

# Neurocardiogenic syncope

# Syncope Definition

- Collapse, Blackout
- A sudden, transient loss of consciousness and postural tone, with spontaneous recovery

# Syncope Prevalence

- Very common
- All age groups (particularly elderly)
- 3 % of attendances in A & E (6% elderly)
- 1% of all hospital admissions

# Aetiology

- Consciousness depends upon normal functioning of both the cerebral hemispheres and the brain stem

# Initial evaluation

- History
- Orthostatic BP measurements
- 12 lead ECG

- Syncope or non-syncope?
- Any clinical features within the history to suggest diagnosis?
- Is heart disease present or absent?

# Non-syncope

- Impaired consciousness (hypoxia, hyperventilation, hypoglycaemia, epilepsy)
- Apparent loss of consciousness (psychogenic disorders)

# The value of history

## ■ Eyewitness

- Seizure likely; tonic-clonic movements tongue biting, blue face
- Syncope likely; tonic-clonic start after loss of consciousness

## ■ Symptoms prior to the event

- Seizure; Aura
- Syncope; nausea, vomiting, sweating, pallor

## ■ symptoms after the event

- Seizure; prolonged confusion, muscle ache
- Syncope; nausea, vomiting, sweating, pallor



# Heart disease or not?

- Presence of heart disease – strong predictor of cardiac syncope
- Absence of heart disease usually precludes cardiac cause except if due to tachycardia
- Absence of heart disease may be due to neurally mediated tachycardia

# Cerebral Hemisphere Dysfunction

- Impaired cerebral perfusion due to a cardiovascular cause
  - Brady-Tachy arrhythmias
  - LV/RV outflow tract obstruction
  - Orthostatic hypotension
  - Neurocardiogenic syncope

# Cerebral Hemisphere Dysfunction

- Hypoglycaemia
- Hypoxia
- Hyperventilation
- Generalized Seizures

# Local Brain Stem Dysfunction

- Vertebrobasilar transient ischaemia
- Complex partial seizures
- Migraines

# Aetiology

- Long list of potential causes
- Precise diagnosis is often difficult, protracted and expensive
- Essentially – Cardiovascular vs Neurological
- Large number of undiagnosed - neurocardiogenic

# Neurocardiogenic syncope

## Synonyms

- Neurally mediated syncope
- Neuro-cardiogenic syncope
- Reflex syncope
- Neuro-regulatory syncope

# Neurocardiogenic Syncope Definition

- 'Autonomically-mediated reflex mechanisms associated with inappropriate vasodilation and/or bradycardia causing syncope'

# Specific syndromes

- Vasovagal syncope
- Situational syncope
- Carotid sinus syncope



# Autonomic nervous system

## Function

- Short term control of the internal environment of the body
- Innervation of smooth muscle, cardiac muscle and glands
- Able to control – BP, peristalsis, body temperature, micturition etc.
- All control occurs at a subconscious level
  - Reflexes in spinal cord
  - Influence of higher centers (brainstem)

