





Welcome - The agenda

9h00 -10h30: DIZZINESS and SYNCOPE, S.Gillain, Geriatrician

Break

10h45-12h15: OPHTALMOLOGICAL DISORDERS, J.M. Rakic, Ophtalmologist

Lunch

13h00 - 14h30: EARS NOSE and THROAT disorders and VERTIGO, P. Lefebvre, ENT Specialist

Break

14h45 -16h15 ONCOLOGICAL CARES, J. Collignon, Oncologist

16h15-16h30: Last QnA session











Professor Jean Marie Rakic

Head of the department of ophthalmology in CHU Liège

Publications list available at: https://orbi.uliege.be/phsearch?uid=U013922







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Lecturers

Professor Philippe Lefebvre

Head of the department of Ear Nose and Throat in CHU Liège

Publications list available at: https://orbi.uliege.be/ph-search?uid=U008700









Lecturers

Professor Joelle Collignon

Oncologist – Digestive pathologies in CHU Liège

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16h15mleh3QvidastaQnAusession







DIZZINESS and SYNCOPE

S. GILLAIN sgillain@chuliege.be

DIZZINESS and SYNCOPE

Symptoms

Frequent

Multiple causes

Decrease in Survival - Autonomy - QOL

Identified – Investigated - Integrated

Dizziness -Content

Definition and epidemiology

Clinical consequences

Physiopathology

Etiology and clinical presentations

Evaluation

Management

Take home messages

Learning objectives - « to be able to »

At the end of this lecture, you should be able to manage

- A goal-oriented anamnesis;
- A goal-oriented physical exam;
- A diagnostic algorithm;
- > A multidisciplinary management;
- A patient and caregivers education (if possible)

Dizziness - Definition

Dizziness is a **broad term**

→ « A various abnormal sensation arising from perception of the body's relation to space or of unsteadiness »

→ « A sensation of postural instability or imbalance »

« Chronic » if present for > 2 months

Dizziness - Epidemiology

About 15% to over 20% of adults yearly in large population-based studies.

Most frequent identified causes are

→ 25% Vestibular vertigo (next lecture)

 \rightarrow 75 % who are left

Benign positional paroxysmal vertigo (BPPV)

Vestibular migraine,

Comorbidities consequences, toxic effects...

Menière's disease

Dizziness - Clinical Consequences

- > Fear of falling;
- Reduced spontaneous mobility;
- > Falls and injuries;
- > Mobility decline;
- > Sarcopenia;
- > Thymic disorders behavioural disorders;
- Reduced energy incomes;
- > Frailty

Dizziness - Clinical Consequences

- > Fear of falling;
- Reduced spontaneous mobility;
- > Falls and injuries;
- > Mobility decline;
- > Sarcopenia;
- > Thymic disorders behavioural disorders;
- Reduced energy incomes;
- Frailty

Add the clinical consequences of frailty

- → Disability
- → Increased risk of
 - → Delirium
 - → Hospitalisation
 - → Longer inpatient after procedure
 - → Institutionalisation
 - → Death

