Syncope, POTS, and Other Neurocardiogenic Syndromes

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“Sudden Death and Syncope are the same... Except in one you wake up.”
“…cardiac syncope can be a harbinger of sudden death.”

- Framingham study
  Survival with and without syncope
- 6-month mortality rate of greater than 10%
- Cardiac syncope doubled the risk of death

Syncope - Definition

Transient Loss of Consciousness, loss of postural muscle tone due to Pan-Cerebral Hypoperfusion with complete recovery without Focal Neurologic Deficit

Excludes Seizures, TIA/ CVA, Metabolic etiologies, Psychogenic, Trauma

Part of the spectrum of "TLOC" - Transient Loss of Consciousness
Guidelines for the diagnosis and management of syncope (version 2009)

The Task Force for the Diagnosis and Management of Syncope of the European Society of Cardiology (ESC)

Developed in collaboration with, European Heart Rhythm Association (EHRA), Heart Failure Association (HFA), and Heart Rhythm Society (HRS)

Endorsed by the following societies, European Society of Emergency Medicine (EuSEM), European Federation of Internal Medicine (EFIM), European Union Geriatric Medicine Society (EUGMS), American Geriatrics Society (AGS), European Neurological Society (ENS), European Federation of Autonomic Societies (EFAS), American Autonomic Society (AAS)

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The disclosure forms of the authors and reviewers are available on the ESC website www.escardio.org/guidelines
Syncope: Etiology

1. Neurally-Mediated
   - Vasovagal
   - Carotid Sinus
   - Situational
     - Cough
     - Post-micturition
   - 24%

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

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

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

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

Unknown Cause = 34%
Approach to Syncope

“Spell”
Unexplained altered or loss of consciousness

History
Physical (Cardiac-Neurologic)
ECG

Probable Clinical Diagnosis

1 Cardiac
Arrhythmias Obstructive

Cardiac Risk Factors?
- LV dysfunction
- Abnormal ECG
- Older Patients
- ASHD

Cardiac Cath
Echocardiogram

EEG
CT scan
Other tests as appropriate

2 Neuro-Regulatory
Head-up tilt
Hemodynamics
Blood Volume
Autonomic Testing
Carotid Sinus Massage

3 Neurologic Consultation

Presumptive Diagnosis with Clinical Correlation

Definitive Diagnosis
Appropriate Therapy

Extended ECG
Monitoring
Return to 1,2 or 3

Return to 1 or 2
Center for Syncope – Initial Assessment

- History/ Physical
- Orthostatics
- EKG
- Carotid sinus massage
- Echo +/- Stress
- Holter/ Ambulatory monitoring
- Tilt if suggestive of VVS, POTS, Dysautonomia
- EPS/ Loop recorder implant, if decreased LV, conduction abnormalities
Syncope - Clues from the History

- Family History of SCD = LQTS, Brugadas, HCM, ARVD/C
- Tongue biting, incontinence = Seizure
- Motion sickness, migraines, childhood fainting, athlete, hypervagotonia, early repol on EKG, phobic/pain fainting = Vasovagal
- Head turning = carotid sinus syndrome
- Many episodes, vagal symptoms, prolonged warning, = Vasovagal “Status Vasovagalis”
- No prodrome, no symptoms post, Male sex, first episode over age 55 = AV block or VT
Syncope – When to Admit for expedited evaluation?

- **S. Francisco rule** – CHF, Crit<30%, abnormal ECG, SOB, Systolic<90 “CHESS”
- **OESIL** - Abn ECG, CV disease, no prodrome, age >65
- **EGSYS, ROSE** – Palps, syncope supine or during exercise, High BNP, Low O2, Q waves, occult blood
- **Structural heart disease** – CAD, DCM, CHF, MIs
- **Family history of SCD**, HOCM, Brugadas Syndrome, DCM
- **Conduction Abnormalities** - Trifascicular Block, LBBB
- **Features** – sudden LOC without prodrome, MVAs, possible SZs, Fractures, Head or bodily injuries, Chest pain or exercise induced
- **EASYAS Trial 1 and 2** – implantable loop recorders in ED
Syncope – EKG monitoring

