Vasovagal Syncope

(Neurocardiogenic/Reflex Syncope)

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VASOVAGAL SYNCOPE: TALK OUTLINE

- Epidemiology and Clinical Impact (2 slides)
- Clinical presentation (1 slide)
- Pathophysiology (5 slides)
- Head-Up Tilt Table Testing (4 slides)
- Treatment of VVS (2 slides)
- Teleology of VVS (3 slides)
- Take-Home messages (1 slide)
INCIDENCE AND IMPACT (OF SYNCPE IN GENERAL)

- 3% of all E.R. visits; 6% of all Hospital admissions
- About 1/3 of ALL human beings will experience VVS at least once in their lifetime
- 1 million people seek Rx for syncope every year in US
- >$1 billion spent on evaluation/Rx of syncope every year
- Over all, incidence of syncope ↑ with age; however, vasovagal syncope ↓ with age (but never goes away)
- Retrograde amnesia common → makes diagnosis challenging, since ≈ 70% of diagnostic power is in the history!
COMMON CAUSES OF SYNCOPE BY AGE

Vasovagal Syncope (including situational)

- Hypertrophic Cardiomyopathy
- Carotid Hypersensitivity
- Aortic Stenosis
- Atrioventricular Block Sinus Node Dysfunction
- Inherited “Channelopathies”
- MMVT
- Drug/Alcohol related

Syncope of Unknown Origin

Slide Adapted from Dr. Suneet Mital
Clinical Presentation of VVS

• Typically, young pts; syncope often started during childhood/teenage yrs
• More often female; athletic, low BMI, and avoid salt
• Describe fainting in warm environments, while standing (rarely while seated, never while lying down), after exertion and excessive perspiration
• Typical prodrome of nausea, diaphoresis, pallor, feeling clammy, blurred vision, impaired hearing, paresthesia, yawning, lightheadedness
• Progresses to syncope, followed by spontaneous awakening; may be weak but not confused upon awakening
Pathophysiology of VVS

- Need 3.5 ml O$_2$/100 gm of brain tissue/minute to maintain consciousness

- Normal cerebral blood flow is 50-60 ml/100 gm of brain tissue/minute; human brain demands 20% of cardiac output

- It takes 7-10 sec of cerebral hypoperfusion to induce syncope

- This “Safety Factor” is reduced in elderly, and in patients with hypertension, hypovolemia, diabetes, peripheral vascular disease, CHF, and in patients on vasodilators
Pathophysiology of VVS (continued)

• Gravitational stress is the commonest trigger; critical balance between circulating blood volume and vascular capacitance

• Distribution of blood volume:
  • 5% → in the capillaries
  • 8% → in the heart
  • 12% → in the pulmonary vasculature
  • 15% → in the arterial system
  • 60% → in the venous system

• A sudden ↑ in venous capacitance of only 10 - 15% (i.e., about 300 - 450 cc) can transiently ↓ effective cardiac output to ZERO! (“preload dependence”)

Normal Reflexes ➔ Prevent Syncope

Gravitational pooling (up to 500 cc) in legs upon standing

Decreased venous return and transiently, cardiac output (up to 25%)

Sympathetic activation and vagal withdrawal

Heart rate increase (~10 to 15 bpm) and peripheral vasoconstriction

Mean BP increases (~10 mm Hg) and cerebral perfusion is maintained
Abnormal Reflexes → Leading to Vasovagal Syncope

Gravitational pooling in legs upon standing

↓

Decreased venous return and cardiac output

↓

EXAGGERATED SYMPATHETIC ACTIVATION

↓

Triggers "C-FIBERS" in base of heart

↓

Paradoxical Sympathetic withdrawal and Vagal activation

↓

Hypotension, Bradycardia and Syncope
The Brain Self-Preservation Theory

• Monitoring of cerebral blood flow in pts with syncope during HUT shows that cerebral blood flow ↓ several minutes before syncope

• When brain senses a ↓ in blood supply → initiates self-preservation reflex

• After a period of heightened alertness/fear (when sympathetics are fully ↑↑) → the brain activates the para-sympathetics (and inactivates the sympathetics) → creates bradycardia and vasodilatation → syncope and “horizontality” → restoration of cerebral blood flow