Dizziness - Content

Definition and epidemiology

Clinical consequences

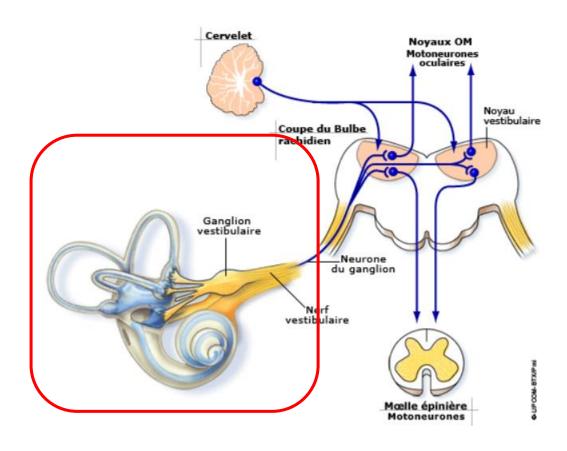
Physiopathology

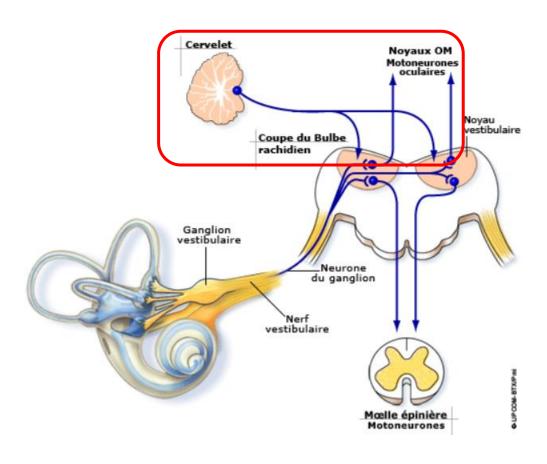
Etiology and clinical presentations

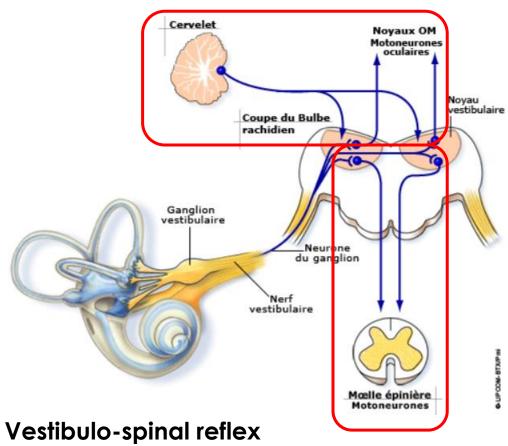
Evaluation

Management

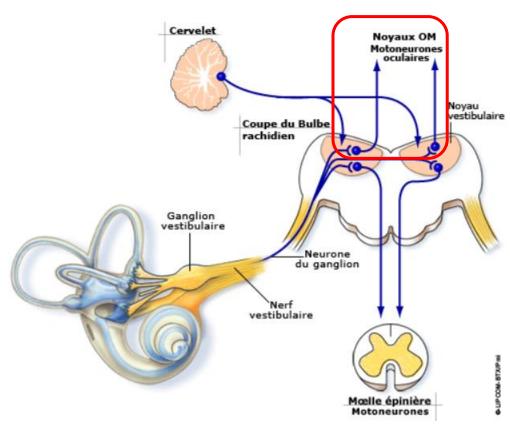
Take home messages





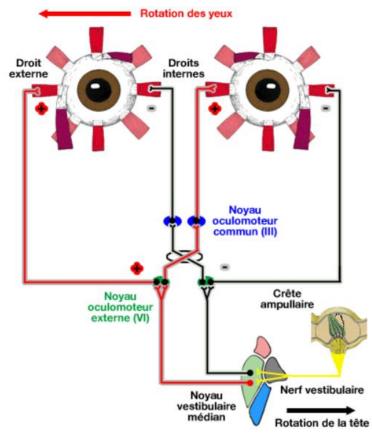


- → keep face and shoulder
- → anti-gravity muscles action



Vestibulo-spinal reflex

- → keep face and shoulder
- → anti-gravity muscles action



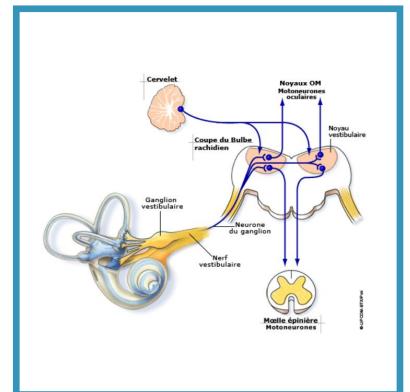
Vestibulo-occular reflex

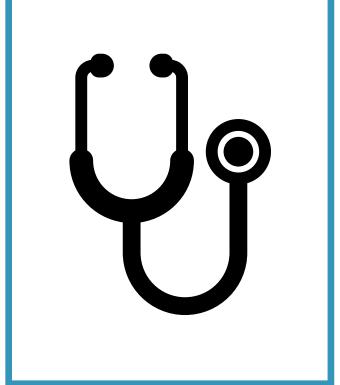
→ keep eyes « focused on »

Vestibular system Anamnesis / Clinical exam

Symptoms: Vertigo, dizziness, nausea, sudation, pallor and not well-being, Balance/walking disorders

Clinical exam:
Nystagmus
Lateralised Romberg
Ataxic walking
Laterilazed walk in
place with closed eyes





Vestibular system Anamnesis / clinical exam





To confirm:

- →Electronystagmography;
- → Inner Ear MRI;
- → Brain MRI → Brainstem lesion

Cerebellum Anamnesis / clinical exam

None vertigo, Nystagmus could be

Wide base support Oscillations during Romberg

Hypermetria
Tremor of intention
Tremor of attitude
A drunken gait
A dysarthria,
An hypotonia,
Pendular reflexes





Vascular Lesion;

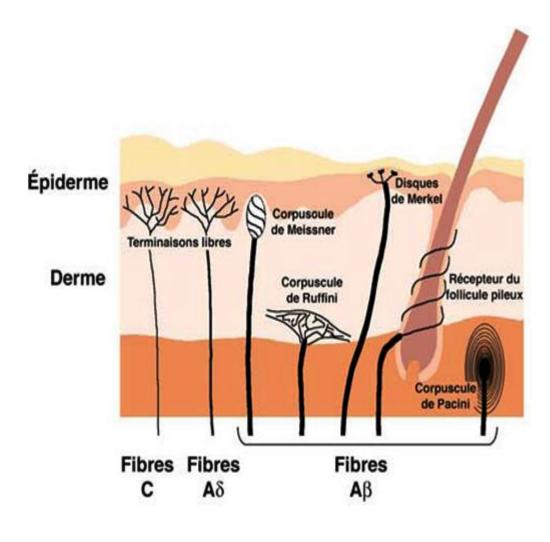
Meningioma, medulloblastome, or glioblastoma;

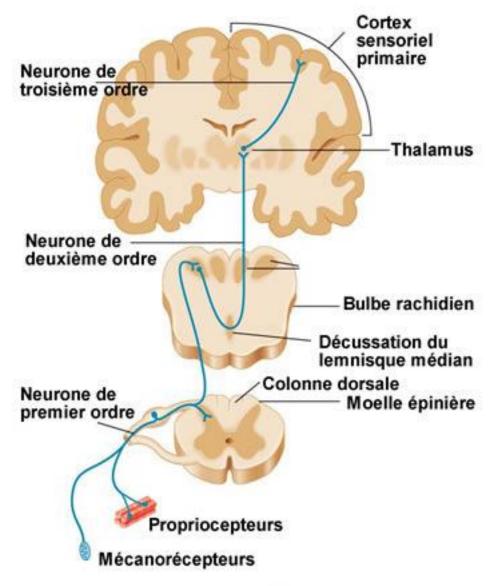
Secondary neoplastic lesions more frequent : breast, pulmonary, kidney, thyroid cancer or melanoma.

