Welcome - The agenda

9h-10h30: Vision disorders, Dr. Anouk GEORGES, from U Liège
10h30-10h45: Break

10h45- 12h15: Sleep disorders, Pr. An MARIMAN, from U Gent

12h15-13h00: Lunch time (lunch not provided)

13h00-14h30: Dizziness and syncope, Pr. Sophie GILLAIN, from U Liège
14h30- 14h45: Break

14h45-16h15: Hearing disorders and vertigo, Pr. Philippe LEFEBVRE, from U Liège, CANCELLED

Lecturers

Dr. Anouk GEORGES

Ophtalmologist in CHU Liège

Publications list available at:

https://orbi.uliege.be/simple-search?query=Anouk+Georges&sort_by=issued_dt&order=desc&filter=author%3A%3Aauthority%3A%3Ap015061%7CGeorges%2C+Anouk

UNIVERSITEIT GENT

Lecturers

Professor An MARIMAN

Psychiatrist and somnologist,

Department of psychiatry, the center of integrative medicine and the center for neurophysiological monitoring of the Ghent University Hospital

Main research interests include

The role of sleep in chronic fatigue and pain syndromes, Insomnia, Sleep in sports,

Interaction between nocturia and sleep fragmentation.

Belgian Association for Sleep Research and Sleep Medicine.

Lecturers

Pr. Sophie GILLAIN

Geriatrician in CHU Liège,
Head of department

Filed of interest
Dementia
Walk and balance disorders
Falls risk detection /prevention

Publications list available at:
https://orbi.uliege.be/simple-search?query=Sophie+Gillain&sort_by=issued_dt&order=desc&filter=author%3A%3Aauthority%3A%3Ap009705%7CGillain%2C+Sophie

DIZZINESS and SYNCOPE

BIUCGM – January 2025

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;
- Education of patients and caregivers (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.

3 ♀ / 1 ♂

Most frequent identified causes are

→ **25% Vestibular vertigo** (podcast)

→ 75 % who are left

Benign positional paroxysmal vertigo (BPPV) (podcast)

Vestibular migraine, (podcast)

Comorbidity consequences, toxic effects, … on vestibular, proprioceptive, visual systems and on cerebellum

Menière's disease (the less frequent) (podcast)

Dizziness – Clinical Consequences

- Loss of confidence
- Fear of falling and potential reduced spontaneous mobility;
- Cautious gait;
- Falls and injuries;
- Mobility decline;
- Sarcopenia;
- Thymic disorders - behavioural disorders;
- Reduced appetite and energy incomes;
- Frailty

Dizziness – Clinical Consequences

Then clinical consequences of frailty should be added to this list

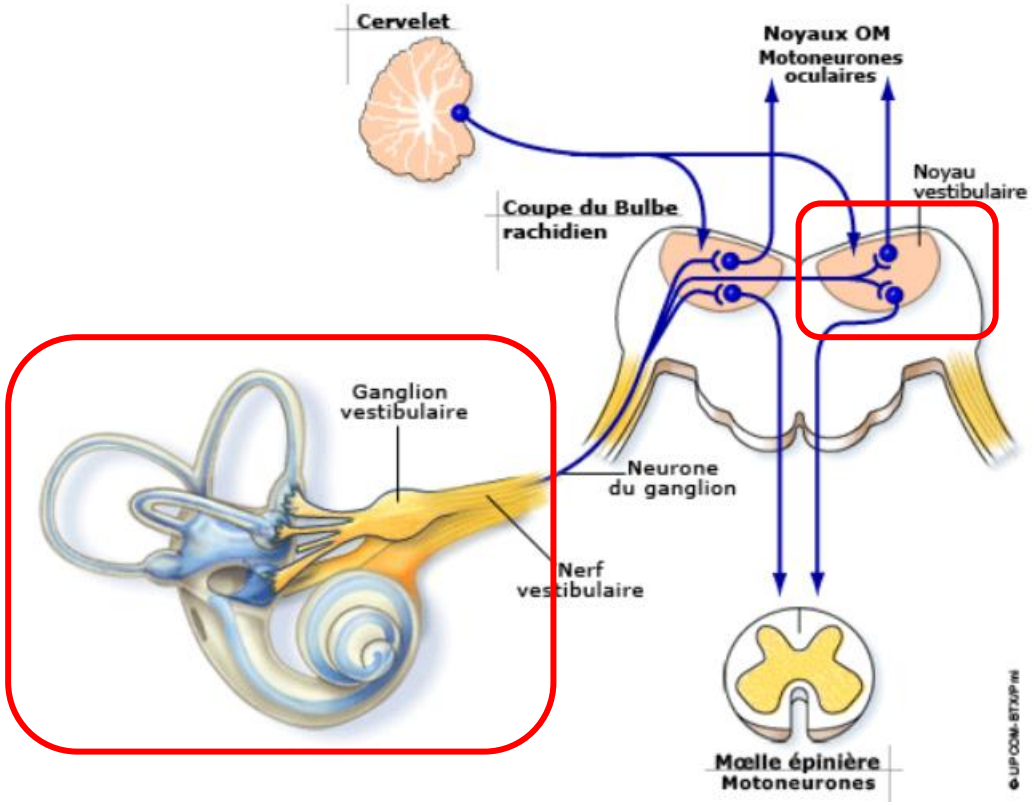
→ Disability

→ And finally, an increased risk of
- → Delirium
- → Hospitalisation
- → Longer inpatient stay after procedure
- → Institutionalisation
- → Death

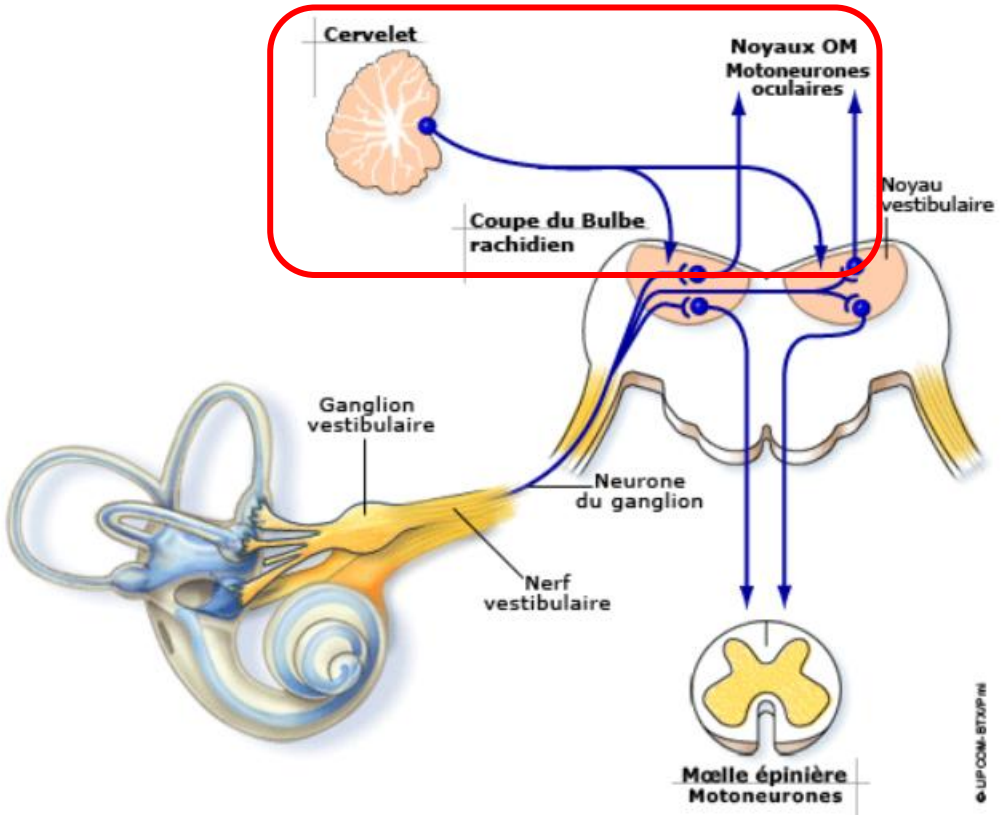
Dizziness - Content

- Definition and epidemiology
- Clinical consequences
- **Physiopathology**
- **Etiology and clinical presentations**
- Evaluation
- Management
- Take home messages