# Autonomic Nervous system Structure

- Two divisions
  - Sympathetic limb
  - Parasympathetic limb

# Sympathetic system

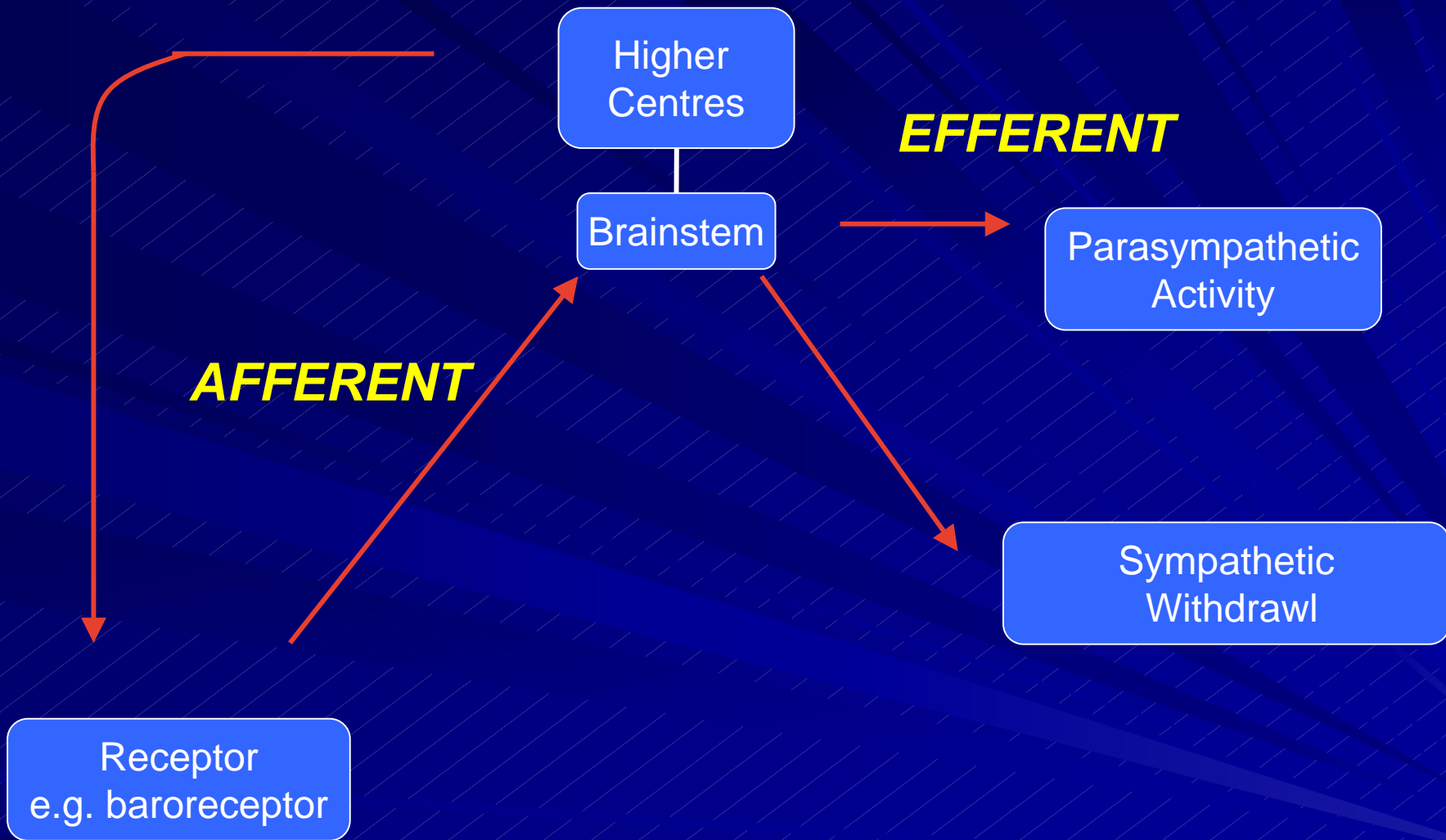
- Preganglionic neurones from thoracic/lumbar spinal cord
- 'Flight or fright' effect, which prepares the body for activity required in a hostile environment
- Increases heart rate and blood flow to skeletal muscles

# Parasympathetic system

- Preganglionic neurones in the cranial/sacral segments
- Control of internal functions
- Decreases HR and promotes digestive function

# Mechanism

- Involves pathophysiological autonomic reflex
- Triggering factors, modulating factors and afferent pathways vary



# Receptors

## Receptor

## Syndrome

■ Vasovagal syncope

Ventricular  
mechanoreceptors

■ Micturition syncope

Bladder  
mechanoreceptors

■ Carotid Sinus  
hypersensitivity

Carotid sinus  
baroreceptors

# All induce either;

- Vasodepressor effect
- Cardio-inhibitory effect
- Mixed



# Diagnostic tests

- Carotid sinus massage
- Tilt testing
- Others; EP testing, signal averaged (V) ECG, Echocardiography, ETT, cardiac catheterisation, neurological/psychiatric evaluation,

# Carotid sinus massage

- CSM recommended in patients > 40yrs, syncope of unknown cause
- Avoid if risk of stroke
- ECG monitoring, BP monitoring
- Minimum 5 minutes, maximum 10 minutes
- Perform patients supine and standing
- Avoid patients carotid bruits

# Tilt table testing

- Supine at least 5 minutes prior to tilt
- Supine at least 20 minutes prior to tilt if cannulation is preferred
- Tilt angle 60 - 70 degrees
- Passive phase min 20 minutes, max 45 minutes
- Use either intravenous isoprenaline or sublingual GTN if passive phase is negative
- Pharmacological phase – 15 to 20 minutes
- End-point; induction syncope or completion planned tilt

# Vasovagal Syncope Features

- Always occurs with the thorax in the vertical position
- Often seen in the young
- May occur in response to fear, injury, prolonged standing
- Provoked – motionless, upright position (Tilt tests)