Proprioceptors

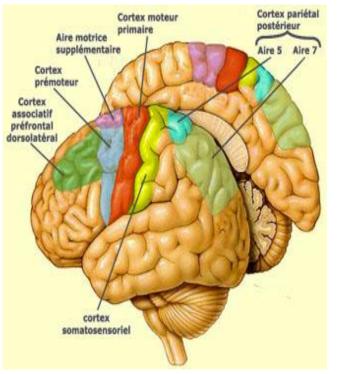
Collaterals to dorsal columns Dorsal root Dorsal root ■ Inhibitory ganglioninterneuron la fiber · lb fiber LMNs in ventral root LMN Muscle spindle (activated by ↑ in Leg extensormuscle stretch) muscles (quadriceps) Motor end plates Leg flexor muscles (hamstrings) Patellar tendon Golgi tendon organ (activated by ↑ in muscle Muscle stretch Inverse muscle force) reflex stretch reflex (causes stretched (causes activated muscle to contract) muscle to relax)

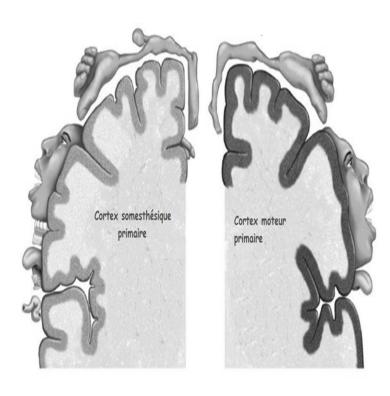
Mechanoreceptors





Sensitive Lemniscal Ascending Path



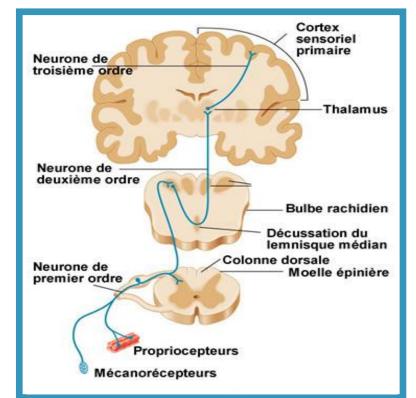


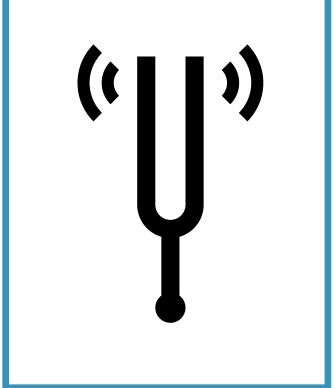
Proprioceptive system Anamnesis / clinical exam

Symptoms: dizziness and instability

Clinical exam:

- little oscillations when the eyes are closed (Romberg)
- Patient needs to look his feet when is aked to join them. In order not to fall he needs to put his feet apart.
- Walk: wide base of support, irregular steps and lateral deviations
- Vibration sensitivity





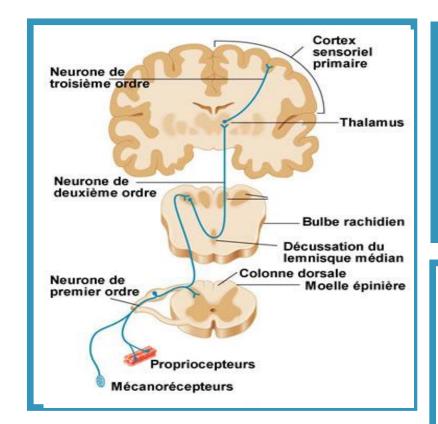
Proprioception Investigations

Medical history:

Diabetes, Chronic alcohol consumption, Cancer, Hypothyroid, Renal deficiency, Chronic hepatitis, Neurotoxic molecules and VIH

Electromyogram of lower limbs not systematically

(Spinal MRI)



Vincristine, cisplatine, vinblastine, doxyrobutine;

Antibiotics: isoniazide, métronidazole, éthambutol, nitrofurantoïne, colistine, dapsone;

Anti-malaria

Antiretroviral

Lead and mercury

Investigating proprioception

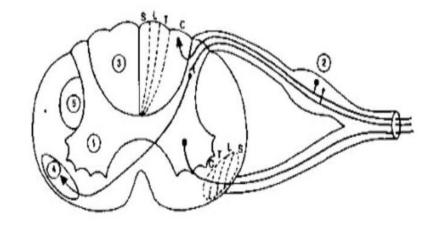
Electromyogram of lower limbs not systematically

Spinal MRI in case of spinal lesion suspected

As a reminder a compressive spinal lesion will be associated to

→ A lesionel syndrome : radiculopathy

→ A sub-lesionel syndrome : homolateral pyramidal syndrome, posterior cord syndrome, heterolateral thermoalgesic anesthesia.