- EKG/ SAEKG
- Holters/ Event recorders - continuous loop recorder
- Implantable
  - Reveal and Confirm - insertable loop recorder
  - Pacemakers/ ICDs with wireless transmission
- Ambulatory Wireless long term monitoring for diagnosis/prevention – MCOT (Mobile Cardiac Outpt Telemetry) continuous loop recorder using cellular networks, bluetooth e.g. Cardionet, Lifewatch
- Future devices will incorporate BP, O2 etc
Wireless Ambulatory ECG Monitors

- CardioNet, Lifewatch, others
- Automatic/Patient activated
- External 3 Lead/Portable Monitor Wireless Connection to Service
- GPS Capable, Altimeter
- Aimed at Low Risk Patient - Not in 911 business
  - Atrial fibrillation
  - SVT
  - SSS
  - Syncope of Undetermined Origin
  - Cryptogenic Stroke
  - Refractory Seizures
Ambulatory Monitoring - Summary

• Choose monitor likely to have highest yield.
• Pts with daily events, palpitations, syncope/near syncope, holters frequently are sufficient.
• Weekly or monthly events use wireless monitors
• Pts with rare events, Implantable Loop recorders.
• Elderly, No prodrome or no warning, prolonged spells, solitary, Use wireless or implanted loop
• Valuable if symptoms but NSR, Sinus Tachycardia. Tells you what its not.
• High Risk Pts, Structural Heart disease/Conduction disease or MVAs severe Trauma, should receive expedited, in house evaluation, EPS etc.
• Take advantage of PPM and ICD monitoring capabilities
Wireless Monitoring - Conclusions

- Very effective at demonstrating suspected, covert arrhythmias.
- Good compliance, all pt has to do is put it on.
- Limitations – Very expensive range from $100-150 per day
- Not very “green”, Pts can generate 100’s of pages of reports.
- No Hemodynamic data, Symptoms with Sinus Rhythm, Need BP monitoring
Reveal/Reveal Plus/ DX /XT - Medtronic Insertable Loop Recorder - ILR

- Records EKG – not electrogram
- Minimal Incision – Low Risk
- Battery Life 14 – 22 months
- Autoactivation and Patient Activation – up to 40 minutes pre, and 2.0 minutes post event
- Appropriate Patient Selection – early utilization (RAST, ISSUE)
  - SUO in Normal Hearts
  - Fleeting Suspected SVT’s
  - Drug Refractory Seizures
- Inappropriate for High Risk Patients
  - Severe LV/Post MI
  - VT Suspected
  - Structural Heart Disease
Sixty Pts recurrent syncope, normal LV function.

Randomized to Tilt, EPS (Conventional) versus Loop recorder.

Crossover at one year if no diagnosis.

47% diagnosed with ILR, $2731 per Pt, $5852 per diagnosis

20% conventional approach, $1683 per Pt, $8414 per diagnosis

Higher yield more cost effective with Loop recorder
Figure 2. Cost and diagnostic yield of the two treatment strategies. The results of the testing strategy are shown for all patients in the trial, including those who crossed over. The overall diagnostic yield of the two strategies was comparable, but the cost per diagnosis of the strategy of primary monitoring was significantly reduced by $2,016 (p = 0.002).
Syncope due to Idiopathic Paroxysmal AV Block

- 18 patient’s with recurrent syncope
- Normal heart
- Implantable loop recorders in most
- Spontaneous third degree/complete heart block without evidence of vagal stimulation
- Normal conduction system/Normal EKGs, normal PR and QRS/normal His-Purkinje
- Etiology? Intrinsic AV node disease or autonomic reflex

Brignole JACC 2011
A 57 yo male with dilated cardiomyopathy presents to the ED with a facial laceration. He reports he was urinating at night and suddenly lost consciousness, falling and sustaining the injury. He felt fine before and after the event. He has an EF of 25% secondary to probable viral myocarditis. His EKG reveals IVCD and occasional multifocal PVCs. What is the next step?

