Medicine/ Dr.sabah

Syncope and Presyncope

Syncope =sudden loss of consciousness due to reduced cerebral perfusion.transient,self limitted

(onset is rapid, duration brief, and recovery spontaneous and complete)

Presyncope(syncopal prodrome) = lightheadedness where individual thinks he or she may black out. (dizziness, lightheadedness or faintness, weakness, fatigue, and visual and auditory disturbances)

- Syncope affects around 20% of the population at some time

- accounts for more than 5% of hospital admissions.

- Dizziness and presyncope are very common in old age .

VERTIGO: Abnormal perception of movement of the environment

SEIZURES: clinical event caused by abnormal electrical discharge in brain

Pathophysiology

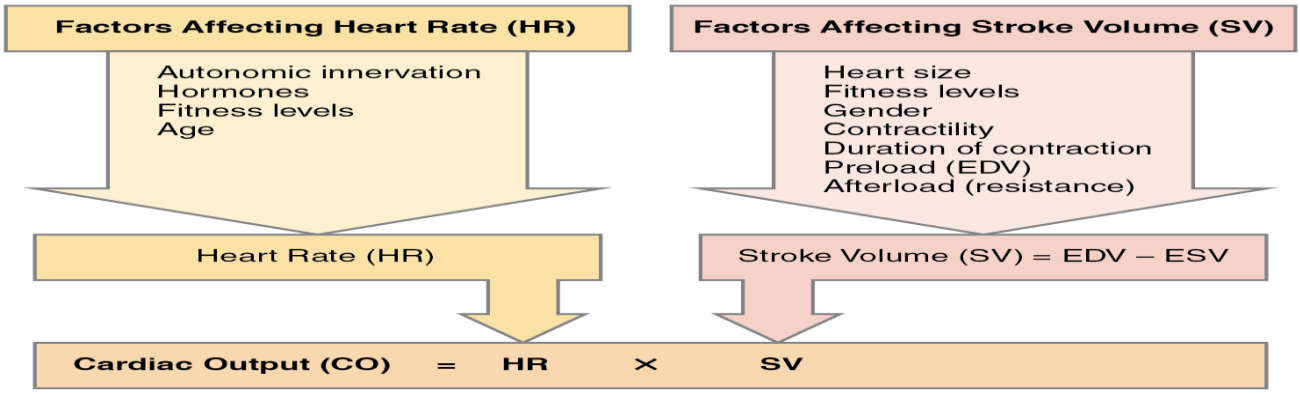
Cerebral blood flow ranges from 50 to 60 mLlmin per 1 00 g brain tissue

Cessation of blood flow for 6-8 s ➔loss of consciousness,

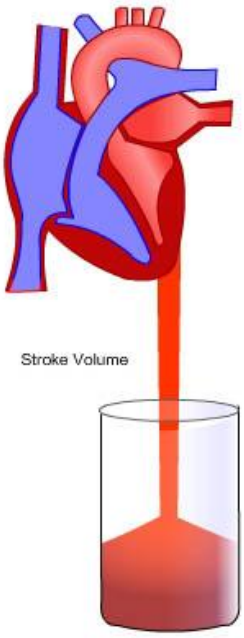
BF decreases to 25 mLlmin per 100 g BT➔ impairment of consciousness

clinically =fall in systemic systolic BP to -50 mmHg or lower ➔syncope.

decrease in cardiac output and/or systemic vascular resistance-the determinants of BP—underlies the pathophysiology of syncope.



impaired cardiac output = decreased effeıtive circulating B volume; increased thoracic P; massive pulm.y embolus; cardiac brady- and tachyarrhythmias; valvular heHD; and myocardial dysfunction.