- 1: Anterior Horn (second motor neuron)
 - 2: Spinal nodes (sensitive neurone)
 - 3: Lemniscal path (Goll et Burdach)
 - 4: Spnio-thalamic bundle
 - 5: Pyramidal bundle

Dizziness – Causes

Information from vestibular/ Visual/ Proprioceptive systems

Integrated in cerebellum and hemispheres -> adapted motor order

Causes of failure could be

- Age-related
- Disease-related
- Drug-related
- Toxicant-related

Age-related changes

Vestibular system: Hair cell loss, Neuronal loss;

Visual system: Age related clouding of the lens, and macular degeneration, reduced visual acuity, reduced dark adaptation, reduced contrast sensitivity, reduced accompodation;

Proprioceptive system: Reduced mechanoreceptors sensitivity, slower peripheral conduction;

Central nervous system including cerebellum: neuronal loss, reduced synapsis plasticity, reduced neurotransmitter concentration;

Vestibular system: peripheral failure

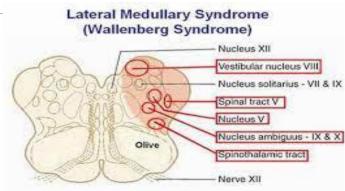
- Ménière disease,
- Benign paroxysmal positional vertigo,
- Acoustic neuroma,
- Recurrent vestibulopathy
 - attacks of vertigo without auditory or neurological symptoms, two-thirds of patients experienced a spontaneous resolution
- Viral vestibular/ labyrinthitis neuronitis,
- Drug toxicity (aminoglycosides)
- Head trauma

Vestibular system: central failure

Most often: Tumor of brainstem

Wallenberg syndrome

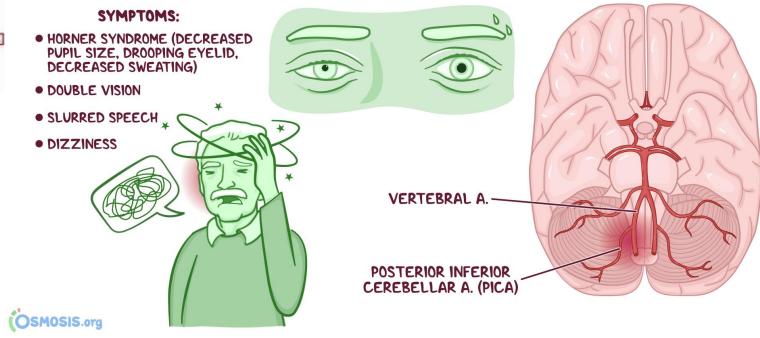
Rarely a multiple sclerosis lesion



Side of PICA lesion
Thermo-algesic
anesthesia of the
hemiface (and the
body of the
opposite side)
Claude B. Horner
Vestibular S.
Cerebellar S.
/2 larynx,
/2 pharynx,
vocal cord
and velum paresis

WALLENBERG SYNDROME

"LATERAL MEDULLARY SYNDROME" OR
"POSTERIOR INFERIOR CEREBELLAR ARTERY (PICA) SYNDROME"



Dizziness - Pathological processes

Visual system: Cataract, glaucome, stroke, diabetes...

refer to Pr. Rakic's presentation.

Cerebellum: more frequent

- Acute and/or chronic alcohol consumption
- Vitamine B1 or B12 deficiency
- TIA or stroke and hypoperfusion situation involving vertebrobasilar blood flow → cerebellar infarction, posterior lateral medullary infarction (Wallenberg),
- Leucoariosis, and Biswanger disease
- Tumor or paraneoplastic syndrome
- Drugs: benzodiapines, antihistaminic, neuroleptic, morphinic, ...
- Chimiotherapy

Cerebellum: less frequent

- Hypothyroid
- Neurodegenerative disorders: multiple systemic atrophy, Friedreich
- Coeliac disease
- Infectious disease : Toxoplasm, Whipple disease
- Multiple sclerosis

Proprioceptive system:

More frequent → diabetes, chronic alcohol consumption,

Neoplam, Severe Hypothyroid, Renal deficiency, Chronic hepatitis,

Neurotoxic drugs as some chimiotherapy or antibiotics or antimalarian and antiretrovial treatment

e.i; vincristine, cisplatine, vinblastine, doxyrobutine, isoniazide, métronidazole, éthambutol, nitrofurantoïne, colistine, dapsone,

Neurotoxic agent exposure: lead, mercury

Dizziness - Content

Definition and epidemiology

Clinical consequences

Physiopathology

Etiology and clinical presentations

Evaluation

Management

Take home messages

Dizziness - Anamnesis

Whether attacks are episodic or continuous

Frequency and duration

Precipitating factors

 Alcohol, medications, changing head or neck position, standing from lying, bending forward to pick something up, meals

Symptoms associated with

- Tinnitus, fullness in ear, fluctuating hearing loss, nausea, vomiting (Menière, Neurinome)
- Double vision, dysarthria, sudden blackout (vertebro-basilar)
- Sensitive signs: dysesthesia, pain,
- Motor signs: weakness, loss of strength

