8th Annual Emirates Cardiac Society Conference

ACC Middle East Conference 2017

DUBAI

OCTOBER 19 – 21, 2017

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Vasovagal Syncope

(Neurocardiogenic/Reflex Syncope)

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Jefferson

Disclosures

- Speaker for St. Jude Medical, Zoll
- Fellowship support from Medtronic and Boston Scientific

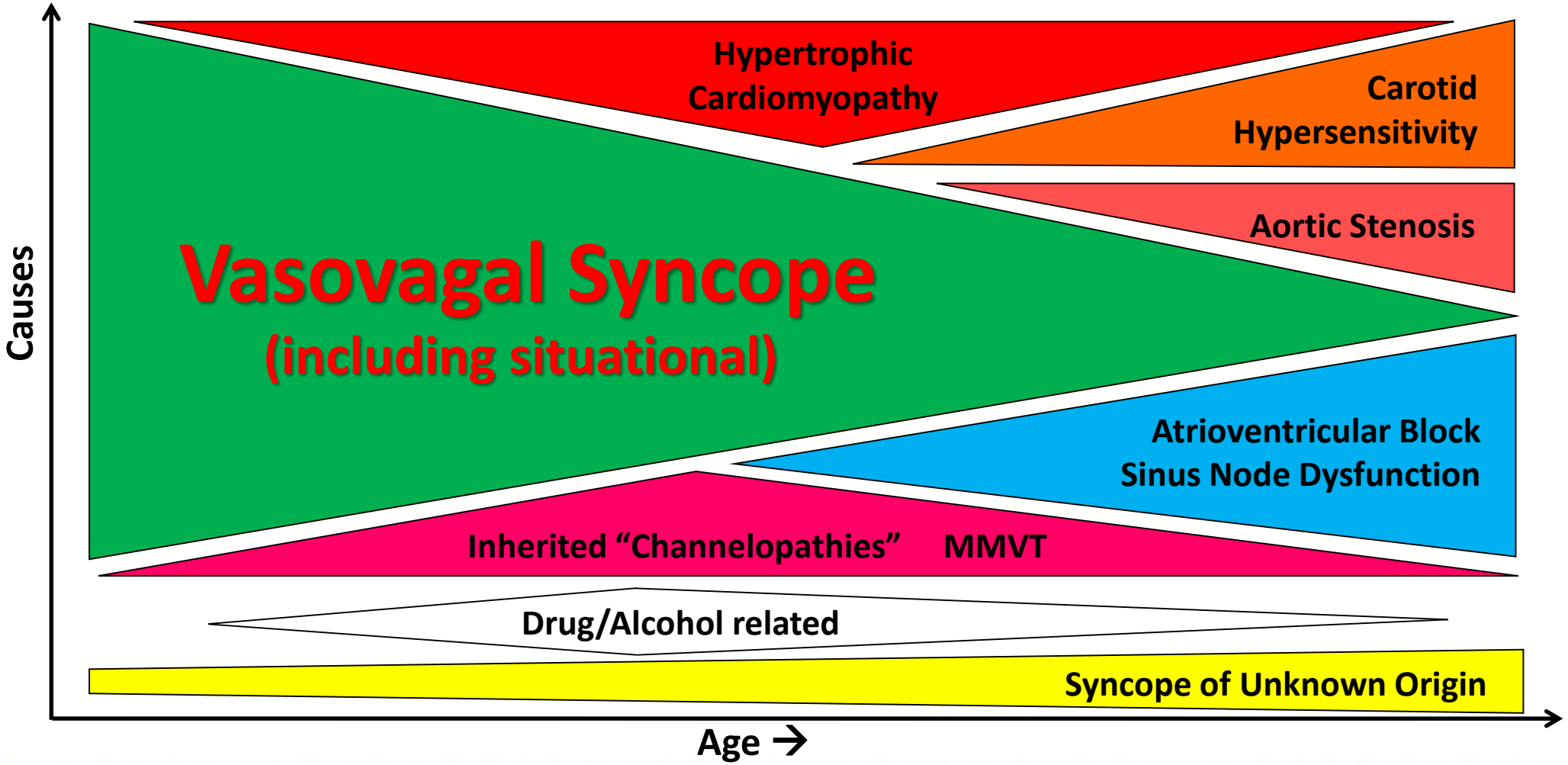
VASOVAGAL SYNCOPE: TALK OUTLINE

- 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 SYNCOPE 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



