
- LOC assoc. with stress
- Cyanosis observed
- Limb jerking observed
- Postictal confusion
- Postictal headache

**Suggests syncope**
- Presyncope/prodome
- Warmth before spell
- Remembered LOC
- Prolonged sitting or standing
- Any chest pain
- Palpitations
- Dypsnea

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**Syncope History - Step 1: Risk Factors**

- **Step 2: The episode**

**Step 3: What are the meds?**

Syncope Clinic, Duke Univ
- 70 pts - 13% of syncope due to meds

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**Syncope vs. Seizure**

Don’t confuse with “*convulsive syncope*”
- 0.03% of all blood donors
- 12% of all syncope
- Men > women
- Individual variable response to global cerebral hypoperfusion

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Syncope History-

Step 3: What are the meds?

Drugs that cause hypotension
– Alpha blockers, diuretics

Drugs that cause bradycardias
– B-blockers, Ca+ channel blockers,
  – Alzheimer meds?

Drugs that cause prolonged QT

Cholinesterase inhibitors and syncope

- Methods: population-based cohort study,
  - Location: Ontario, Canada; 2002-2004
  - Patients with dx: dementia

- Results:
  
<table>
<thead>
<tr>
<th></th>
<th>Controls</th>
<th>(+) cholinesterase</th>
</tr>
</thead>
<tbody>
<tr>
<td>n=61,499</td>
<td>n= 19,803</td>
<td></td>
</tr>
</tbody>
</table>

  - Hospital visits for syncope: 1.76x (1.57-1.98)
  - Bradycardia: 1.69x (1.32-2.15)
  - Pacer insertion: 1.49x (1.12-2.00)
  - Hip fracture: 1.18x (1.04-1.34)


Orthostatic hypotension

Defined: 20mmHg drop in BPsystanding

May indicate:
– volume depletion
– Cardiac pump failure
– Autonomic insufficiency
– Medications

However, it is present in asymptomatic pts

40% in age >70
23% in age <60


Drugs that can cause prolong QTc/torsades

<table>
<thead>
<tr>
<th>Antiarrhythmics</th>
<th>Macrolides</th>
<th>TCA's/SSRI's</th>
<th>Antipsychotic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amiodarone</td>
<td>Erythromycin</td>
<td>Amitriptyline</td>
<td>Droperidol</td>
</tr>
<tr>
<td>Disopyramide</td>
<td>Clarithromycin</td>
<td>Desipramine</td>
<td>Haloperidol</td>
</tr>
<tr>
<td>Sotalol</td>
<td>Azithromycin</td>
<td>Doxepin</td>
<td>Pimozide</td>
</tr>
<tr>
<td>Flecaïnide</td>
<td>Metliamycin</td>
<td>Fluoxetine</td>
<td>Quetiapine</td>
</tr>
<tr>
<td>Buhtide</td>
<td>Levoteridol</td>
<td>Imipramine</td>
<td>Risperidone</td>
</tr>
<tr>
<td>Ppropafenone</td>
<td>Quinolines</td>
<td>Paroxetine</td>
<td>Perazine</td>
</tr>
<tr>
<td>Quinidine Anti-arrhythmia</td>
<td>Ciprofloxacin</td>
<td>Venlafaxine</td>
<td>Chlorpromazine</td>
</tr>
<tr>
<td>Ranolazine (Renexa)</td>
<td>Levofloxacin</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Moxifloxacin</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

4.8 million pts. - 22.8% (1.1 million pts.) received 1 QTc Rx
- 103,119 (2.1%) received 2+ QTc Rx's


www.torsades.org

Syncope History-

Step 3: What are the meds?

Drugs that cause hypotension
– Alpha blockers, diuretics

Drugs that cause bradycardias
– B-blockers, Ca+ channel blockers,
  – Alzheimer meds (+)

Drugs that cause prolonged QT

Syncope History-

Step 1: Risk Factors
Step 2: The episode
Step 3: What’s the meds?

Next--- The Physical Exam
But, what’s useful?

- Vitals Signs
- Cardiac Exam
- Abdominal Exam
- Neuro Exam

???

- Orthostatics?
- Carotid sinus massage?

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Carotid sinus hypersensitivity

- First described by Ibn Sina (Avicenna) 980-1037
- Asystole > 3 sec after 5-10 sec of carotid massage OR drop of 50mm Hg BPsys
- Suggested as common cause of syncope and falls in the elderly
- Pacing not effective for vasodepressive

- How common is it?
- Is it the cause of the syncopal episode?