Dizziness - Anamnesis

Comorbidities

- Cardio-vascular: ischemia, embolism, rythmic, arteriopathy, valvulopathy
- Diabetes
- Anxiety
- Anemia
- Dementia
- Hypothyroid
- Neoplams and treatment
- Infectious / Systemic disease

Dizziness - Physical exam

Spontaneous walk and turn

Motricity and reflexes

Tonicity, Parkinsonism, Tremor

Wide-based stance and Romberg test

Cerebellum testing: finger-nose testing, dysarthria, voice, walk on a line

Fukuda stepping test

Proprioceptive, Vibration, Thermo-algesic and Tactile sensitivity

Dizziness - Physical exam

Cranial nerves testing

- Especially 2; 3,4,6; 7; 8 (vision, occulomotricity, audition, facial motor/sensitive)
- Corneal reflex (+ unilateral hearing loss -> acoustic neuroma)

Otoscopic examination, Dix-Hallpike maneuver

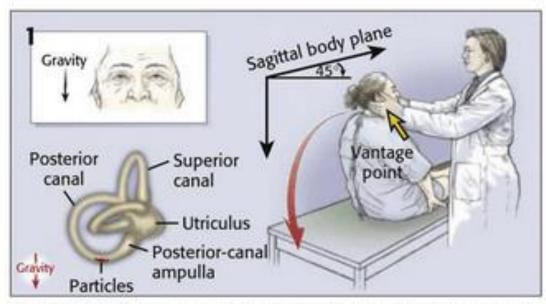
Neck: local tenderness or restriction in the range of movement

Congestive heart failure clinical signs: oedema

Carotid or heart murmur

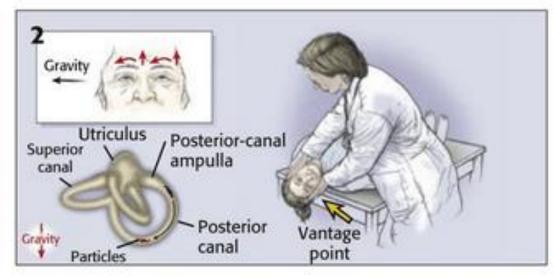
Peripheral vascularisation

DIX-HALLPIKE MANEUVER



The examiner stands at the patient's head, 45° to the right, to align the right posterior semicircular canal with the sagittal plane of the body.

The hear posted toward the ground is tested (the right hear in this picture). In case of Benign Positionel Paroxystic Vertigo \rightarrow vertigo + nystagmus \rightarrow vertical, latency, short, fatigability, and inversion when the patient recovers sit position.



The examiner moves the patient, whose eyes are open, from the seated to the supine, right-ear-down position and then extends the patient's neck slightly so that the chin is pointed slightly upward. The latency, duration, and direction of nystagmus, if present, and the latency and duration of vertigo, if present, should be noted. *Inset:* The arrows over the eyes depict the direction of nystagmus in patients with typical BPPV. The presumed location in the labyrinth of the free-floating debris thought to cause the disorder is also shown.



Dizziness - Parameters

Blood pressure,
Heart rate
Orthostatic hypotension test
Glycemia

Dizziness - Investigations

Depending on

- medical history, anamnesis, physical exam
- supposed diagnosis
- patient's procare plan

- → « How this exam will be helpfull to take care of this patient? »
- → « Is the treatment would be modified by the result of this Test? »

Dizziness – Management

Dizziness could be secondary to several causes

Most often causes are not modifiable

Goal-oriented management

- → Apply disease-specific treatment
- → Identify the modifiable causing or contributing factors
- Avoid negative clinical consequences

Dizziness - Management

- → GOAL = apply disease-specific treatment
- Surgery and/or stereotaxy in case of neurinome (if in line with patient care plan)
- Vestibular rehabilitation
- Exercices combining movement of eyes, head and body

Dizziness - Management

- → GOAL = Identify the modifiable causing or contributing factors
- > Anemia, metabolic disorders, vit B12 deficiency, thyroid abnormalities
- Correction of vision and hearing deficit
- ➤ Anxiety have to be considered as a cause and also a consequence of the dizziness → dilemma to manage anxiety with drugs able to cause dizziness → wich is the main cause of anxiety → wich could be the most adapted care

Dizziness – Management

→ GOAL = Avoiding negative clinical outcomes

Comprehensive Geriatric Assessment

Including nutrition, cognitive, thymic, mobilty assessment

Fall prevention program

Including Physical therapy, behavioural and environemental review

Fall consequences prevention program

vitD, calcium, osteodensitometry if needed and if estimated survival > 1 year.

Dizziness - Take Home Messages

Frequent with several causes

- → numerous **negative clinical consequences**,
- → Dizziness have to be **systematically screened** by anamnesis

Investigation, based on anamnesis and physical exam, has to

- → Deal with the patient plan care and wishes
- → Discuss target disease treatment if indicated
- → Identify the modifiable causing or contributing factors
 - → Avoid negative clinical outcomes

Syncopa -Content

Definition and epidemiology

Clinical outcomes

Physiopathology

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Take home messages

Syncope - Definition and epidemiology

« Transient loss of consciousness due to transient global cerebral hypoperfusion »

rapid onset, short duration, spontaneous and complete recovery

Experienced by up to 30 % of healthy adults at least once in their lifetime.