Vestibular and visual afferences, reflexes and integration



Vestibular and visual afferences, reflexes and integration



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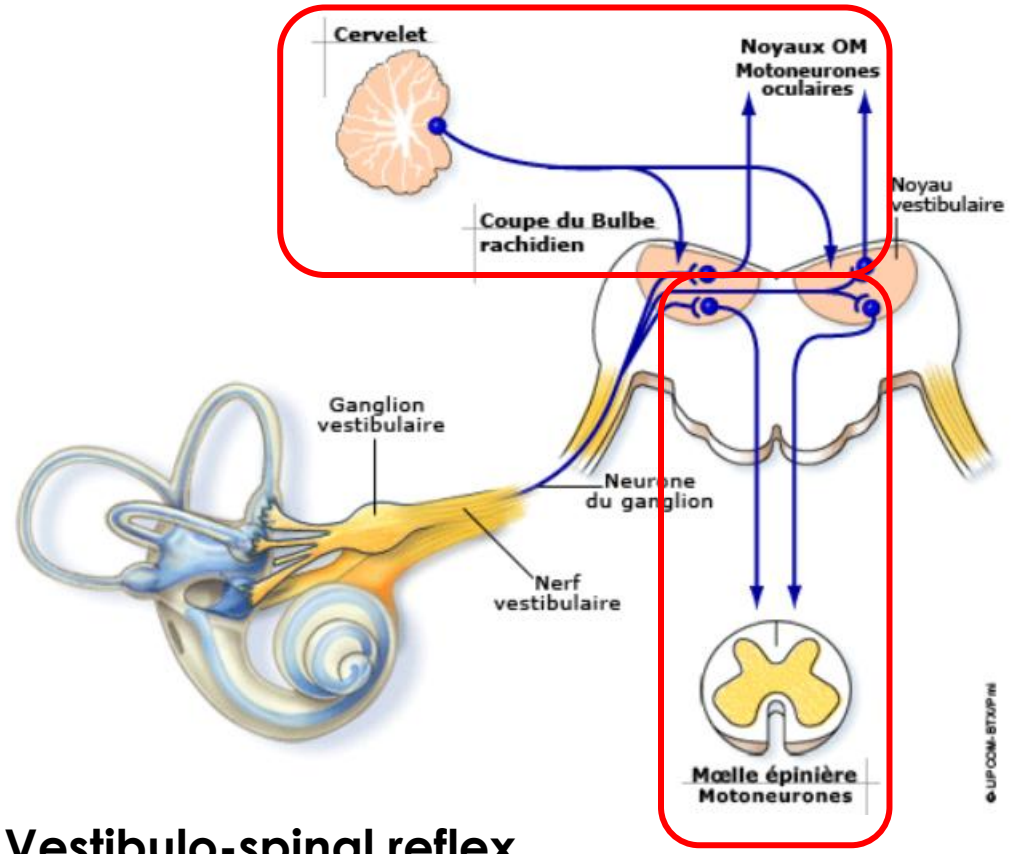
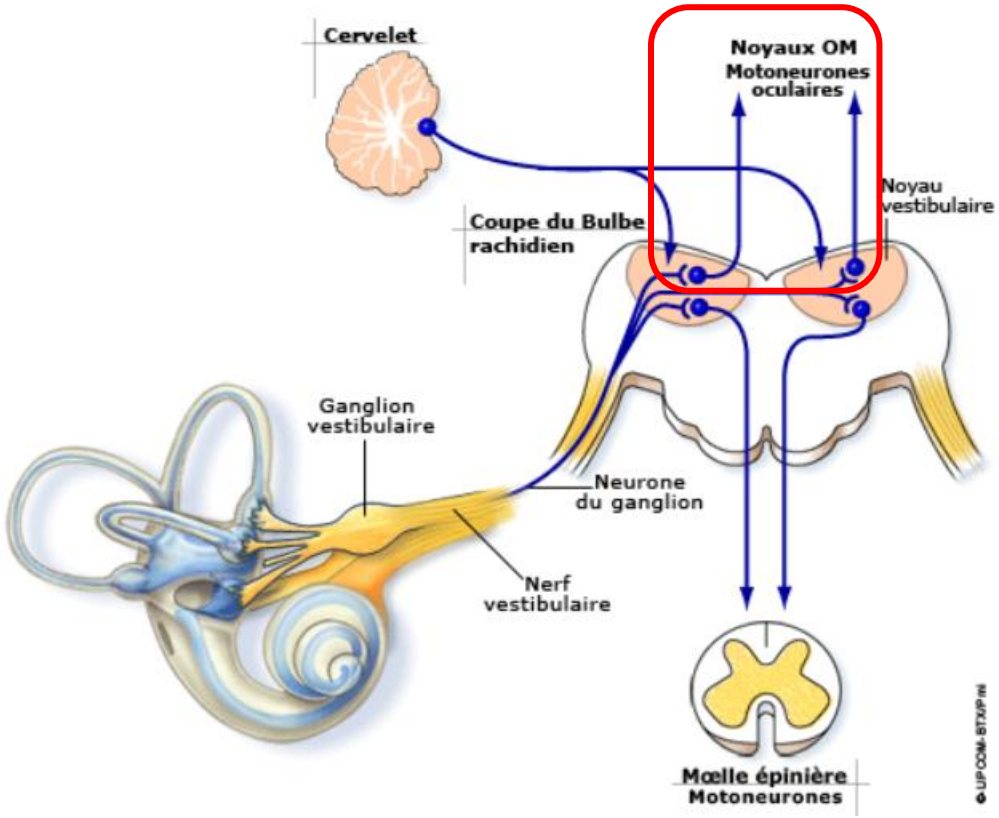


Vestibulo-spinal reflex

→ keep face and shoulder

→ anti-gravity muscles action

Vestibular and visual afferences, reflexes and integration



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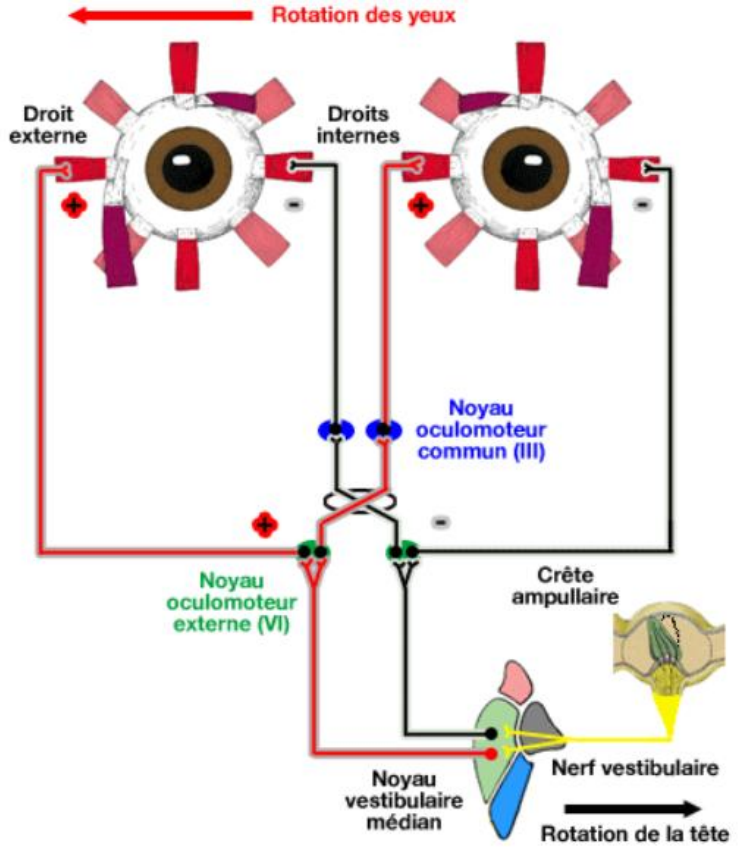


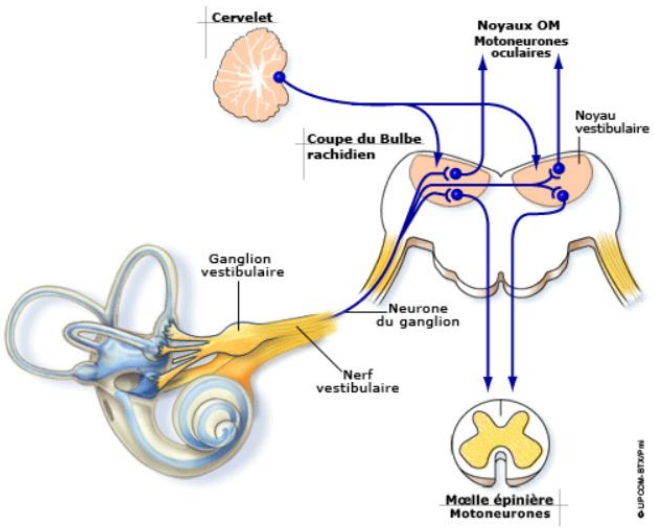
Vestibulo-occular reflex

→ keep eyes « focused on »