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 out put 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

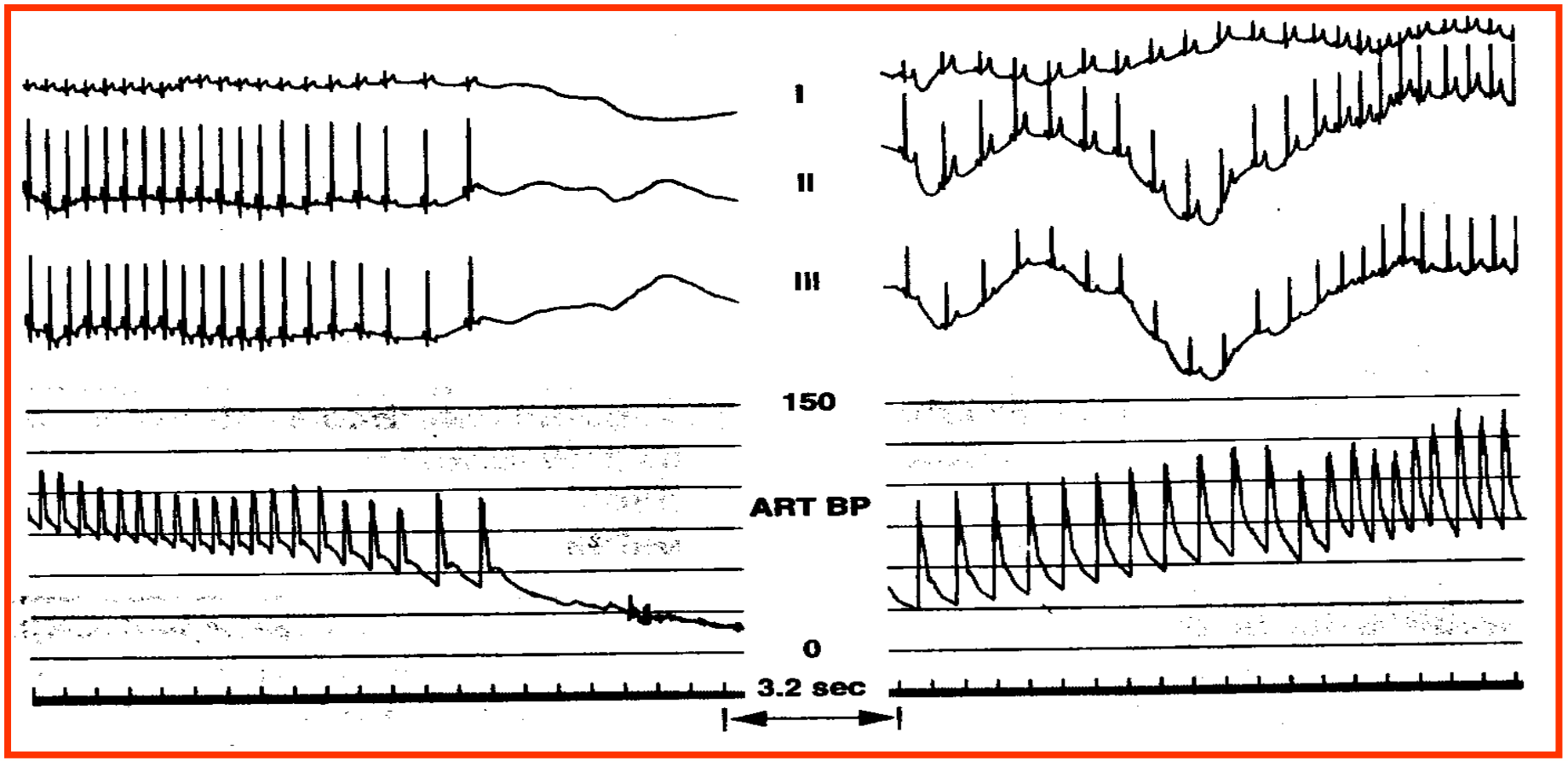
The Brain Self-Preservation Theory

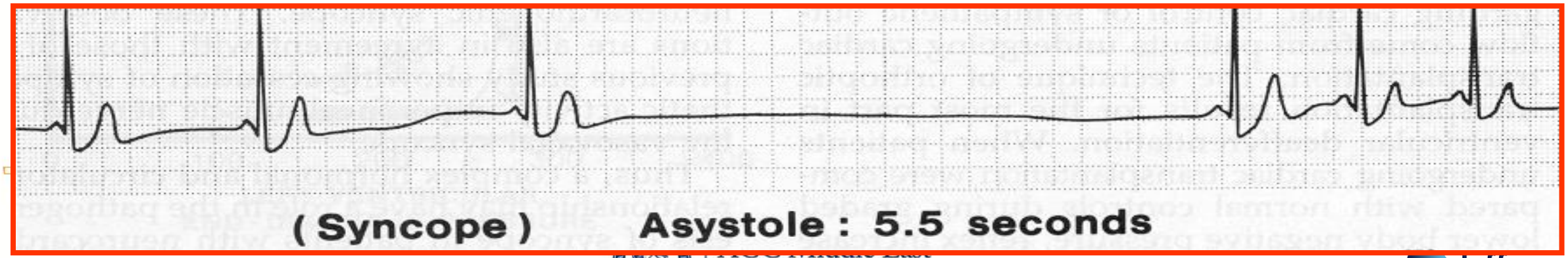
- Monitoring of cerebral blood flow in pts with syncope during HUT shows that **cerebral blood flow ↓ several minutes before syncope**
- When brain senses a ↓in blood supply → initiates **self-preservation reflex**
- After a period of heightened alertness/fear (when sympathetics are fully ↑↑) → the brain 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

(Sneddon J et al. Br Heart J 1994)





(Syncope) Asystole : 5.5 seconds

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 → **a + HUT in the elderly is more likely to be a TRUE POSITIVE**
- Greater ↓ in SBP; greater ↑ in DBP; no difference in Mean BP
- Lesser ↑ in HR