The seventh most common reason for emergency admission of patients over
 65 years.

Mortality depends on etiology, congestive heart failure history, male sex and clinical consequences (brain injury, hip fracture...)

Syncope – Clinical consequences

- > Falls
- > Injuries related to falls
- > Loss of self-esteem, self-confidence, QOL
- > Hospitalisation and negative clinical/functional consequences
- Institutionalisation

Syncope - Physiopathology and etiology

Age related physiological changes

Reduced baroreflex sensitivuty, reduced blood flow, reduced blood volume

Atherosclerosis

Specific disorders

- orthostatic hypotension, postprandial hypotension,
- vasovagal syncopa and carotid sinus hypersensitivity
- cardiac and cerebro-vascular syncope

Aspecific contributing factors

- Anemia, chronic lung disease, congestive heart failure, dehydratation,
- Long standing, hot weather, alcohol, prolonged recumbency, large meals, ...

Syncope – Specific disorders

- Orthostatic hypotension
- ➤ Postprandial hypotension
- > Reflex syncopal syndromes
 - > Vasovagal syncopa and carotid sinus hypernsitivity
- ➤ Cardiac syncopa
- ➤ Cerebrovascular syncopa

Orthostatic hypotension

Mechanism: Physiological responses are overloaded, not sufficient to maintain a minimal cerebral blood flow

Diagnostic criteria:

- ➤ A decrease ≥ 20 mm of systolic blood pressure
- >OR a decrease ≥ 10 mm of diastoling blood pressure
- Mesured 2- 3 minutes after standing up.
- > Measure heart beat are not necessary but recommended
 - ➤ Bradycardics or autonomic sytem disorder

Orthostatic hypotension - Causes

- Not only age-related
- Medications
- >Autonomic failure.
 - >Primary: multiple system atrophy, parkinson disease or pure autonomic failure
 - Secondary: diabetic neuropathy or amyloid neuropathy

▶ Volume depletion

hemorrage, diarrhea, febrile illness, hot weather, extensive burn, third sector, mineralocorticoids deficiency

Orthostatic hypotension - Management

- > Medications review: diuretics, vasodilators, antihypertensives, morphinics
- > Avoid potential situation increasing reduced blood flow and correct anemia
- ➤ Secure the standing up
- ➤ If no lower limbs occlusive arteriopathy → compression stocks
- ➤ (Fludrocortisone): few EBM data available. Consider 0.1 mg 1-3/day by patients with Parkinson or severe diabète (Cochrane review). Pay attention to oedema and HTA (→ reduce à 0.05 mg 1-3/day)
- (Midodrine): = a + , but several contraindication including HTA, cardiomyopathy, artériopathy, retinopathy, glaucoma

Postprandial hypotension

Mechanism:

- An increase splanchnic and superior mesentery artery blood flow and a rise in plasma insulin level
- Without corresponding rises in sympathetic nervous system activity

Clinical presentation: similar to orthostatic hypotenion with and a temporal relationship with the meal and without relation with orthostatisme.

Management:

- Small and frequent meals including complex carbonhydrates
- -Review of medications
- -Avoidance reduced blood flow situations

Syncopa - Reflex syncopal syndromes

- Carotide sinus syncopa
- Vasovagal syncopa secondary to
 - Acute Pain: visceral pain, trigeminal neuralgia
 - Cough
 - Defecation
 - Micturition
 - Intensive exercice
 - Anxiety

Vasovagal syncope - Mechanism

Hypothese → abnormal Bezold-Jarish reflex.

In case of arterial pressure variability or decreased venous return as well in case of visual or nociceptive stimuli \rightarrow abnormal Bezold-Jarish reflex

- → an abnormal vagal stimulus leading to a bradycardia AND a decrease in sympathetic vessels tonicity.
- \rightarrow bradycardia and a blood pressure decrease \rightarrow hypotension and/or bradycardia \rightarrow sudden decrease of the cerebral blood flow.

Vasovagal syncope – Clinical presentation

A prodrome or aura / loss of consciousness / postsyncopal phases

<u>Precipitatnig factor</u>: extreme emotional stress, anxiety, physical pain, warm, environnement, air travel, prolonged standing

<u>Prodrome</u>: weakness, nausea, visual defects, dizziness, visual or auditory hallucinations, dysarthria or paresthesias. Partial recall of prodromal period.

Syncopal period: brief, myoclonic jerk, or myoclonic movement.

Recovery is rapid (+/-)dizziness, nausea, headache, confusion, general sense of ill health

Vasovagal syncope - Diagnostic

Anamnesis and heteroanamnesis

Valsalva manœuvre:

- symptoms are reproduced and/or,
- decrease in blood pressure > 50 mm Hg or less than 90 mm Hg and/or,
- asystole > 3 secondes or heart rate < 40 beats/min for at least 10 secondes.