A. Discharge from the ED with a 48 hour holter monitor  
B. Admit, perform EPS and if negative implant a Reveal  
C. Schedule outpatient Tilt Test  
D. Admit, implant ICD
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Non Ischemic Dilated Cardiomyopathy and Syncope

• Portends a high mortality
  —(45% at one year) regardless of cause

• EPS for SMVT - often unrevealing/non predictive

• No prospective studies of “Empiric ICD”

• Now Moot by Definite, SCD-Heft
Syncope

- Indications for EPS
  - Prior MI, LV Dysfunction/Cardiomyopathy
  - Wide QRS – LBBB/RBBB, ?Brugadas
  - Systemic Illness
    - Sarcoid
    - Lymes
  - Prolonged PR, Mobitz 1
  - “Complex” Ventricular Ectopy – NSVT
  - SVT, Delta Wave or sustained palpitations
  - Family HX of SCD
  - Trauma, MVA, Recurrent, High Risk occupations
  - “Final Court of Appeals” Although EPS likely low yield
A Case of Recurrent Unexplained Syncope

55 y/o female presents to office for consult reporting multiple abrupt syncopal events since childhood, more frequent lately. She is generally healthy but takes Beta blocker for long standing Hypertension. Has had recent negative stress echo and holter. She has normal EKG. Episode have resulted in minor injuries and occasionally accompanied by vagal symptoms.

What is the first/next step?

A. Schedule pacemaker
B. EP study
C. Tilt test/event recorder
D. Schedule loop recorder implant
E. EEG/Head CT
A Case of Recurrent Unexplained Syncope

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Tilt test was performed and provokes a vasovagal episode at 18 minutes of 70 degree tilt with moderate hypotension and mild sinus slowing. Patient states her symptoms on the tilt were similar although milder than clinical. You advise the perfunctory recommendations and schedule follow up.

She returns 3 weeks later reporting several more episodes. What are possible next treatment/diagnostic options.

A. Permanent Pacemaker  
B. Midodrine  
C. Beta blockers  
D. Levsin, implant loop recorder  
E. Florinef
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A Case of Recurrent Unexplained Syncope

The medication you prescribed may be helping somewhat but he is still passing out. You offer a reveal which captures an event.
A Case of Recurrent Unexplained Syncope

You discuss the options with the patient and based on the loop recorder you make the following recommendation.

A. SSRI
B. Permanent pacemaker
C. Cautious trial of midodrine and florinef
D. Begin a program of tilt table training
E. Disopyramide
A Case of Recurrent Unexplained Syncope

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A Case of Recurrent Unexplained Syncope

She receives a DDD medtronic pacemaker with rate drop algorithm. Her episodes become less but she is still having occasional episodes of near syncope and syncope. She calls you several weeks later c/o chest pain. You order a CXR. What does it show?

A. Normal
B. Pneumothorax
C. Lead dislodgement
D. Possible perforation
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Man is the only animal that blushes. Or needs too.

Mark Twain

Evolution of Vasovagal Syncope

THE ASCENT (AND DESCENT) OF MAN
Vasovagal Syncope

- Comprises significant proportion of unexplained syncope
- 60% of population are estimated to have at least one episode
- ANS perturbations - Sudden hypotension/bradycardia resulting in loss of consciousness
- Presumed trigger is augmented inotropic, chronotropic cardiac state
- Bezold Jarisch Reflex - sudden withdrawal of sympathetic/heightened or unopposed vagal
- Episodes may cluster, the disappear for long intervals
Vasovagal Syncope: Features

- Crumple to ground - injuries are rare
- May recur
- Slow recovery- “Vagal” for hours
- Myoclonic jerking “anoxic seizure, Convulsive Syncope”

“Fight or Flight”

- Diming of vision, “grey out”, Diaphoresis, nausea, pallor, warmth
- Hyperventilation - “shortness of breath”
- Yawning, weakness
- Palpitations, “chest pain”
Vasovagal Syncope

“By Any Other Name”

- Vasodepressor syncope
- Neurally mediated / Reflex syncope/hypotension “NMH”
- Neuro-cardiogenic syncope
- Empty heart syndrome
- Ventricular syncope

A Lecture
ON
VASOVAGAL SYNCOPE AND THE CAROTID SINUS MECHANISM
WITH COMMENTS ON GOWER’S AND NOTHNAGEL’S SYNDROME
BY
Sir THOMAS LEWIS, M.D., F.R.C.P., F.R.S.
(From the Department of Clinical Research, University College Hospital Medical School)
The British Medical Journal 873 May 14, 1932
Vasovagal Syncope