To increase cardiac output

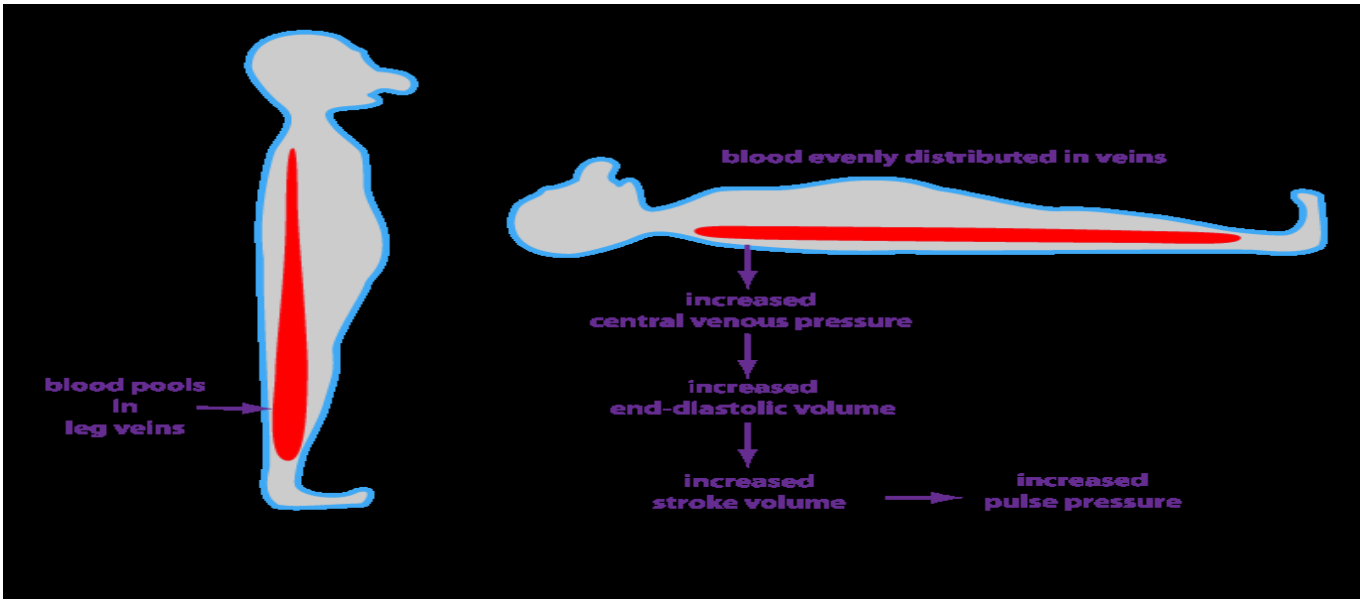
Increase stroke volume
or

Increase heart rate
or
increase both

Systemic vascular resistance =decreased by central and peripheral autonomic nervous system diseases, sympatholytic medications and transiently during neurally mediated syncope. Increased cerebral vascular resistance(hypocarbia induced by hyperventilation) contribute.

•Upright posture, Standing ➔ pooling of 500–1000 mL of blood in lower extremities & splanchnic circulation.➔ decrease in venous return to the heart and reduced ventricular filling➔ diminished cardiac output and blood pressure.

compensatory reflex response: (initiated by baroreceptors in carotid sinus and aortic arch)➔increased sympathetic outflow and decreased vagal nerve activity . == increases peripheral resistance, venous return to the heart, and cardiac output and thus limits the fall in blood pressure.

