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
Higher Centres

Brainstem

Parasympathetic Activity

Sympathetic Withdrawal

Receptor e.g. baroreceptor

AFFERENT

EFFERENT
Receptors

Syndrome

- Vasovagal syncope
- Micturition syncope
- Carotid Sinus hypersensitivity

Receptor

- Ventricular mechanoreceptors
- Bladder mechanoreceptors
- 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 ↑ sympathetic activity
- Mild ↑ 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 (vagus nerve)
- Withdrawal of sympathetic activity
- BP and HR fall
- Reduced cerebral perfusion - syncope
Bezold-Jarisch Reflex

TILT

venous return

BP

Small vigorous ventricle

Brain stem

HR

BP SYNCOPE

Vasodilation

chateholamines
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
Head up tilt identifies those at risk of neurocardiogenic syncope
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
Muscle bed vasodilatation usually always precedes cardiac slowing and may contribute further to a reduced venous return - perpetuates the response
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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