- Benign/Situational
  - Emotional faint
  - Fear, pain - “The Paleolithic – Threat Hypothesis” Darwinian Fitness
  - Dentist, church, Restaurant, phlebotomy

- Malignant
  - No recognized stimulus
  - Little or no prodrome
  - Prolonged asystole
  - Injuries
  - Social impact – Loss of Occupation, Driving
Malignant Vasovagal Syncope
Situations which Provoke Vasovagal Syncope

• Fear, anxiety, “flight or fight”, pain, venipuncture

• Pregnancy, standing “at attention”

• Hypovolemia, anemia, hemorrhage

• Head-up tilt, lower body negative pressure

• “First dose phenomena”, nitrates

• Beta-blocker withdrawal

• Prolonged bed rest, prolonged head down tilt, microgravity
Neurally Mediated Syncope

- Vasovagal syncope
- Carotid sinus syncope
- Tussive syncope
- Glossopharyngeal neuralgia / deglutition syncope
- Pallid breath holding spells
- Aortic stenosis
- Hypertrophic obstruction cardiomyopathy
- Pacemaker syncope
- Syncope secondary to pulmonary hypertension
- Micturition syncope
- Mess trick – Fainting Lark
- Diving reflex

- Syncope during Atrial fibrillation, VT and SVT may have Neurally mediated contribution and component
24-year-old female, nursing college student, presents with dizziness, lightheadedness, near syncope, palpitations, which began recently. Her echo and EKG are normal. Her standing systolic blood pressure in the 90s with a heart rate of 120BPM. She has headaches, has been very fatigued, muscle aches and pains, and is having a hard time with her school work. What is the underlying likely diagnosis?

A. Inappropriate sinus tachycardia  
B. Orthostatic hypotension  
C. Supraventricular tachycardia  
D. Dysautonomia such as POTS  
E. Anxiety depression
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You suspect she is experiencing a form of dysautonomia. You recommended high salt diet, compression stockings, augmented fluid and electrolyte intake, and discussed pharmacologic and nonpharmacologic approaches. What test do you order to confirm the diagnosis?

A. Stress Echo  
B. Tilt Test  
C. Event recorder  
D. Psych consult  
E. QSART
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E. QSART
During Tilt test her HR reaches 140 bpm with severe symptoms. The non-pharmacologic recommendations are not helping. What medication might you treat with initially?

A. Midodrine  
B. Beta blockers  
C. Florinef  
D. SSRI  
E. Any of the above
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Postural Orthostatic Tachycardia Syndrome

“POTS” – The new Pandemic

- Orthostatic intolerance, Hyper Beta state, Idiopathic Hypovolemia
- Predominantly young, female
- Exaggerated Heart Rate +/- Orthostasis
  - Response to Tilt/Exercise
- Partial Dysautonomia, GI, GU, Cognitive Dysfunction
- Chronic Fatigue/ Fibromyalgia/ Lymes Disease/ Hyper-mobility syndrome - Gulf War Syndrome/illness
- Vasovagal Susceptibility
- Heterogenous Pathophysiology
  - Hypovolemia
  - Venous Pooling - ?Defect in vasoreactivity
  - Norepinephrine Transporter Deficiency - Robertson NEJM 2000:342
POTS
“Grinch Syndrome”

- POTS pts had decreased LV mass by MRI, lower blood volumes, decreased cardiac outputs
- Intense 3 month exercise program – recumbent bike, rowing, swimming
- Upright exercise later added
- LV mass, HR, BV improved in 50%, QOL improved in all

Levine JACC 2010
Cardiac Origins of the Postural Orthostatic Tachycardia Syndrome

Qi Fu, MD, PhD,*† Tiffany B. VanGundy, MS,* M. Melyn Galbreath, PhD,*† Shigeki Shibata, MD, PhD,*† Manish Jain, MD,*† Jeffrey L. Hastings, MD,*† Paul S. Bhella, MD,*† Benjamin D. Levine, MD*†

Dallas, Texas

Objectives

The purpose of this study was to test the hypothesis that a small heart coupled with reduced blood volume contributes to the postural orthostatic tachycardia syndrome (POTS) and that exercise training improves this syndrome.