- More vasodepressor/dysautonomic, less cardio-inhibitory response

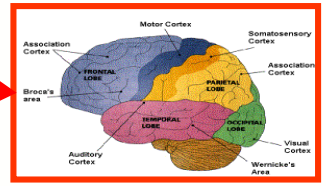
Treatment for VVS

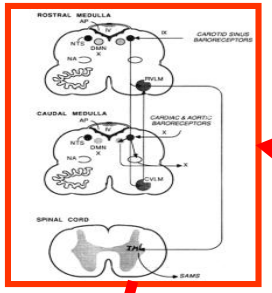
- **Non-Pharmacologic – most important!**
  - Salt, fluids, assume supine position with prodrome
  - Orthostatic self-training
  - Leg crossing with muscle tensing
  - Isometric arm contraction
- Medical
  - Midodrine
  - Fludrocortisone
  - SSRIs
- ? Pacing – rarely, in selected pts
- Rx can allow return to exercise in majority[1,2]

1. Kosinski D et al. Am Heart J 1999
2. Calkins H et al. Am Heart J 1995

Targets for Rx of VVS

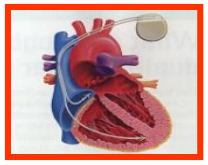
Higher centers
Emotional Conditioning
Counseling

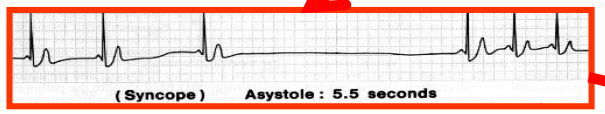




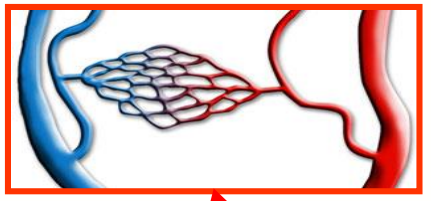
Nucleus Tractus Solitarius
SSRI
Thoephylline
Tilt training

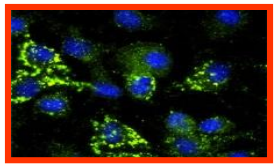
Bradycardia
?PM (rate and contractility)