Tilt test

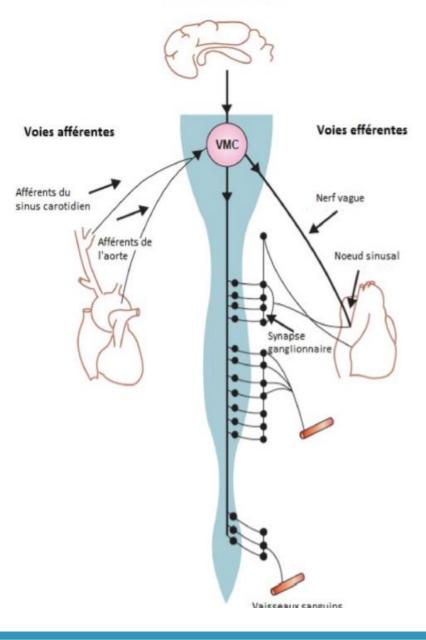
One-week external loop recording

(Implantable loop recorder)

Vasovagal syncope - Management

- > Review medications: diuretics, vasodilators, antihypertensives, morphinics...
- > Avoid potential situation reducing blood flow
- Correct anemia, dehydratation, any circumtances with volume depletion, ...
- ➤ If no occlusive arteriopathy, → compression stocks
- ➤ If at least three seconds asystole → permanent cardiac pacing

Centre cortico-thalamique

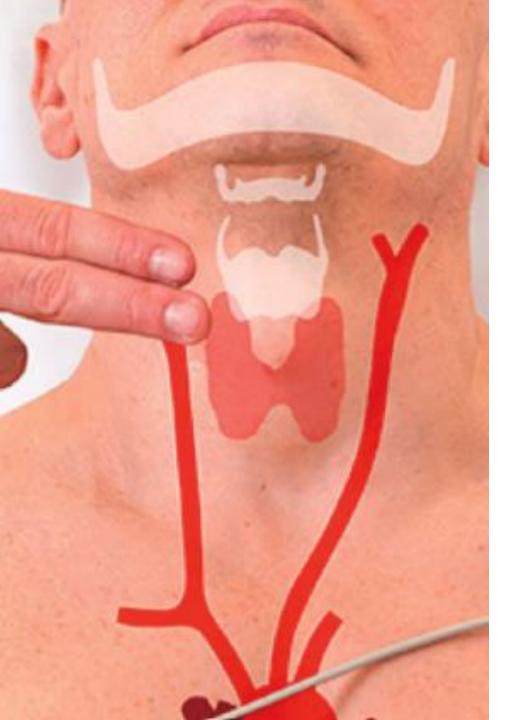


The carotid sinus syndrome or carotid sinus hypersentivity

Mechanism: An episodic bradycardia and/or hypotension resulting from exagereted baro-receptor-mediated reflexes secondary to a mecanical stimulation of the carotid sinus such as head turning, tight neckwear, neck pathology or vagal stimuli.

Carotid sinus hypersensitivity is dived in three forms:

- the cardio-inhibition form wich is defined by an **at least three seconds asystole** (without PA decline),
- the vasopressive form defined by **a decrease of at least 50 mm Hg** (without asystole)
- the **mixt form** combining an asystole with a decline in arterial pression.



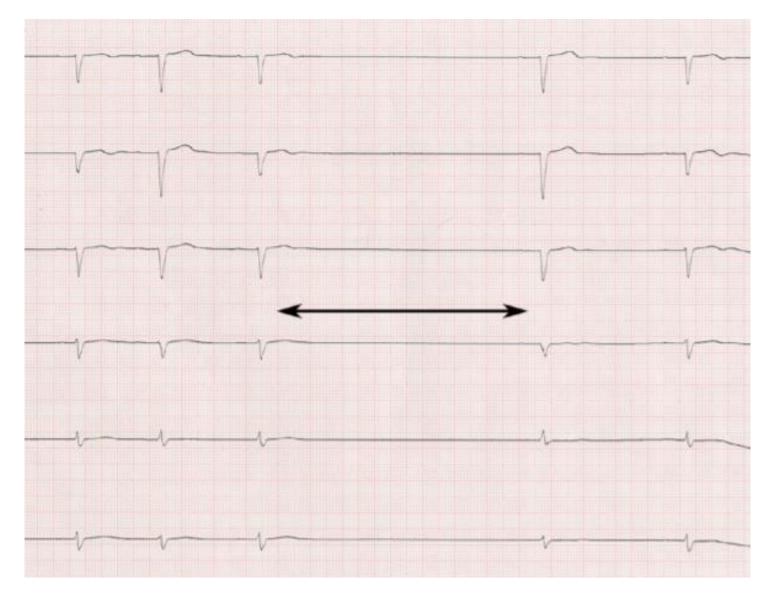
The carotid sinus syndrome or carotid sinus hypersentivity

Diagnosis: Anamnesis and heteroanamnesis

Carotid sinus massage with a cardiac monitoring and physician able to manage severe bradycardia. The duration of carotid sinus massage is from 5 to 10 secondes.

Complications include cardiac arythmia and neurologic disorders

Carotid sinus should not be performed in patients who have had a recent cerebrovascular event or myocardial infarction (3 months), patient with a carotid stenosis history or patient who have a carotid murmur (American Heart Association). Caution should be take in case of ventricular arythmia.