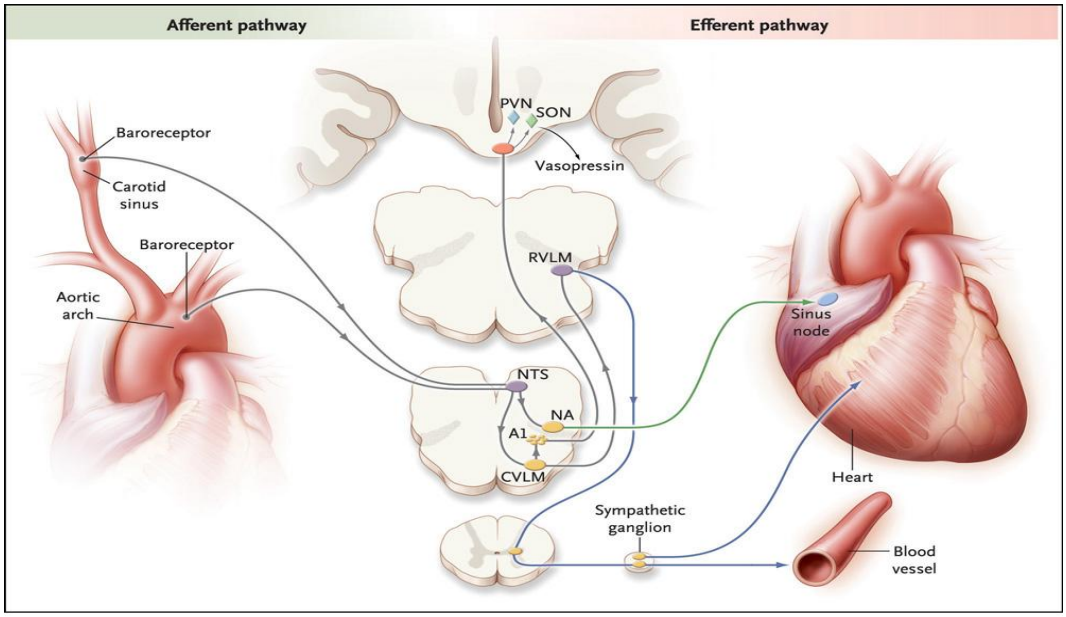


Impaired -- cerebral hypoperfusion

---chronically in orthostatic hypotension and

---transiently in neurally mediated syncope,

Baroreflex. decrease in arterial pressure unloads baroreceptors(terminals of afferent fibers of the glossopharyngeal and vagus nerves- situated in carotid sinus and aortic arch). ➔reduction in afferent to medulla. Reduced baroreceptor afferent activity ➔decrease in vagal nerve input to the sinus node ,increase in sympathetic efferent In response to a sustained fall in blood pressure, vasopressin release



MECHANISMS-CAUSES

Three principle mechanisms underlie recurrent presyncope or syncope:

•**cardiac syncope** = mechanical cardiac dysfunction or arrhythmia

• **neurocardiogenic syncope=** Abnormal autonomic reflex ➔bradycardia and/or hypotension

• **postural hypotension**=physiological peripheral vasoconstriction on standing is impaired ➔hypotension.

•**Non-cardiac** pathology causes of blackout = epilepsy, cerebrovascular ischaemia or hypoglycaemia

Classification

A-NEURALLY MEDIATED SYNCOPE:

•**Vasovagal syncope** (Provoked fear, pain, anxiety, intense emotion, sight of blood, unpleasant sights and odors, orthostatic stress)