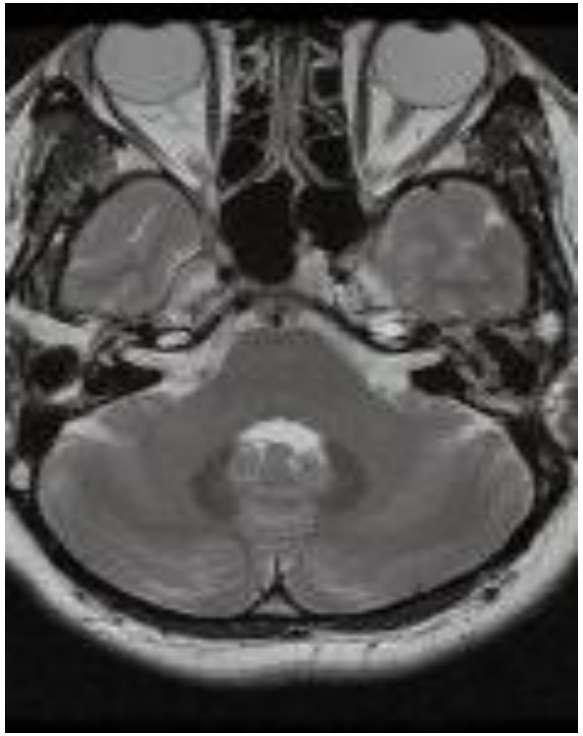
Vestibular system
Anamnesis / Clinical exam

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

Clinical exam :
Nystagmus
Lateralised Romberg
Ataxic walking
Lateralised 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

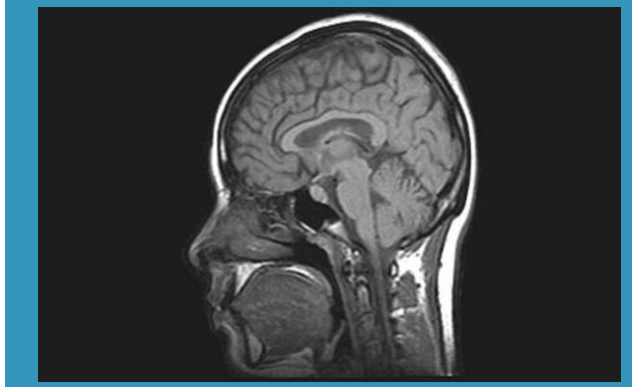
No vertigo,

Nystagmus could be present

Wide base support

Oscillations during Romberg

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

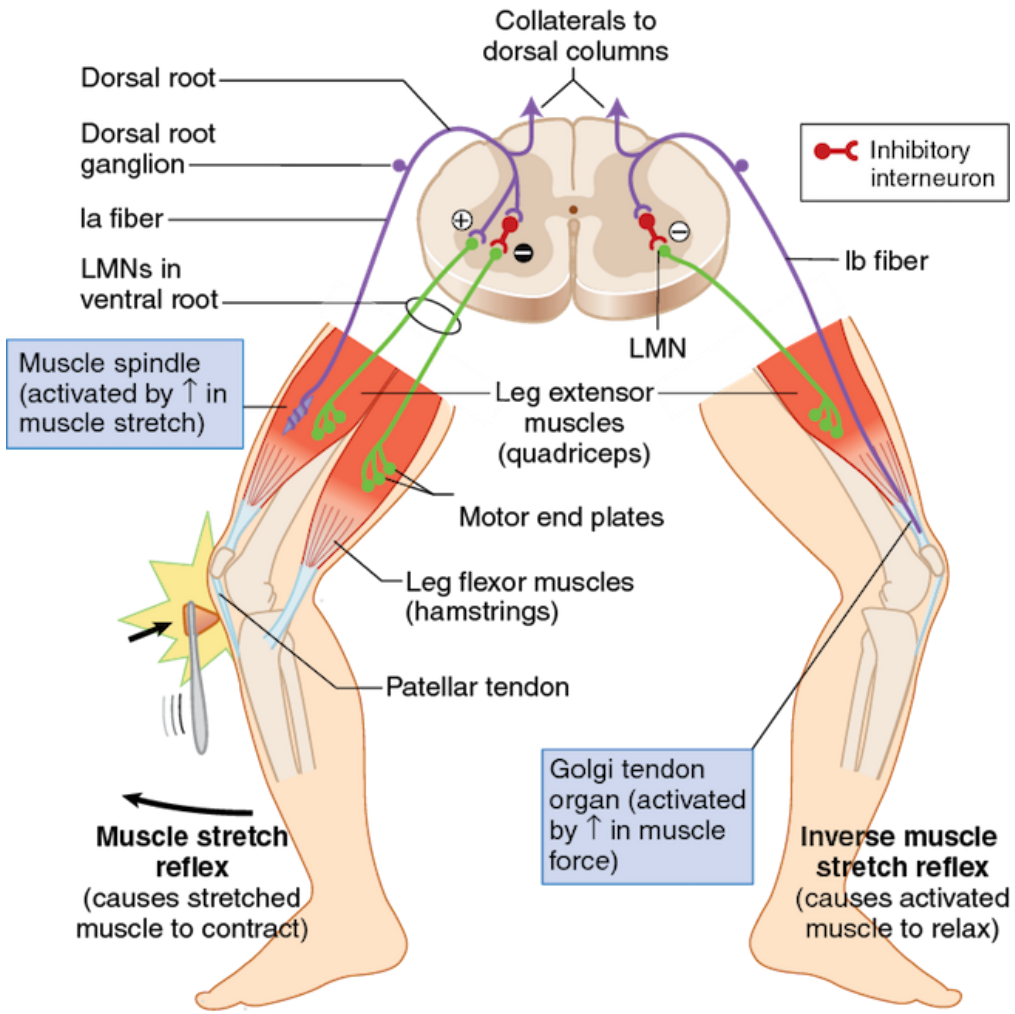


Vascular Lesion;

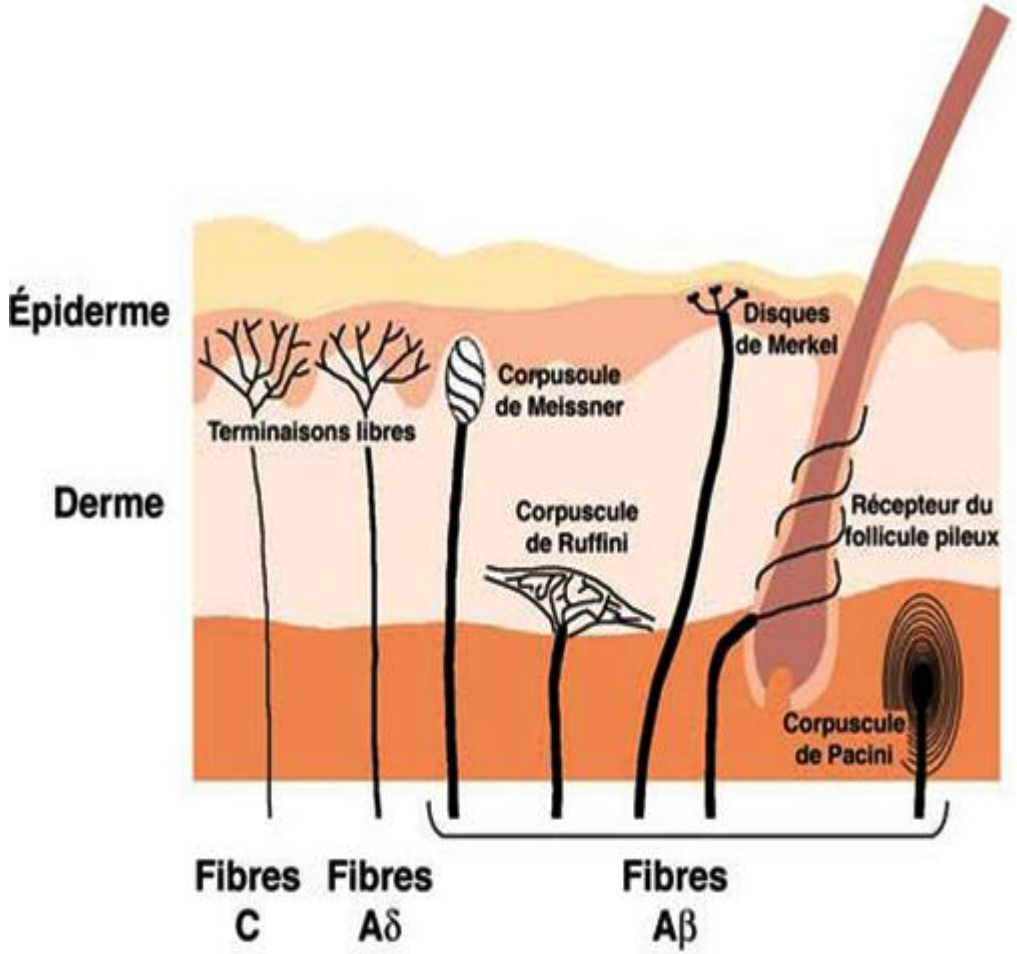
Meningioma, medulloblastoma, or glioblastoma;