Background

Patients with POTS have marked increases in heart rate during orthostasis. However, the underlying mechanisms are unknown and the effective therapy is uncertain.

Methods

Twenty-seven POTS patients underwent autonomic function tests, cardiac magnetic resonance imaging, and blood volume measurements. Twenty-five of them participated in a 3-month specially designed exercise training program with 19 completing the program; these patients were re-evaluated after training. Results were compared with those of 16 healthy controls.

Results

Upright heart rate and total peripheral resistance were greater, whereas stroke volume and cardiac output were smaller in patients than in controls. Baroreflex function was similar between groups. Left ventricular mass (median [25th, 75th percentiles], 1.26 g/kg [1.12, 1.37 g/kg] vs. 1.45 g/kg [1.34, 1.57 g/kg]; p < 0.01) and blood volume (60 ml/kg [54, 64 ml/kg] vs. 71 ml/kg [65, 78 ml/kg]; p < 0.01) were smaller in patients than in controls. Exercise training increased left ventricular mass and blood volume by approximately 12% and approximately 7% and decreased upright heart rate by 9 beats/min [1, 17 beats/min]. Ten of 19 patients no longer met POTS criteria after training, whereas patient quality of life assessed by the 36-item Short-Form Health Survey was improved in all patients after training.

Conclusions

Autonomic function was intact in POTS patients. The marked tachycardia during orthostasis was attributable to a small heart coupled with reduced blood volume. Exercise training improved or even cured this syndrome in most patients. It seems reasonable to offer POTS a new name based on its underlying pathophysiology, the “Grinch syndrome,” because in this famous children’s book by Dr. Seuss, the main character had a heart that was “two sizes too small.” (J Am Coll Cardiol 2010;55:2858–68) © 2010 by the American College of Cardiology Foundation.
Treatments for “POTS”

- High sodium diet (5-7 g)
- 2 L electrolytes/sports drinks daily
- Compression stockings – knee high 20-30 mmHg/”Spanx”
- Sleep with the head of bed elevated 4” – 6”
- Cardiovascular reconditioning, swimming, recumbent bike, rowing machine
- Beta blockers, usually low dose, Florinef, SSRIs, SNRIs, midodrine
- Mestinon if small fiber neuropathy
Carotid Sinus Syncope

- Highest Incidence in Elderly
- Hypersensitive carotid sinus reflex with transient asystole or AV block and hypotension
- Episodes occur with head turning, shaving, adjusting tie, looking up, etc
- Treatment with PPM
- Testing with carotid sinus massage
- Amnesia of Event - “Fits, Falls, Faints and Fractures Clinic”
Deglutition/ Swallow Syncope
Cough/Tussive Syncope

- Cardiopulmonary Baroreflex with vasodepression, occasional pauses, bradycardia, AV block, ? Elevation of CSF pressure
- Search for ENT/pulmonary cause
- Treatment: cough suppression, aerosol Lidocaine
A 21 yo female college student presents to her local ED with complaints of fainting twice earlier that day. She reports the first episode occurred while in the shower. It was preceded by nausea, diaphoresis with sudden loss of consciousness. After she awoke on the floor, she felt very nauseated, diaphoretic and vomited. She tried to stand but fainted again. She is otherwise healthy but has had the “flu” for three days. You are asked to consult. Her PMH, EKG, physical exam and lab are normal. She fainted once while donating blood for a blood drive. What tests do you order?

A. Tilt Test
B. Holter Monitoring
C. Stress echo
D. None of the above
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A. Tilt Test  
B. Holter Monitoring  
C. Stress echo  
D. None of the above
Tilt Testing according to House
Tilt Table Test

- Traditional research tool for postural effects on BP and arrhythmias
- Control subjects with syncope were observed to faint
- Maximizes venous pooling
- Provokes vasovagal syncope in susceptible patients