•**Situational reflex syncope**

Pulmonary. Urogenital. Gastrointestinal. Cardiac. Carotid sinus. Ocular

B. ORTHOSTATIC HYPOTENSION:

-Primary autonomic failure

-Secondary autonomic failure,DM

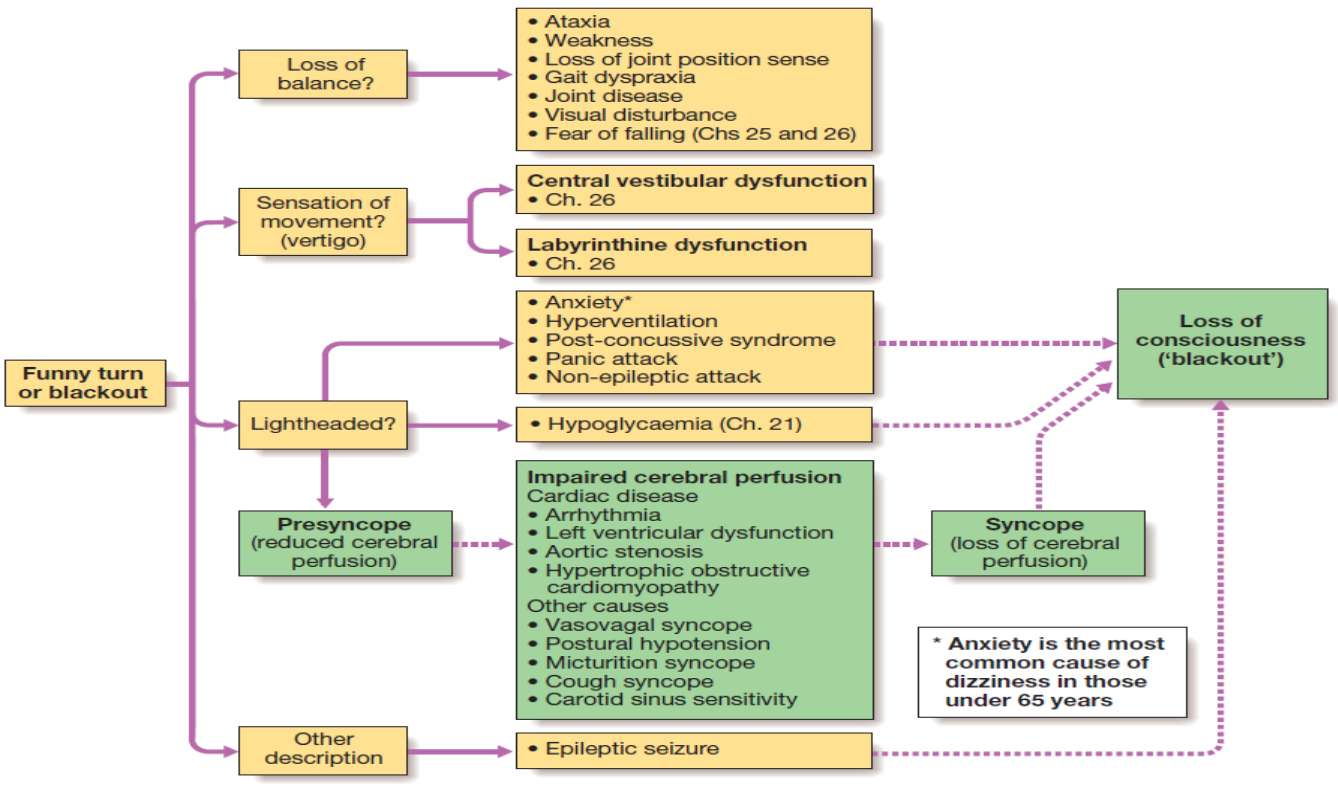
-Postprandial hypotension

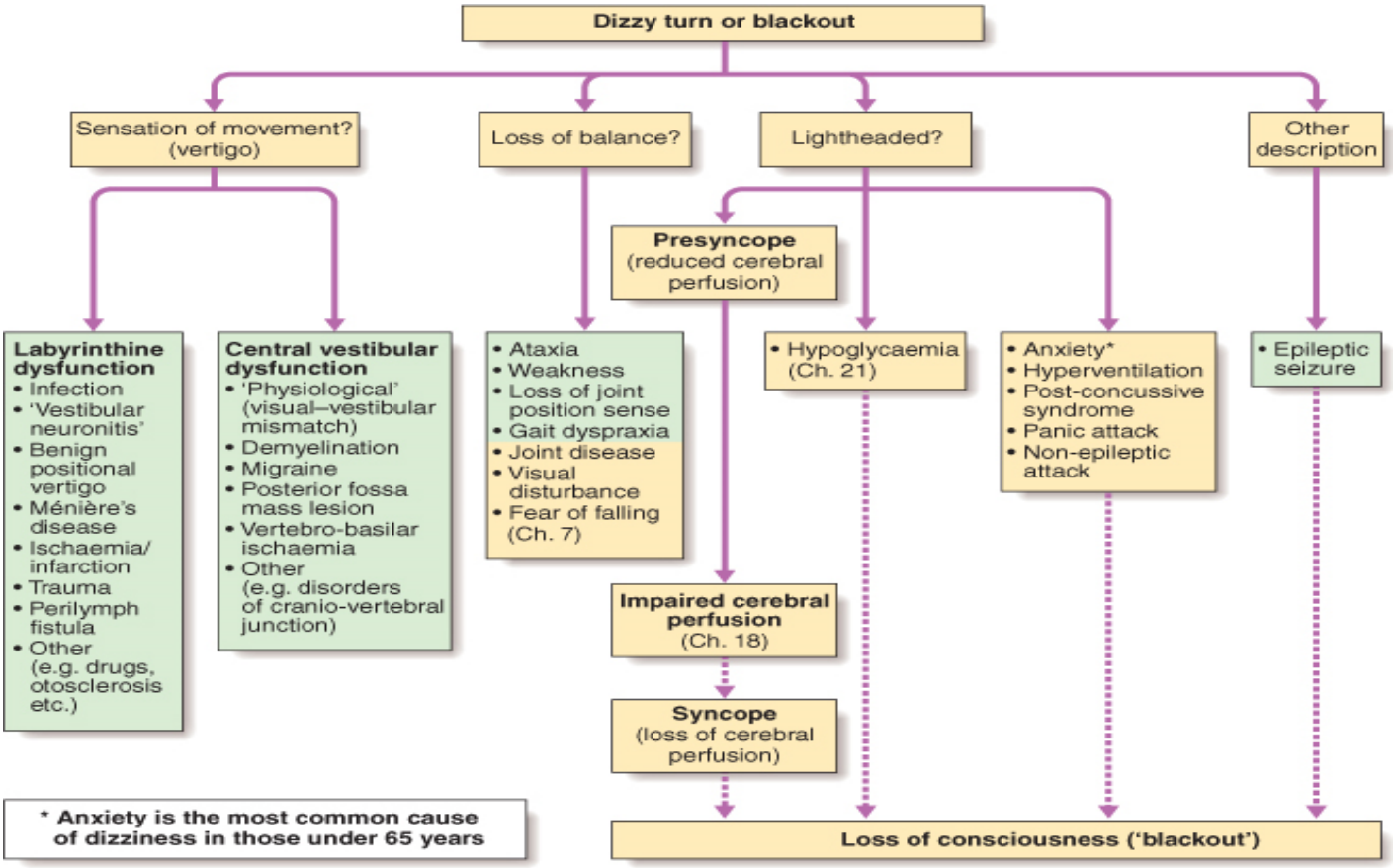
-Iatrogenic (drug-induced)