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

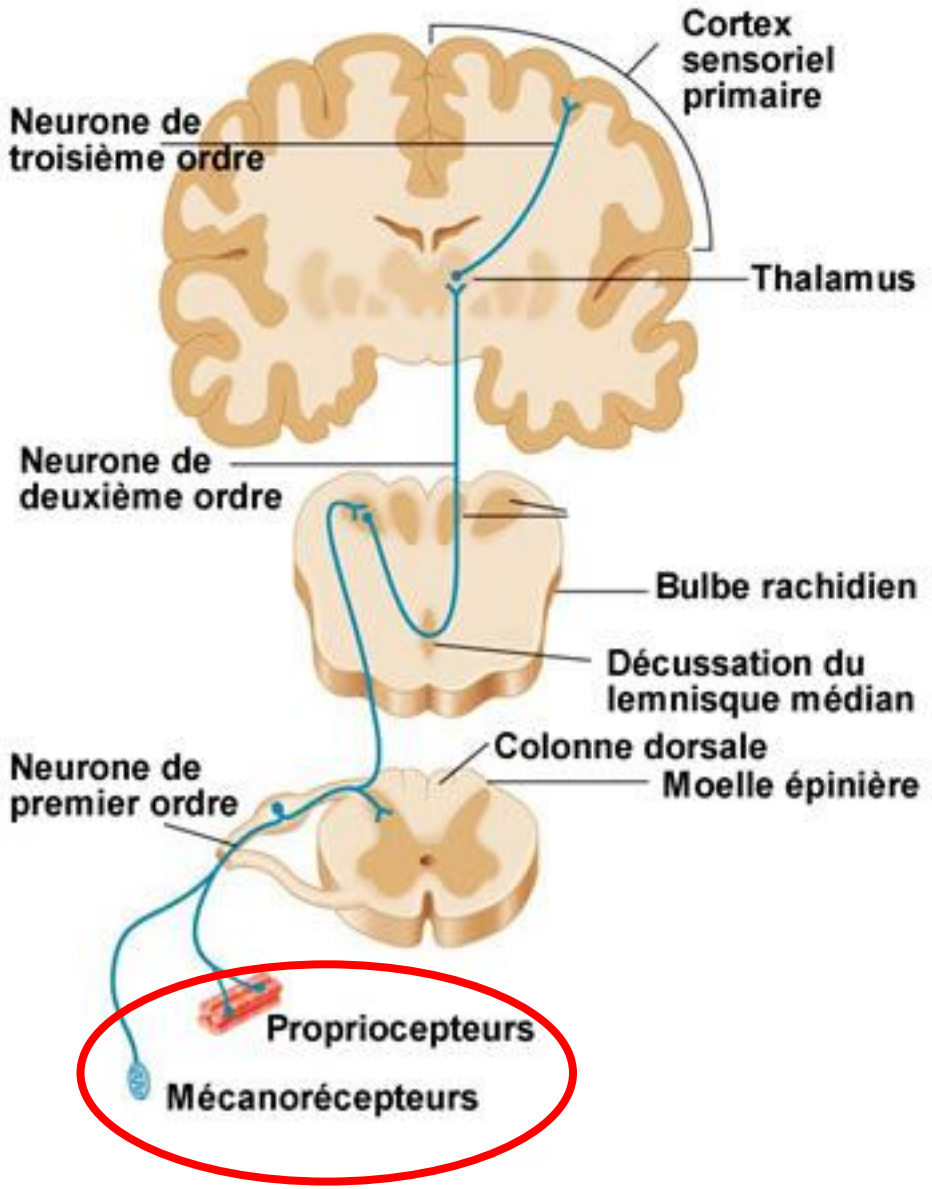
Proprioceptors

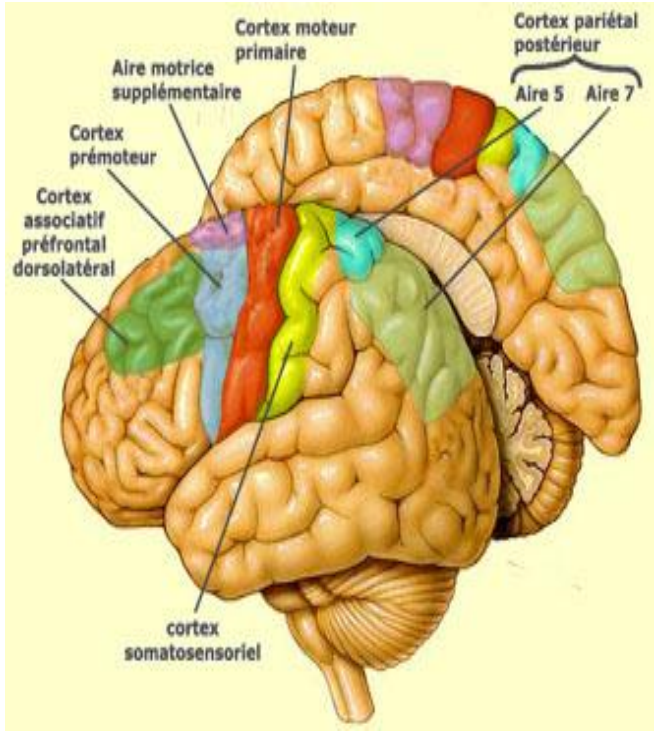


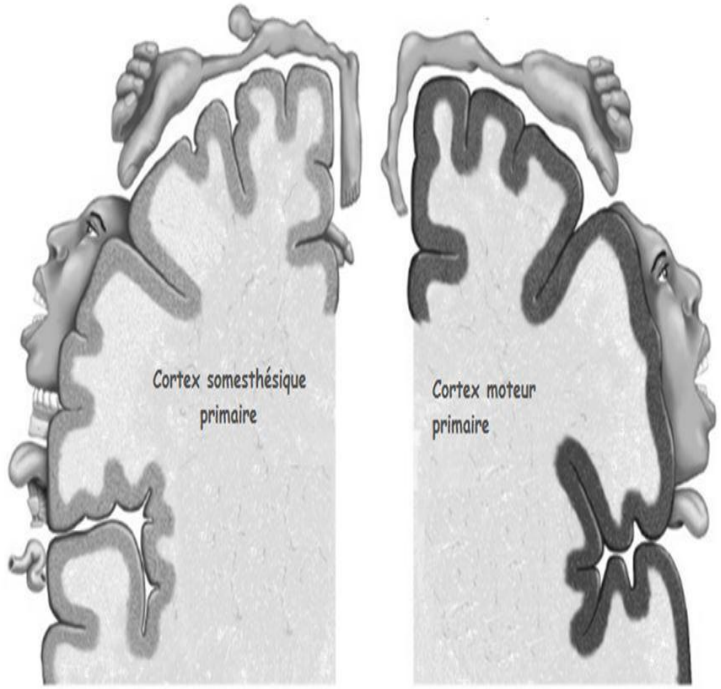
Mechanoreceptors



Sensitive Lemniscal Ascending Path







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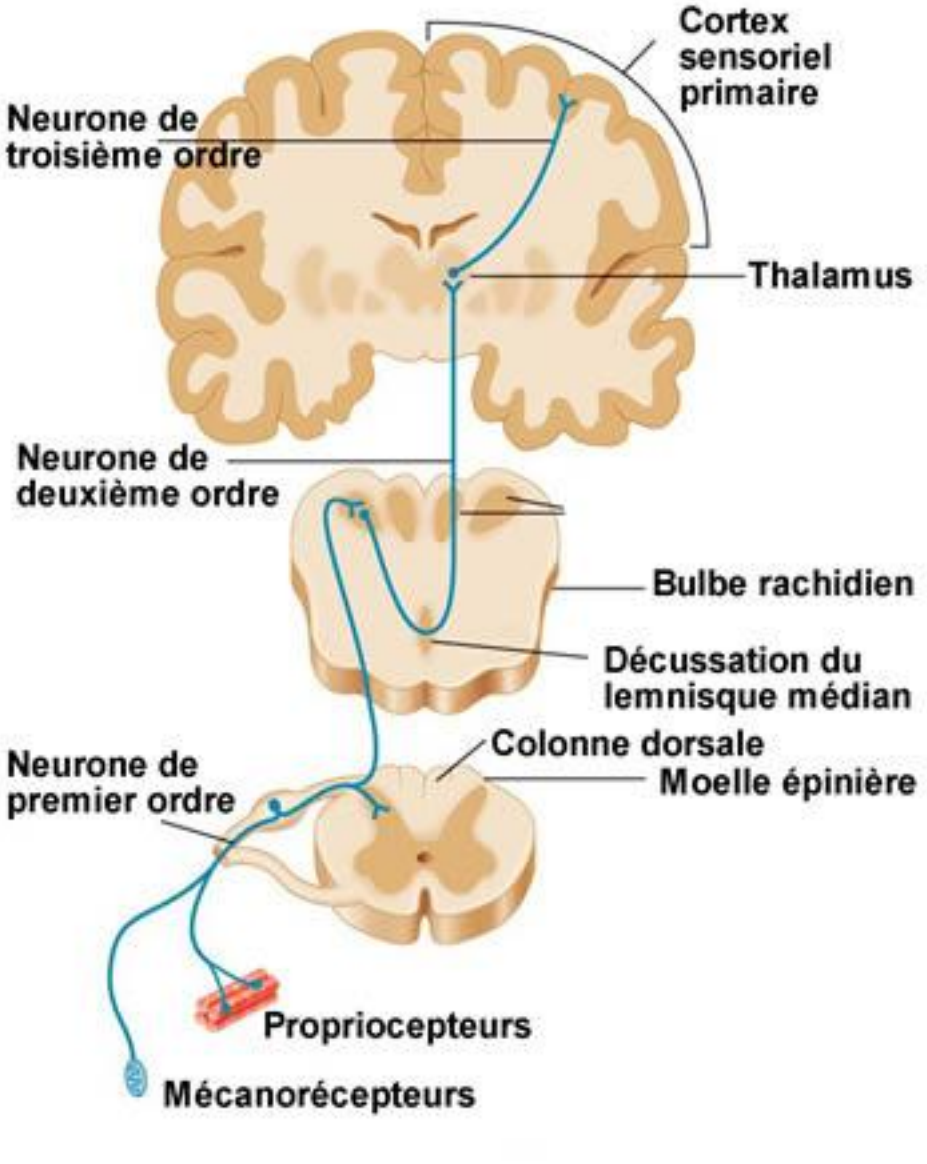
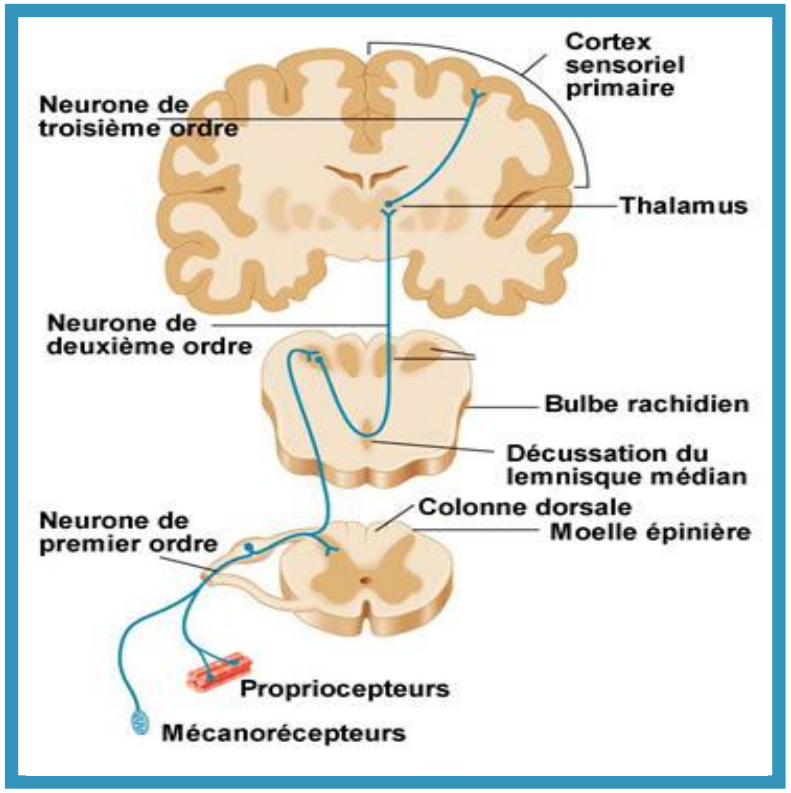


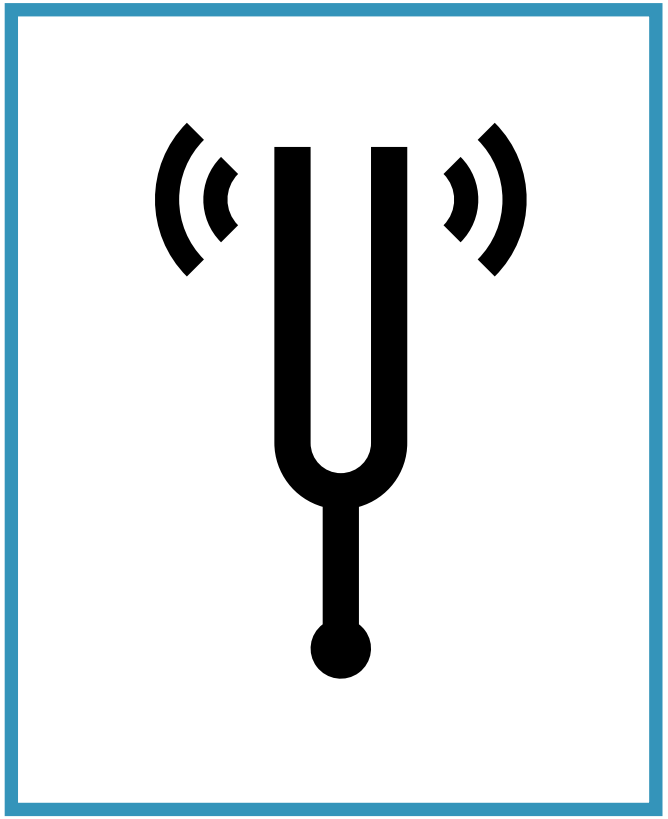
- The main cause of proprioception lesion is PNP
- 80 years ; 50 + have PNP signs on EMG