The Simulated Faint, Pietro Longhi 1745
Head Up Tilt: Indications

• Syncope of unknown origin
  – Suspected vasovagal

• Syncope with observed SA or AV dysfunction
  – Extrinsic sick sinus syndrome

• Seizures versus Syncope

• Pseudoseizure/Psychogenic Syncope
  – Arterial line, Transcranial Doppler, EEG

• PAF with Syncope
Tilt Table Testing Indications

Emerging Indications

• POTS – Postural Orthostatic Tachycardia Syndrome
• Orthostatic Hypotension, Autonomic Insufficiency
• Idiopathic Vertigo, Dizziness, Lightheadness
• TIA’s
• Chronic Fatigue Syndrome, Fibromyalgia, Gulf war syndrome
• Sudden Infant Death Syndrome (SIDS), Pallid Breath Holding Spells
• Unexplained Falls

Not Warranted

Single episode in “classic” patient
Alternative specific cause demonstrated
Controversies in Tilt Testing

• Reproducibility - day to day variability

• Sensitivity/Specificity - too many protocols

• Guidance of Therapy - serial tilt testing not predictive

• Necessity? Superfluous? Syncope with normal heart almost always vasovagal anyway, so why test?

• Who should see Vasovagal pts? EPS? Cardiology? Neurology?

• Pharmacologic  Adjuvant testing

• Isoproterenol – Kapoor “A Non Specific Test” contraindicated in ischemic disease

• Adenosine

• Nitroglycerine – SL spray or tablet-”The Italian Protocol”

• Clomipramine –Theodorakis  JACC  2000

• Edrophonium - anticholinesterase

• Alcohol – Post Alcohol Syncope

• Methodology
Vasovagal Syncope

**Tenets of Treatment**

- Reassurance / Recognition / Avoidance
- Increase salt/ electrolyte fluids - 5-7 grams per day / florinef
- Physical Countermeasure Maneuvers (PCM) Supine position, Leg Crossing, Muscle tensing   Circ 2002 Coughing

- Jobst / physical therapy / Avoid deconditioning
- Serial tilt testing not predictive
- Tilt Table Training - Ector
Vasovagal Syncope

**Medical Therapy**

- Fludrocortisone - Mineralocorticoid
- β-blockers (Class 3)
- Disopyramide
- Serotonin reuptake inhibitors - Sertraline, Fluoxetine
- Anticholinergics - Levsin, Transderm scopolamine, Robinul
- NET - Norepi reuptake transport inhibitor - Meridia
- Theophylline - adenosine receptor blockade
- Amphetamines, Ritalin
- α Agonists - IV phenylephrine, oral pseudoephedrine, Midodrine (Class 2B)
- Calcium channel blockers
- Epogen, DDAVP, Yohimbine, Mestinon - Grubb

**Fainting Cures**

- Warm patients - avoid hot foods - get plenty rest
- Cool patients - eat spicy foods - walk briskly

**It’s going to work.**

Sucrosa placebo

*It’s a pill.*

*AstraZeneca*
Recurrent Vasovagal Syncope - Pharmacologic Treatment

- Florinef if low Blood Volume
- Levsin for young, hypervagotonic pts
- Beta blockers if tilt suggests hyperadrenergic state, hyperkinetic circulation
- Midodrine if low grade orthostasis, CFS, FM
- SSRI esp if additional DXs
Spontaneous Resolution in Untreated Patients

Recurrence - Free Probability

n = 54
Age = 48 ± 12 yrs
Episodes = 10.2 ± 7
Duration = 6.4 ± 85 yrs

Natale, AJC 1995
VPS - Time to Syncope Recurrence

Connolly, JACC, 1999

Cumulative Risk (%)

Time in Months

Number C 27
At Risk P 27

Pacemaker

No Pacemaker

2P = 0.000022

Connolly, JACC, 1999
Pacemakers for Vasovagal Syncope
“Closed Loop Stimulation” CLS

INVASY Trial

- Biotronik PPMs with Closed loop stimulation (CLS)
- CLS measures RV Impedence and correlates to dP/dT
  Senses increasing RV contractility in impending VVS
- 50 Pts with recurrent VVS
- PPM – DDI(40bpm) versus DDD-CLS
- No recurrence in the DDD-CLS group, 7/9 recurrence in PPM off group

Occhetta Europace 2004
Pacemakers for Vasovagal Syncope

- Cardioinhibitory response confirmed by loop recorder
- "Highly" symptomatic, multiple episodes with injuries
- Drug failures
- Elderly – overlap with SSS, CSS
- Rate drop / hysteresis /CLS
- High risk occupations - Pilot / high steel / commercial driver
# Unexplained Syncope and Driving