-Volume depletion

C. CARDIAC SYNCOPE: •Arrhythmias •Cardiac structural disease

Differential diagnosis of syncope and presyncope:

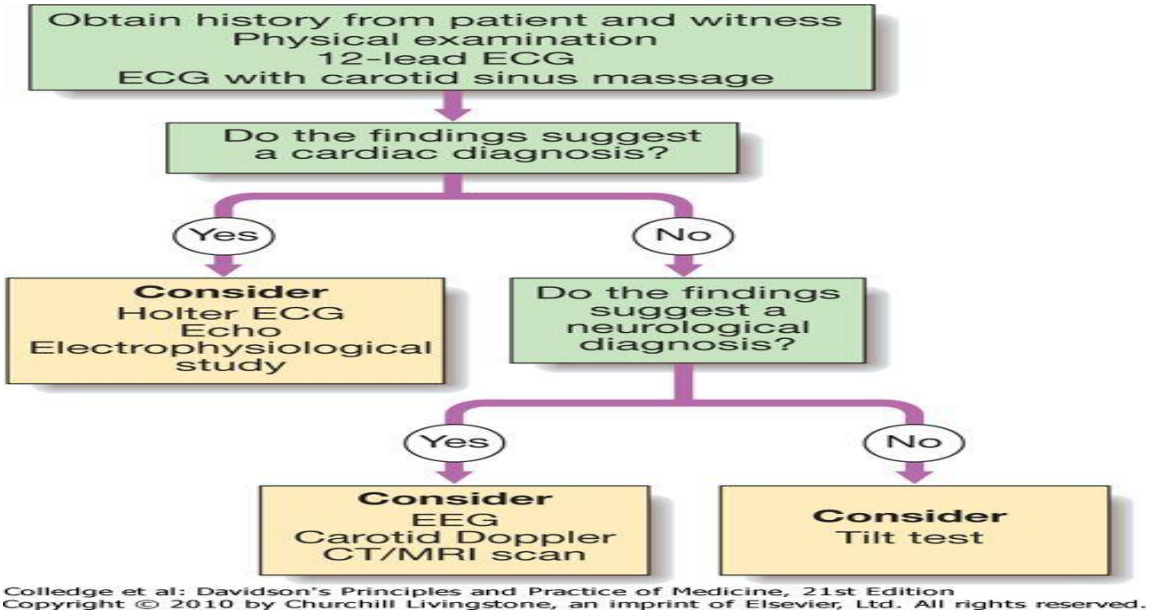




18.20 Typical features of cardiac syncope, vasovagal syncope and seizures

	Cardiac syncope	Neurocardiogenic syncope	Seizures
Premonitory symptoms	Often none Lightheadedness Palpitation Chest pain Breathlessness	Nausea Lightheadedness Sweating	Confusion Hyperexcitability Olfactory hallucinations 'Aura'
Unconscious period	Extreme 'death-like' pallor	Pallor	Prolonged (> 1 min) unconsciousness Motor seizure activity* Tongue-biting Urinary incontinence
Recovery	Rapid recovery (< 1 min) Flushing	Slow Nausea Lightheadedness	Prolonged confusion (> 5 mins) Headache Focal neurological signs

*N.B. Cardiac syncope can also cause convulsions by inducing cerebral anoxia.