- Pain is the main complaint
- Hypoesthesia « in socks » is almost always present
- Ataxic walk is less frequently shown

Proprioceptive system Anamnesis / clinical exam

Symptoms : dizziness and instability

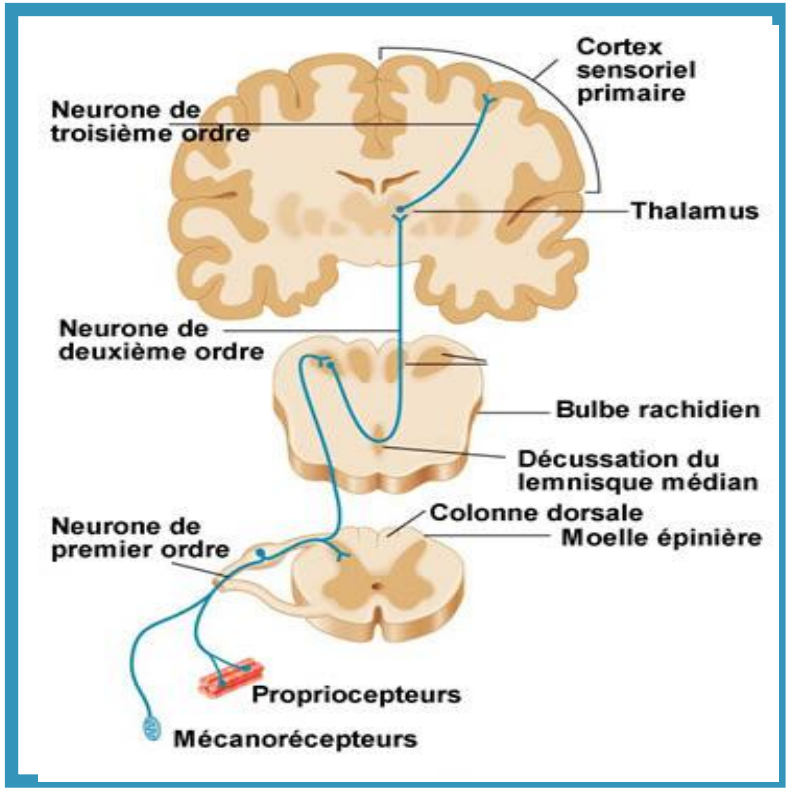
Clinical exam:
- little oscillations when the eyes are closed (Romberg)
- Patients need to look at their feet when asked to put them together. In order not to fall they need to put feet apart.
- Walk: wide base of support, irregular steps and lateral deviations
- Vibration sensitivity