## Recommendations concerning driving in patients with syncope

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th><strong>Group 1 (private drivers)</strong></th>
<th><strong>Group 2 (professional drivers)</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac arrhythmias</td>
<td>After successful treatment is established</td>
<td>After successful treatment is established</td>
</tr>
<tr>
<td>Cardiac arrhythmia, medical treatment</td>
<td>After 1 week</td>
<td>After appropriate function is established</td>
</tr>
<tr>
<td>Pacemaker implant</td>
<td>After successful treatment is established</td>
<td>After long-term success is confirmed</td>
</tr>
<tr>
<td>Successful catheter ablation</td>
<td>In general low risk, restriction according current recommendations</td>
<td>Permanent restriction</td>
</tr>
<tr>
<td>ICD implant</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reflex syncope</td>
<td>No restrictions</td>
<td>No restriction unless it occurred during high risk activity*</td>
</tr>
<tr>
<td>Single/mild</td>
<td>After symptoms are controlled</td>
<td>Permanent restriction unless effective treatment has been established</td>
</tr>
<tr>
<td>Recurrent and severe*</td>
<td>No restrictions</td>
<td></td>
</tr>
<tr>
<td>Unexplained syncope</td>
<td>No restrictions unless absence of prodrome, occurrence during driving, or presence of severe structural heart disease</td>
<td>After diagnosis and appropriate therapy is established</td>
</tr>
</tbody>
</table>
Ictal Asystole

- Sinus bradycardia, pauses very common during true seizures
- Marked bradycardia or prolonged asystole has been observed during seizures
- Suspected when SZ result in sudden falls, injuries or trauma or change in severity of SZs
- Possible autonomic etiology Sz activity in higher centers propagates to Brainstem
- May exacerbate seizure severity - injuries and death  Sudden Unexplained Death in Epilepsy (SUDEP)
- Video EEG/ECG monitoring, Implantable Loop recorders
- Pacemakers advised but no controlled trials
Syncope Center Testing

- Heart Rate Variability (HRV) – Vagal or sympathetic predominance?
- Blood Volume - RISA
- Radionuclide Hemodynamic Studies
  - Mean transit time
  - Venous pooling
  - Total peripheral resistance
- Autonomic Reflexes – Valsalva, Baroreflex testing
- QSART - Quantitative Sudomotor Axon Reflex Test
- Paraneoplastic Autoantibody Panel
- Skin, Lip, Fat pad Biopsy to rule out small fiber neuropathy, amyloidosis, etc
- BioZ – hemodynamic monitor
- Ambulatory BP monitor
- Thermoregulatory Sweat Testing (TST)
Autonomic Insufficiency/Failure

• Primary Causes-
  Multi- System Atrophy (MSA) formerly Shy Drager - Marked OH with extrapyramidal signs, Parkinsons disease

  Pure or Primary Autonomic Failure (PAF) formerly Bradbury Eggleston – Marked OH

• Secondary Causes – Meds, Chemo, CVAs, Amyloidosis, renal failure, etc
Asystole and Convulsive Syncope During Head-up Tilt

A. Onset of Vasovagal Syncope

B. Prolonged Asystole

C. After IV Atropine
The PolyVagal Theory: Phylogenetic Contributions to Social Behavior

• Theory suggests there are multiple Vagal levels of activity and function
• Limbic (Reptilian) Vagal activity triggers Passive avoidance, death feigning, Vasovagal Syncope
• Cerebral (Mammalian) Vagus facilitates active avoidance, facial expression, excitation, “Body Language”
• Vasovagal reflex was preserved by evolution to cause LOC during overwhelming physical injury and allow painless transition to Death

Porges, Physio and Behavior, 2003
Tree Snakes Don’t Faint

"Ontogeny Recapitulates Phylogeny"
Heart rate and blood pressure changes in patient 37 undergoing Head-up tilt who developed a typical vasovagal response at 6 minutes of 60° tilt.
Vasovagal Syncope – Arterialization of Venous Blood

“I bled a lady but she fainted and while she continued in the fit, the color of the blood that came from the vein was a fine scarlet.”

John Hunter, 1793