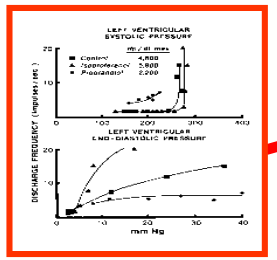




VVS







Increased Contractility
Beta-blockers?
Disopyramide

Peripheral Vasculature
Non-pharmacologic
Isometrics
Fluids/Salt
Florinef
Midodrine

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

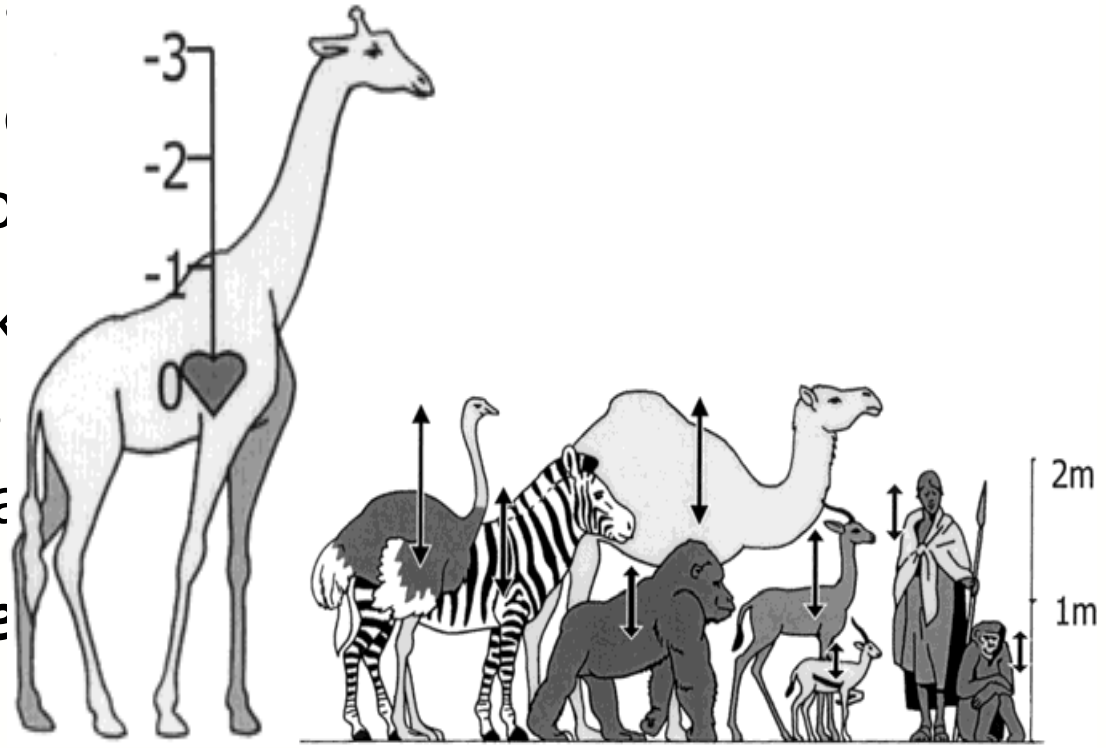
1. Human Conflict theory: During our evolution, inter-group attacks and killing, loss of consciousness triggered by fear-circuitry activation may have **conferred a survival advantage on non-combatants** (particularly children and women) when threats were inescapable – "sham death"
2. Clotting hypothesis: During traumatic bleeding, the lowering of the BP from VVS would **reduce blood loss** until stable blood clots formed

Bracha HS et al. The human fear-circuitry and fear-induced fainting in healthy individuals: The paleolithic-threat hypothesis. Clin Auton Res 2005;15:238–241

Levi M. Vasovagal fainting as an evolutionary remnant of the fight against hemorrhage Clin Auton Res 2005;15:69–70