Proprioception Investigations

Medical history: Diabetes, Chronic alcohol consumption, Cancer, Amyloid pathology, Hypothyroid, Renal deficiency, Chronic hepatitis, Neurotoxic molecules and, Syphilis and HIV



Vincristine, cisplatine, vinblastine, doxyrobutine;

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

Anti-malaria

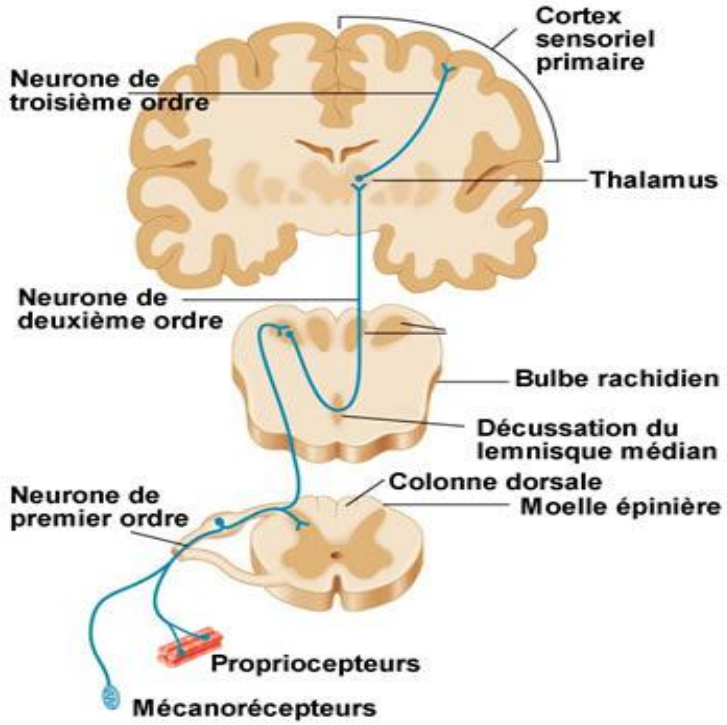
Antiretroviral

Lead and mercury

Proprioception Investigations

Medical history: Diabetes, Chronic alcohol consumption, Cancer, Amyloid pathology, Hypothyroid, Renal deficiency, Chronic hepatitis, Neurotoxic molecules and, Syphilis and HIV

Biological assessment



Thyroid tests T4 and TSH,
Proteins Electrophoresis,
Light chains immunoglobins detection in urine,
And serology

Vincristine, cisplatine, vinblastine, doxyrobutine;

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

Anti-malaria

Antiretroviral

Lead and mercury

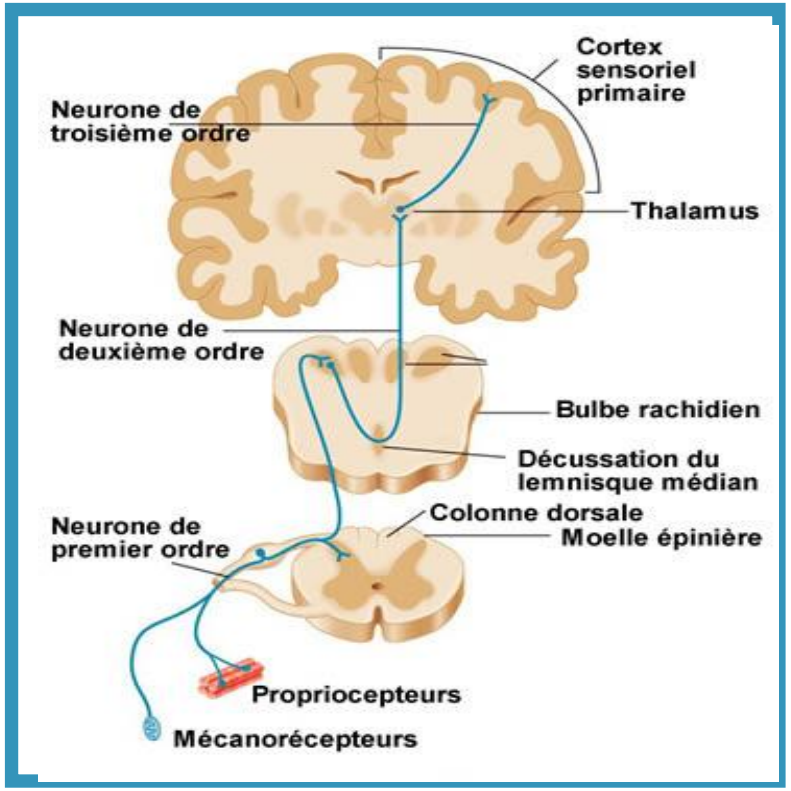
Proprioception Investigations

Medical history: Diabetes, Chronic alcohol consumption, Cancer, Amyloid pathology, Hypothyroid, Renal deficiency, Chronic hepatitis, Neurotoxic molecules and, Syphilis and HIV

Biological assessment

Electromyogram of lower limbs not systematic

Spinal MRI (rarely)



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Anti-malaria

Antiretroviral

Lead and mercury

Investigating proprioception

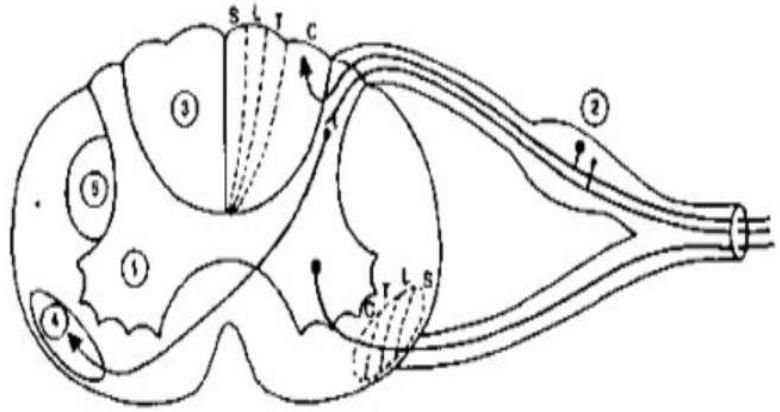
Electromyogram of lower limbs not systematic

Spinal MRI in case of spinal lesion suspected

As a reminder a compressive spinal lesion will be associated with

→ Lesional syndrome : radiculopathy

→ Sub-lesional 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

Proprioception Investigations

Medical history: Diabetes, Chronic alcohol consumption, Cancer, Amyloid pathology, Hypothyroid, Renal deficiency, Chronic hepatitis, Neurotoxic molecules and, Syphilis and HIV

Biological assessment

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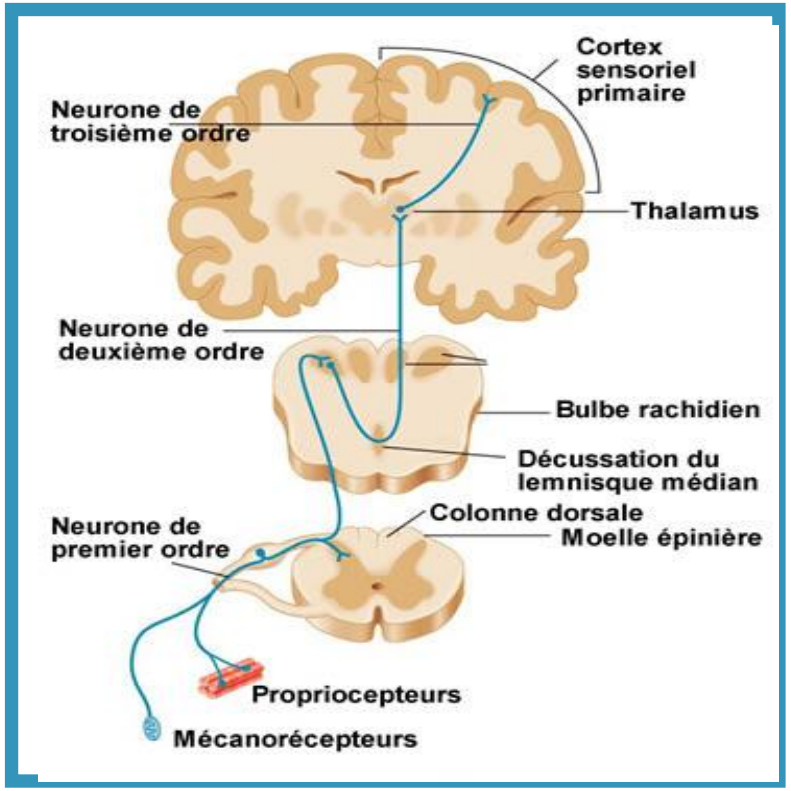