The carotid sinus syndrome or carotid sinus hypersentivity

Dual-chamber cardiac pacing is the treatment of choice in patients with symptomatic cardioinhibitory carotid sinus syndrome.

Asymptomatic patient with hypersensitivity mustn't be treated by pacing.

Cardiac syncope

Frequent among older adults

Syncope caused by either cardiac disorders (rythmic or ischemic or metabolic disorders) either mixed cardio-respiratory disorders.

Prodrome: palpitation, chest pain when supine or during exercises, dyspnea, dizziness, pre-syncopal feelings. Sometimes absent.

Causes: ischemic, rhythmic, vavulopathy, metabolic disorders

Cardiac syncope

Diagnosis: anamnesis and heteroanamnesis;

Electrocardiogram > conduction ? Ischemia ? long-lasting QT ? T waves?

One-week external cardiac rhythm recording \rightarrow asystole > 3 seconds, rapide supraventricular or ventricular tachycardia;

An echocardiography;

An exercise stress test;

An electrophysiologic study.

Cardiac syncope, ESC recommendations

Recommendations of the European Society of Cardiology

- Mecanisms
- Classification
- Algorithm of investigation and management
- → https://www.heartrhythmalliance.org/files/files/stars/180320-dm-2018%20Syncope%20Guidelines.pdf

Cardiac Syncope and medications

As reminder,

You can find there a list → medications → a long QT syndrome

DCI	Nama da antistalità
DCI	Noms de spécialité
Amiodarone	Cordarone®, Corbionax®
Amisulpride	Solian®
Arsenic	Trisénox®
Bépridil	Unicordium®
Chlorpromazine	Largactil®
Clarithromycine	Naxy®, Mononaxy®, Zéclar®, Monozéclar®
Cyamémazine	Tercian®
Disopyramide	Rythmodan®, Isorythm®
Dolasétron	Anzemet®
Dropéridol	Droleptan®
Ebastine	Kestin®, Kestin Lyo®
Érythromycine	
Fluphénazine	Modécate®, Moditen®
Halofantrine	Halfan®
Halopéridol	Haldol®
Indapamide	Preterax®, Fludex®, Bipreterax®
Lévofloxacine	Tavanic®
Lévomépromazine	Nozinan®
Méthadone	
Mizolastine	Mizollen®
Moxifloxacine	Izilox®
Penfluridol	Sémap®
Pentamidine	Pentacarinat®
Perphénazine	Trilfan®
Pimozide	Orap®
Pipampérone	Dipipéron®
Pipotiazine	Piportril®
Propériciazine	Neuleptil®
(hydro)quinidine	Sérécor®
Sotalol	Sotalex®
Spiramycine	Rovamycine®
Sulpiride	Dogmatil®, Synédil®
Sultopride	Barnéti®
Tiapride	Tiapridal®
Voriconazole	Vfend®

Cardiac syncope - Causes

Syncope secondary to a cardiopathy or a cardio-pulmonary disease include

Myocardial ischemic lesion, Valvulopathy (AO stenosis), Pulmonary embolism

Auriclar myxoma or obstructive myocardiopathy , pericarditis and cardiac tamponade

Diagnose: Myocardial enzymes, ECG, Echocardiography, Chest CT angiography

Management: Specific to the cause and according the patient profil

Cerebro-vascular syncope

Causes: Aortic/carotid dissection,

Subclavian derivation,

Transient ischemia, stroke,

Thrombo-embolic events

Diagnose: MRI with gadolinium / CT scan with contrast

Investigation and management are disease-specific

Syncope - A challenge for clinician

As for dizziness, the anamnesis and physical exam are crucial

Investigation and management have to

- Deal with the patient's profile
- Discuss target-disease treatment if indicated
- Identify the modifiable causing or contributing factors
- Avoid negative clinical consequences

Syncope - A challenge for clinician

Management should include

- Fear of falling
- Walk and balance abilities
- Physiotherapy
- Environnement
- Medications review: especially platelet aggregation inhibitors, anticoagulation therapy, sedative, diuretics....
- Ability to drive should be consider.
- Minimal screening CGA -> and more if needed

Take Home Messages

Syncope and dizziness are **frequent**, with potential **negative clinical consequences**.

Both should be **systematically screened**.

Investigation and management should be based on your hypotheses and the patient's profile.

Both are real time-consuming challenge for clinician.

However, both are opportunity to diagnose something which could be curable.

In all cases, don't forget to prevent negative clinical consequences

and to plan a comprehensive geriatric assessment.

Scientific supports used preparing this lecture

Hazzard's Geriatric Medicine and Gerontology, seventh edition, 2017

Oxford textbook of Geriatric Medicine, third edition, 2018

Handbook of Clinical Neurology, Vol. 137 (3rd series), 2016

Massage du sinus carotidien Carotid Sinus Massage, Ann. Fr. Med. Urgence (2018) 8:383-389

Pertes de connaissance brèves de l'adulte : prise en charge diagnostique et thérapeutique des syncopes, Haute Autorité de Santé, HAS, Mai 2008.

https://www.cochrane.org/fr/CD012868/NEUROMUSC_fludrocortisone-dans-le-traitement-de-lhypotension-orthostatique

https://www.heartrhythmalliance.org/files/files/stars/180320-dm-2018%20Syncope%20Guidelines.pdf