Syncope History -
Step 1: Risk Factors
Step 2: The episode
Step 3: What's the meds?
Step 4: Physical Exam

Step 5:
- Time for Labs....Which ones???

Question #1: American College of Emergency Physicians (ACEP) guideline recommends which of the following evaluations in patients with syncope?

1. EKG (+ all other tests guided by exam)
2. EKG, CBC (+ all other tests guided by exam)
3. EKG, CBC, troponin (+ all other tests guided by exam)
4. EKG, CBC, troponin and head CT (+ all other tests guided by exam)

Syncope: the dangerous causes

- ACS
- Aortic dissection
- PE
- AAA
- Ectopic pregnancy
- GI bleed
- SAH

“Be a sniper, don’t use a shotgun!”

A. M. Mattu

Syncope: What diagnostic studies are needed (to risk stratify)?

ACEP policy: April 2007

Answer: EKG only

- Note: the yield is < 5%
- But it is low-cost, non-invasive
- And can potentially identify life-threatening conditions


All other studies are guided by H & P!!!!
Syncope: The evaluation

Step 1: Risk Factors
Step 2: The episode
Step 3: What are the meds?
Step 4: The Physical Exam
Step 5: The EKG (and “sniper” diagnostics)

What about a clinical decision rule (CDR)?

Is there a Clinical Decision Rule (CDR) that can definitively tell me to “admit vs. discharge”? (ie. identify the “high risk” patient)

SF Syncope Rule
Boston
OESIL - Italy
EGSYS - Italy
ROSE - Scotland

Clinical Decision Rules:
1. Derivation
2. Internal Validation
3. External Validation

Syncope
Definition - very useful
Pathophysiology - very useful
H & P elements - that are useful
Tests/Clinical Policies - that might be useful
Clinical Decision Rule - that might be useful
EKG’s - that are worrisome!
Conclusions and outpatient evaluation

Syncope and EKG findings
• Bradycardias, heart block
• Atrial and ventricular tachycardias
• Wolff-Parkinson-White (WPW)
• Prolonged QTc/Long QT syndrome
• Brugada Syndrome
• Hypertrophic Cardiomyopathy

Syncope and EKG findings
• Bradycardias, heart block
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• Prolonged QTc/Long QT syndrome
• Brugada Syndrome
• Hypertrophic Cardiomyopathy

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Long QT syndrome (LQTS)

- Acquired
  - Meds, toxins, electrolyte disturbances, ACS, CNS events, HIV
  - Congenital: Autosomal dominant
    - Associated with 11 genes, 600 mutations
    - Prevalence: estimates 1/2000-7000
    - Median age of sudden death = 32 years
    - Mortality = 20% in first year after syncope
      - 50% mortality within 5 years

  In series of 31 pts. with LQTS, 64% presented with syncope
  40% of the patients were not identified at first presentation!

- LQTS: Men > 460msec, women > 440msec
  - Bazett Formula
  \[ QTc = \frac{QT}{RR^{0.5}} \]
    * In leads II, V5
    * Only for HR 60-100

  In Rapid Bedside
  If QT is < 1/2 of R-R interval is OK*

Long QT syndrome

Brugada Syndrome

First described in 1992
Originally thought to be a disease of men of Southeast Asian descent
  - In Phillipines: “Bangungut”
    - “scream followed by sudden death during sleep”
  - In Japan: “Pokkuri”
    - “unexpected sudden death at night”
  - In Thailand: “Lai Tai” - “death during sleep”
  - In Laos - one death per 1000 inhabitants!!!

Now believed to be responsible for 40-60% of patients with “idiopathic V fib”
  - Second only to MVA cause of death in young adults in some countries

Mutation of SCN5A cardiac Na+ channel on Chromosome 3 ("channelopathy")
Recurrent episodes of polymorphic V tach

Brugada Syndrome: EKG

RBBB with ST elevation V1-3
  - Type I - "coved-shaped" ST
    * is diagnostic
  - Type II - "concave-shaped" ST
    * is non-diagnostic,
      But suggests dx in appropriate patient

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Brugada Syndrome: EKG

Look at V1-3
Type I

30 y/o AA female, chest tightness, palpitations, near-syncope. Previous hx of previous syncopal episodes

9 years earlier...