In 30% of cases the cause of polyneuropthy stays unknown

Vincristine, cisplatine, vinblastine, doxyrobutine;

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

Anti-malaria

Antiretroviral

Lead and mercury

Dizziness – Main mechanisms and causes

Either misinformation coming from vestibular/ Visual/ Proprioceptive systems

Either misintegration in cerebellum and hemispheres

→ Non adapted motor order

The 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 accommodation;

Proprioceptive system : Reduced mechanoreceptors sensitivity, slower peripheral conduction;

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

Pathological processes

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 experience a spontaneous resolution
- Viral vestibular/ labyrinthitis neuronitis,
- Drug toxicity (aminoglycosides)
- Head trauma

Pathological processes

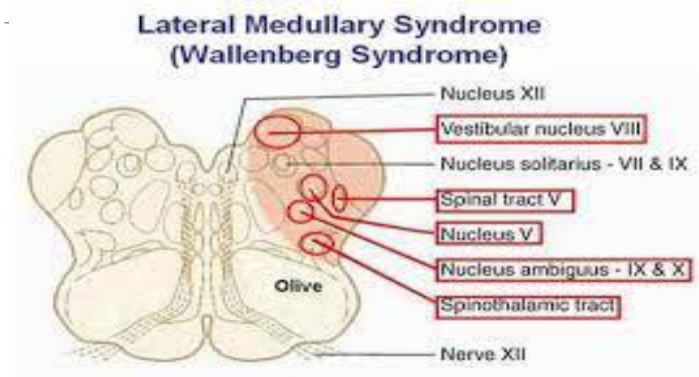
Vestibular system : central failure

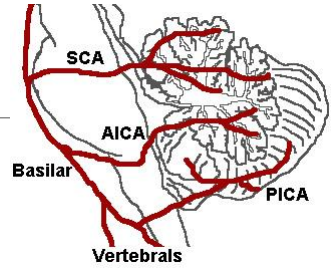
Most often : Tumor of brainstem

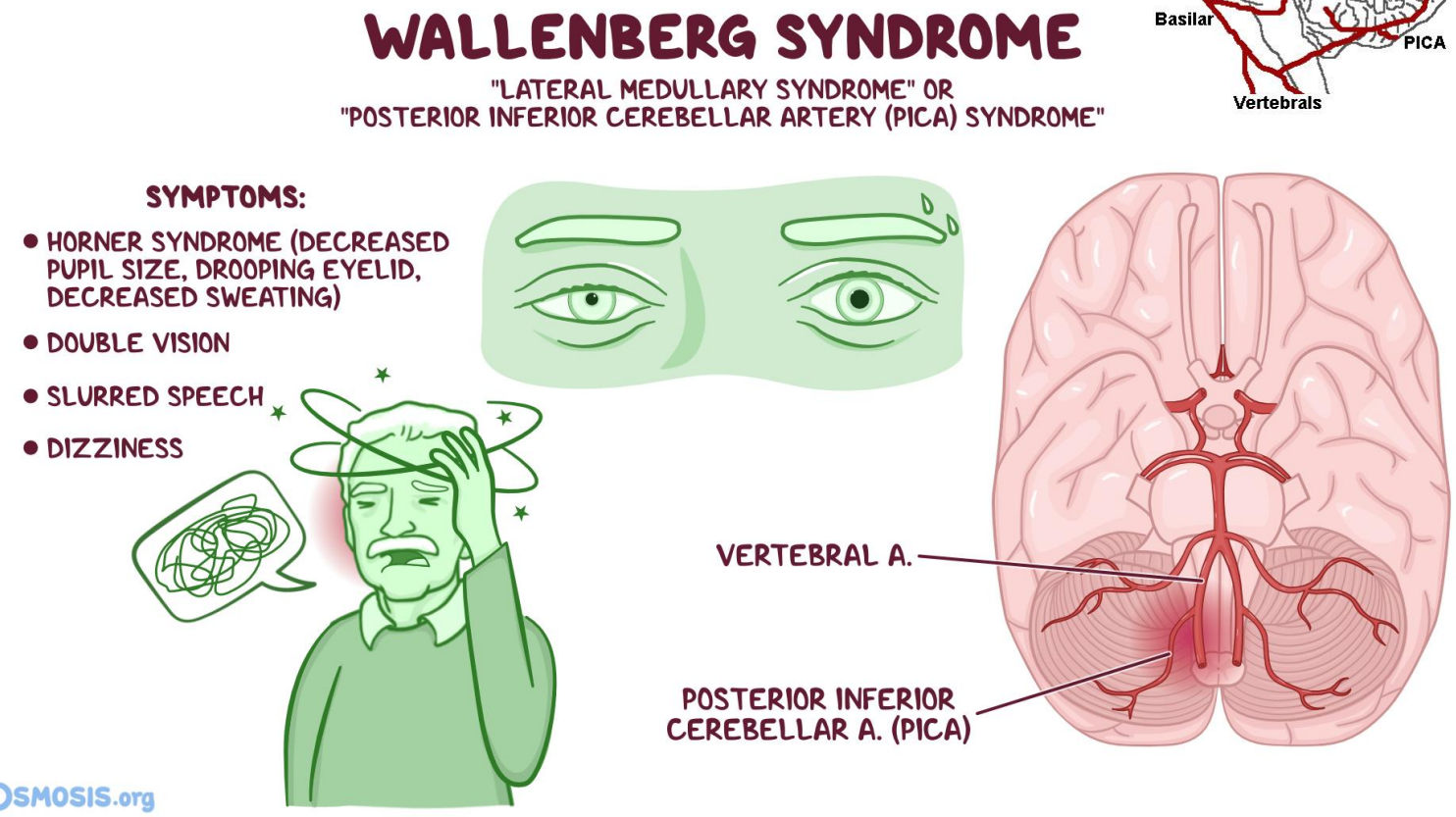
Wallenberg syndrome

Rarely a multiple sclerosis lesion

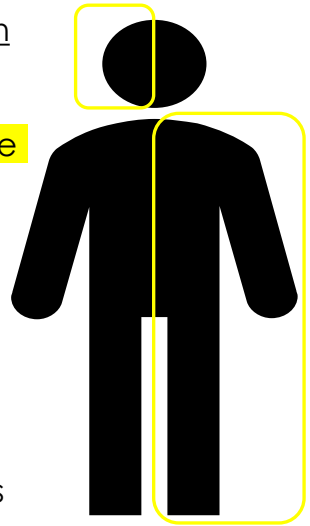
Pathological processes







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



Pathological processes

Visual system : Cataract, glaucoma, stroke, diabetes …

→ Please refer to Dr. Anouk GEORGES slides.

Pathological processes

Cerebellum : more frequent

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

Pathological processes

Cerebellum : less frequent

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

Pathological processes

Proprioceptive system :

More frequent → diabetes, chronic alcohol consumption,

Neoplam, Severe Hypothyroid, Renal deficiency, Chronic hepatitis,

Neurotoxic drugs as some chemiotherapy or antibiotics or antimalarian and antiretroviral treatment

- i.e.; 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, rhythmic, 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 (stepping in place → deviation's side shows vestibular lesion's side