Differential diagnosis

History-taking, from the patient or a witness= establish diagnosis.

Attention should be given to

1.potential triggers (e.g. medication, exertion, posture),

2.victim's appearance (e.g. colour, seizure activity),

3.duration of the episode and

4.speed of recovery .

Cardiac syncope: usually sudden, can be associated with premonitory lightheadedness, palpitation or chest discomfort. blackout is usually brief and recovery rapid.

Neurocardiogenic syncope

often be associated with situational trigger, and the patient may experience flushing, nausea and malaise for several minutes afterwards.

Patients with **seizures** do not exhibit pallor, may have abnormal movements, usually take more than 5 minutes to recover and are often confused.

A history of rotational **vertigo** is suggestive of a labyrinthine or vestibular disorder .

pattern and description of the patient's symptoms i

1-CARDIAC SYNCOPE (Arrhythmia& 2-Structural heart disease)

Arrhythmias

Lightheadedness =many arrhythmias,

Blackouts (Stokes-Adams attacks,) =profound bradycardia or malignant ventricular tachyarrhythmias.

DIAGNOSIS:

1) 12-lead ECG =evidence of conducting system disease (e.g. sinus bradycardia, AV block, BBBor axis deviation) which would predispose a patient to bradycardia,

2) ECG recording *during symptoms*.= key to establishing a diagnosis (minor rhythm disturbances are common, especially in old age, symptoms must occur at same time as a recorded arrhythmia before a diagnosis can be made).

3) Ambulatory ECG recordings =helpful only if symptoms occur several times/week.

4) Patient-activated ECG recorders =useful for examining rhythm in patients with recurrent dizziness, but are not useful in assessing sudden blackouts.

5) implantable 'loop recorder= placed subcutaneously in the upper chest. device continuously records the cardiac rhythm and will activate automatically if extreme bradycardia or tachycardia occurs. The ECG memory can also be frozen by the patient using a hand-held activator. Stored ECGs can be accessed by the implanting centre, using a telemetry device.

CARDIAC SYNCOPE

Structural heart disease

lightheadedness or syncope on exertion ➔

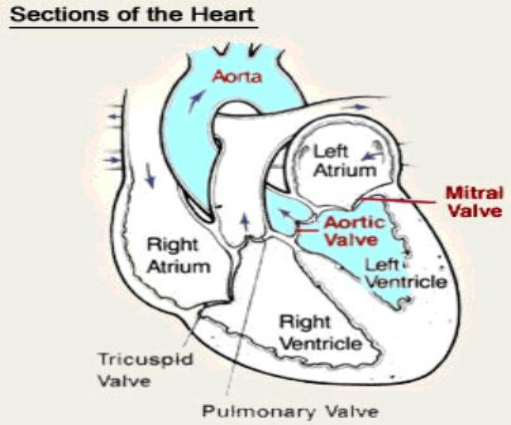
1-Severe aortic stenosis,

2- hypertrophic obstructive cardiomyopathy

3-severe coronary artery disease

caused by profound hypotension due to a fall in cardiac output, or failure to increase output during exertion, coupled with exercise-induced peripheral vasodilatation.

Exertional arrhythmias also occur in these patients.



2-POSTURAL HYPOTENSION

failure of the normal compensatory mechanisms.

1-Relative hypovolaemia (excessive diuretic therapy),

2-sympathetic degeneration (diabetes mellitus, Parkinson's disease, ageing)

3-drug therapy (vasodilators, antidepressants)

Treatment = often ineffective; withdrawing unnecessary medication, wear graduated elastic stockings and get up slowly

Fludrocortisone, which can expand blood volume through sodium and water retention, may be of value.