Heart to Brain Height

- The human brain is about 40 cm above the heart
- Many mammals have greater brain elev
- A giraffe has a very muscular heart, gen
  mmHg, which allows a cerebral perfusic
- Giraffe: Upon lowering it's head to drink
  cerebral pressure should increase by ~3
  slow lowering, and large venous cerebra
- Other species are also vertical, but ***neve***
  triggered VVS... ***Why?***



van Dijk JG. Fainting in animals. Clin Auton Res 2003;13:247–255

Unique to Human Physiology... Three factors

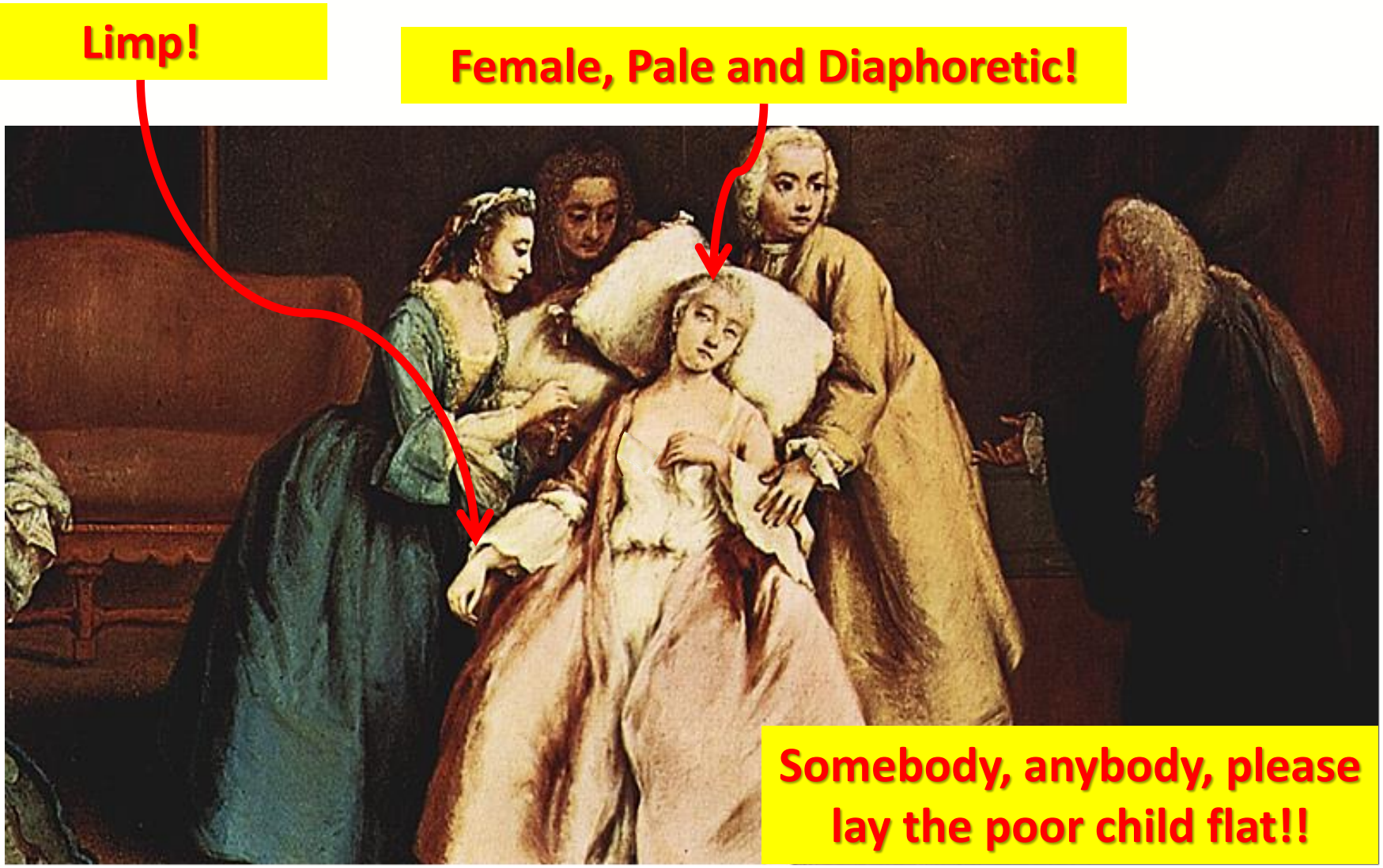
- 20% of cardiac output is destined for the human brain
  - 6.7% in Chimpanzees, 1% in giraffes
  - Also, human brain consumes 25% of whole-body glucose consumption; needs constant blood supply, since neurons cannot store glucose
- Human legs are larger (related to overall body size), with large venous 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!

van Dijk JG. Fainting in animals. Clin Auton Res 2003;13:247–255

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



A 1744 oil painting by Pietro Longhi titled *Fainting*