Hypertrophic Cardiomyopathy (HCM)

First described in mid-19th century
Previous names:
– Hypertrophic obstructive cardiomyopathy (HOCM)
– Idiopathic hypertrophic subaortic stenosis (IHSS)
Characteristics:
– Thickened myocardium
– Without ventricular dilation
– Absence of conditions that result in hypertrophy (HTN, Aortic stenosis)
Incidence: approx. 1 in 500 persons

Hypertrophic Cardiomyopathy (HCM)

Genetics: Autosomal dominant with variable penetrance
– 11 mutant genes, > 500 mutations
Variable manifestations
– Asymmetric ventricular hypertrophy
– Most pronounced in anterior ventricular septum
– Most commonly during periods of growth (ie. adolescence)
Presentation:
– May be asymptomatic
– Chest pain
– Symptoms of LVOT obstruction (SOB, DOE, syncope)
– Sudden death
Hypertrophic Cardiomyopathy (HCM)

PE: murmur noted in 30-40% only
- Increases with Valsalva

Chest x-ray: heart with normal size

EKG: most are abnormal!!!!
- 1) Large amplitude QRS complex (c/w LVH)
- 2) Deep, narrow Q waves in:
  • Inferior leads (II, III, AVF) and/or
  • Lateral leads (I, aVL, V5-6)

HCM - LVH, Q waves in I, aVL, V5-6

30 y/o male lightheaded, palpitations after running. 2 days later, running => SCD.

29 y/o male 3rd ED visit for lightheaded, palpitations with exertion.

Hypertrophic Cardiomyopathy (HCM)

Treatment: often medical (B-blockers)

Complications:
- 10-40% develop atrial fibrillation
- Increase incidence of WPW

Syncope: The evaluation

Step 1: Risk Factors
Step 2: The episode
Step 3: What are the meds?
Step 4: The Physical Exam
Step 5: The EKG (and “sniper” diagnostics)

What if the etiology is still unclear?

Question #2: In cases of unexplained syncope, the ACC guideline recommends which of the following studies be performed?

1. ECHO
2. ECHO, Exercise stress test (EST)
3. ECHO, EST, tilt-table test
4. Tilt-table only
Unexplained syncope: ACC/AHA 2006 guideline

ECHO
EST
- if malignant history

Non-invasive EKG monitoring
- Holter
- Event recorders
- Implantable loop recorders

Tilt-table testing
EP studies

The role of Echo in the evaluation of syncope: a prospective study.

155 patients with unexplained syncope
- All get ECHO

Result: # of abnormal ECHO studies=
0, Zero, Zip, nada, ect...

155 patients with unexplained syncope
- All get ECHO

Result: # of abnormal ECHO studies=
0, Zero, Zip, nada, ect...

323 ED pts admitted to “observation unit”
267 had “normal” EKG
235 underwent ECHO

Result: # of abnormal ECHO studies=
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Syncope episode***

European Society of Cardiology Guideline 2009

1. H & P
2. EKG
3. Other tests (when appropriate)

Diagnosis? -----> YES------> Treat

High short term risk?* -----> YES------> In-patient

Suspect cardiac syncope or Recurrent reflex syncope in high risk settings?

NO

Education/Reassurance

NO

Education/Reassurance

NO

Education/Reassurance

NO

Education/Reassurance
Neurally mediated syncope—Vasovagal syncope

“fainting”

- Failure of autonomic NS to maintain BP & HR
  - Starts with excessive peripheral pooling
- Why? Controversial
  - Can be “situational”
  - Examples: defecation, urination, cough

Vasovagal syncope: Management

- Physical counterpressure maneuvers
- PC Trial: 223 pts - decreased syncope episodes (2006)
- Increased fluid and salt intake
- Midodrine 5mg tid

These do NOT work...
- Beta-blockers (see POST trial, Circulation, 2006)
- Fludrocortisone (POST 2 trial, presented 10/31/11)
- SSRI’s
- Pacemaker (see Vasovagal Pacemaker Study II, JAMA, 2003)

Vaccination syncope: rates are increasing!!!

**Syncope**

- **Definition** - very useful
- **Pathophysiology** - very useful
- **H & P elements** - that are useful
- **Tests/Clinical Policies** - that might be useful
- **Clinical Decision Rule** - that might be useful
- **EKG’s** - that are worrisome!
- **Conclusions and outpatient evaluation**

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Thank you for your time and consideration!!

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Question #3: Which of the following is most commonly associated with sudden death during athletic endeavors?

1. Congenital Long QT syndrome
2. Brugada Syndrome
3. Hypertrophic cardiomyopathy
4. J point elevation

Question #4: Which of the following is not useful in recurrent neurally mediated syncope?

1. Midodrine
2. Isometric exercise during prodrome
3. Mineralocorticoids
4. Increased fluid intake

Thank you!