Orthostatic Hypotension

•**Primary autonomic failure** (idiopathic central and peripheral neuro degenerative diseases-the synucleinopathies"

-Lewy body diseases

-Parkinson's disease

-Lewy body dementia

*Pure autonomic failure

*Multiple system atrophy (the Shy-Drager syndrome)

•**Secondary autonomic failure** =autonomic peripheral neuropathies

-Diabetes

-Hereditary amyloidosis (familial amyloid polyneuropathy)

-Primary amyloidosis (AL amyloidosis; immunoglobulin light chain)

-Hereditary sensory and autonomic neuropathies (HSAN) (especially type II-familial dysautonomia)

-Idiopathic immune-mediated autonomic neuropathy

•**Autoimmune autonomic ganglionopathy**

-sögren's syndrome

-Paraneoplastic autonomic neuropathy

-HIV neuropathy

•**Postprandial hypotension**

•**Iatrogenic (drug-induced)**

•**Volume depletion**

3-Neurocardiogenic syncope

-family of syndromes =bradycardia and/or hypotension occur because of a series of abnormal autonomic reflexes.

-Vasovagal syncope

-Carotid sinus Syndrome

-Situational= neurocardiogenic syncope that occur in the presence of identifiable triggers (e.g. cough syncope, micturition syncope).

Neurally Mediated Syncope

Vasovagal syncope

Provoked fear, pain, anxiety, intense emotion, sight of blood, unpleasant

sights and odors, orthostatic stress

Situational reflex syncope

1.Pulmonary: Cough syncope, wind instrument player's syncope, weightlifter's syncope, "mess trick and "fainting lark sneeze syncope, airway Instrumentatlon

2.Urogenital : Postmicturition syncope, urogenital tract instrumentation, prostatic massage

3.Gastrointestinal: Swallow syncope, glossopharyngeal neuralgia, esophageal stimulation, gastrointestinal tract instrumentation, rectal examination, defecatlon syncope

4.Cardiac: Bezold-Jarisch reflex, cardiac outflow obstruction

5.Carotid sinus: Carotid sinus sensitivity, carotid sinus massage

6.Ocular: Ocular pressure, ocular examination, ocular surgery

Vasovagal syncope

normally triggered by reduction in venous return =prolonged standing, excessive heat or large meal.

mediated by Bezold-Jarisch reflex, (initial sympathetic activation ➔vigorous contraction of relatively underfilled ventricles➔ventricular mechanoreceptors, producing parasympathetic (vagal) activation and sympathetic withdrawal➔causing bradycardia, vasodilatation or both.

Head-up tilt-table testing (provocation test used to establish diagnosis,) patient to lie on table that is then tilted to an angle of 60-70 ° for up to 45 minutes, while ECG and BP are monitored. positive test =bradycardia (cardio-inhibitory response) and/or hypotension (vasodepressor response) +typical symptoms.

Initial management =lifestyle modification (salt supplementation and avoiding prolonged standing, dehydration or missing meals).

Resistant cases= drug therapy

*Fludrocortisone=sodium and water retention and expands plasma volume

*β-blockers, which inhibit initial sympathetic activation,

*disopyramide (a vagolytic agent) or

*midodrine (a vasoconstrictor α-adrenoceptor agonist).

Dual-chamber pacemaker =if symptoms predominantly due to bradycardia.

Patients with a urinary sodium excretion of <170 mmol/day = salt loading.

Carotid sinus hypersensitivity

presyncope or syncope because of reflex bradycardia and vasodilatation.

Carotid baroreceptors -involved in BP regulation --activated by increased BP ➔vagal discharge ➔ compensatory drop in BP.

In HCSS = baroreceptor is sensitive to external pressure (e.g. during neck movement or if a tight collar is worn), =pressure over the carotid artery ➔inappropriate and intense vagal discharge.

Diagnosis :

monitoring ECG and BP during carotid sinus massage for 6 seconds. *(should not be attempted in patients with a carotid bruit or with a history of cerebrovascular disease -risk of embolic stroke).*

positive cardio-inhibitory response =sinus pause of 3 seconds or more;

positive vasodepressor response =fall in systolic BP of >50 mmHg.

Carotid sinus pressure will produce positive findings in about 10% of elderly individuals but less than 25% of these experience spontaneous syncope. Symptoms should not therefore be attributed to HCSS unless they are reproduced by carotid sinus pressure.

Dual-chamber pacing usually prevents syncope in patients with the more common cardio-inhibitory response.

Done by: Sama Adeeb