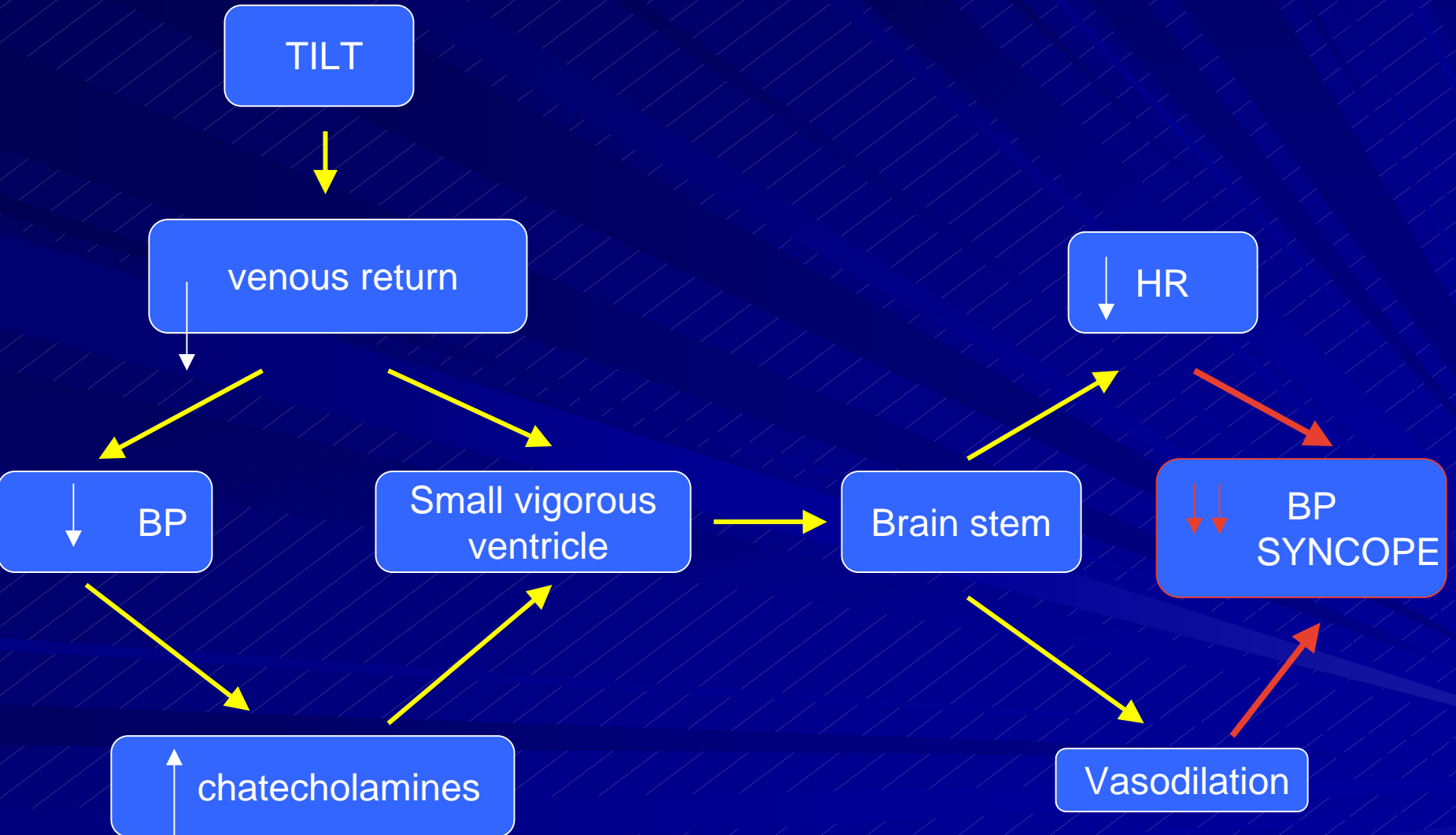
# Mechanism

- Blood pools in venous capacitance vessels
- Reduced venous return and cardiac output
- Baroreceptor mediated  $\uparrow$  sympathetic activity
- Mild  $\uparrow$  in BP and HR
- Paradoxical stimulation of ventricular mechanoreceptors
- Usually stimulated by vigorous contraction

# Contd.

- Afferent pathways activated via Vagal C fibres
- Vasomotor center stimulated
- Stimulation of parasympathetic activity (vagusnerve)
- Withdrawl of sympathetic activity
- BP and HR fall
- Reduced cerebral perfusion - syncope

# Bezold-Jarisch Reflex



# Summary

- The Vasovagal response is an inappropriate vessel dilatation and bradycardia response leading to reduced cerebral perfusion and syncope



# Summary

- Often warning signs
  - nausea
  - warmth
  - lightheadedness

# Summary

- Head up tilt identifies those at risk of neurocardiogenic syncope

# Summary

- Mechanism of tilt induced syncope - Bezold-Jarisch Reflex
- Venous pooling - Vigorously contracting yet small sized ventricle
- Ventricular mechanoreceptor stimulation
- muscle bed vasodilatation and cardiac slowing

# Summary

- Muscle bed vasodilatation usually always precedes cardiac slowing and may contribute further to a reduced venous return - perpetuates the response

# Summary

- Remote from the attack there are no clinical signs to give a diagnosis
- Infrequency of attacks makes diagnosis difficult
- ILR useful, however Tilt table Testing is the test of choice for this patient group.

# References

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- Baron-Esquivas European heart 2002
- Farwell Heart 2004