• If true, VVS is a logical response to extreme ↓ in cerebral blood flow that is unmanageable by all other means!
Head-Up Tilt Table Study

- May be nonspecific (high false positives)
- May be insensitive (high false negatives)
- Most convincing when symptomatic hypotension occurs, and clinical symptoms are reproduced, during HUT

Asystole during HUT

General Considerations

• The **length of asystole** during HUT is:
  - *not* reflective of severity of problem
  - *not* indicative of worse prognosis
  - *not* associated with risk of sudden death
  - *not* necessarily indicative of need for PPM
  - *not* reproducible during repeat HUT
HUT in the Elderly

Older patients show:

• Less susceptibility to vasovagal syncope
• Fewer false positives $\Rightarrow$ a + HUT in the elderly is more likely to be a TRUE POSITIVE
• Greater $\downarrow$ in SBP; greater $\uparrow$ in DBP; no difference in Mean BP
• Lesser $\uparrow$ in HR
• More vasodepressor/dysautonomic, less cardio-inhibitory response

Pin Tan Met al. Vasovagal Syncope in the Older Patient. JACC 2008;51;599-606
Treatment for VVS

• **Non-Pharmacologic – most important!**
  - Salt, fluids, assume supine position with prodrome
  - Orthostatic self-training
  - Leg crossing with muscle tensing
  - Isometric arm contraction

• Medical
  - Midodrine
  - Fludrocortisone
  - SSRIs

• ? Pacing – rarely, in selected pts

• Rx can allow return to exercise in majority\(^1,2\)

Higher centers
Emotional Conditioning Counseling

Bradycardia
?PM (rate and contractility)

Nucleus Tractus Solitarius
SSRI
Thoephylline
Tilt training

Increased Contractility
Beta-blockers?
Disopyramide

Peripheral Vasculature
Non-pharmacologic Isometrics
Fluids/Salt
Florinef
Midodrine

Targets for Rx of VVS
Why are Humans, and No Other Species, Prone to VVS?

• Classical (emotional and orthostatic) VVS may not be a disease, but rather a non-pathological trait

1. **Human Conflict theory:** During our evolution, inter-group attacks and killing, loss of consciousness triggered by fear-circuitry activation may have **conferred a survival advantage on non-combatants** (particularly children and women) when threats were inescapable – “sham death”

2. **Clotting hypothesis:** During traumatic bleeding, the lowering of the BP from VVS would **reduce blood loss** until stable blood clots formed

Heart to Brain Height

• The human brain is about 40 cm above the heart
• Many mammals have greater brain elevations
• A giraffe has a very muscular heart, generating an aortic pressure of 260 mmHg, which allows a cerebral perfusion pressure of ~90 mmHg
• Giraffe: Upon lowering its head to drink, cerebral pressure should increase by ~370 mmHg! However, leg splaying, slow lowering, and large venous cerebral networks blunt this rise.
• Other species are also vertical, but never experience emotionally triggered VVS... *Why?*

Unique to Human Physiology... Three factors

• 20% of cardiac output is destined for the human brain
  • 6.7% in Chimpanzees, 1% in giraffes
  • Also, human brain consumes 25% of whole-body glucose consumption; needs constant blood supply, since neurons cannot store glucose

• Human legs are larger (related to overall body size), with large venous capacitance, as compared to other vertical species

• But… our leg “muscle pump” is less active, since we can “lock” our knees; other species need constant leg muscle contraction for weight bearing

IMPLICATION: Our large brains and bipedalism have inflicted a combination of a large % of CO that must be pumped upwards, but with very massive yet passive legs → predisposition to VVS!

Take-Home Messages

• VVS is very common – most common cause of fainting in humans
• Diagnosis depends on HISTORY and ECG; does not need additional testing when typical
• HUT testing is sometimes helpful, but HUT has high false positive and false negative rates
• 1st step in treatment is non-pharmacologic – supine position, fluids/salt replenishment, isometric muscle contraction; consider midodrine or fludrocortisone only if these measures are ineffective
• VVS is an evolutionary trait, not a disease! May offer a survival advantage in scenarios of conflict or bleeding
Female, Pale and Diaphoretic!

Limp!

Somebody, anybody, please lay the poor child flat!!

A 1744 oil painting by Pietro Longhi titled Fainting