Proprioceptive, vibration, thermo-algesic sensitivity and tactilesensitivity

Dizziness - Physical exam

Cranial nerve testing

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

Otoscopic examination, Hallpike manoeuvre

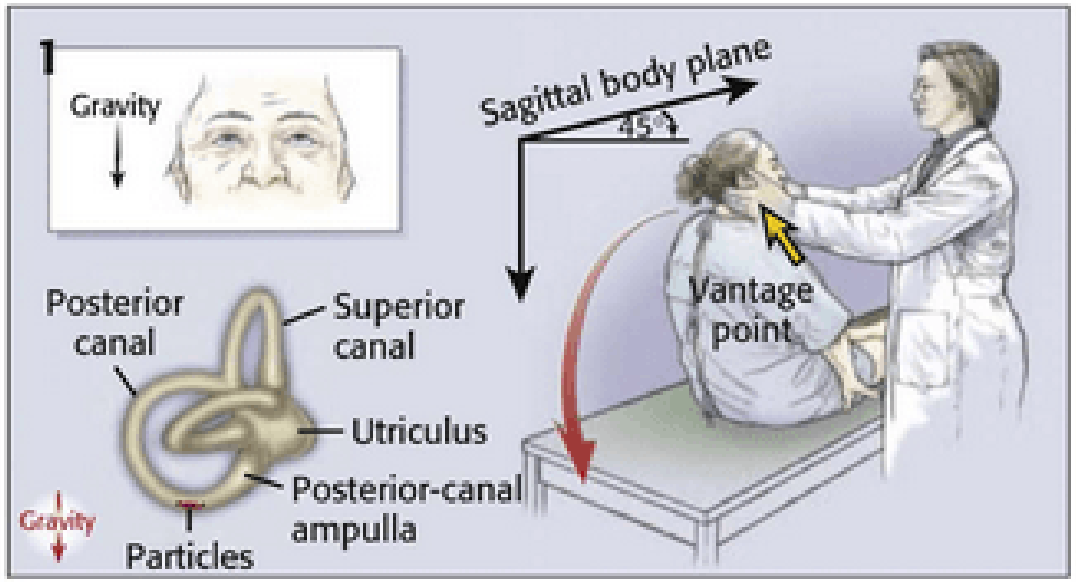
Neck : local tenderness or restriction in the range of movement

Congestive heart failure clinical signs : oedema, jugular turgor

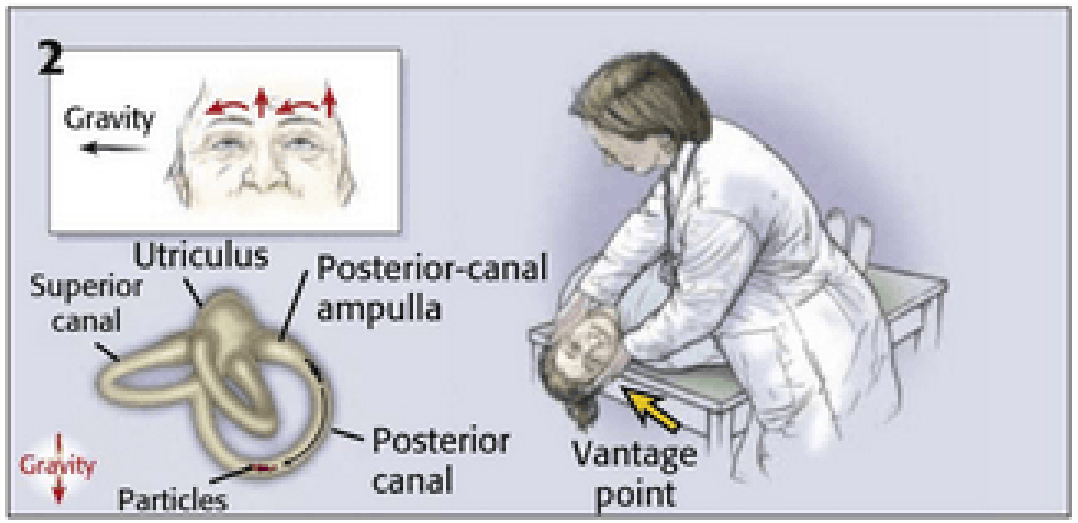
Carotid or heart murmur

Peripheral vascularisation

DIX-HALLPIKE MANEUVER

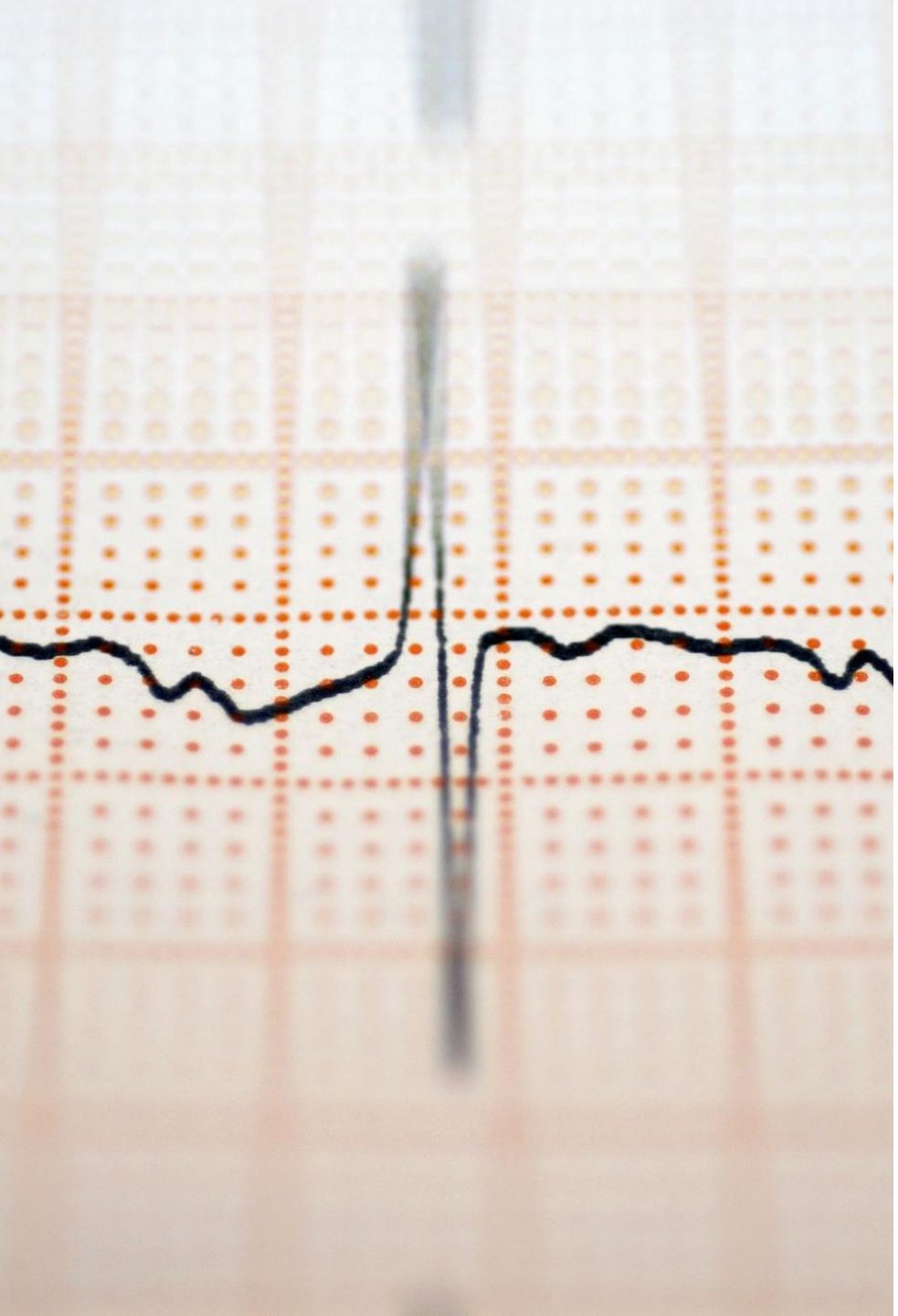


The examiner stands at the patient's head, 45° to the right, to align the right posterior semicircular canal with the sagittal plane of the body.



The 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.

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



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 will this exam be helpful in the care of this patient ? »

→ « Will the treatment 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 cases of neurinome (if in line with patient care plan)
- Vestibular rehabilitation
- Exercises 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 has to be considered as a cause and also a consequence of the dizziness → dilemma to manage anxiety with drugs able to cause dizziness → which is the main cause of anxiety → which 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 environmental review

Fall consequence 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 has to be **systematically screened** by anamnesis

Investigation, based on **anamnesis and physical exam**, **has to**
→ Deal with the patient care plan and wishes
→ Discuss target disease treatment if indicated
→ Identify the modifiable causing or contributing factors
→ Avoid negative clinical outcomes

Have you some questions related to this first part ?

Syncope - Content

- Definition and epidemiology
- Clinical outcomes
- Physiopathology
- Etiology and clinical presentations
- Evaluation
- Management
- Take home messages

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…)

Several clinical consequences

- Falls, fear of falling and increased risk of falling
- Injuries related to falls
- Loss of self-esteem, self-confidence and QOL decrease
- Hospitalisation and its negative clinical/functional consequences
- Institutionalisation
- Death

Physiopathology and etiology

Age-related physiological changes

- Reduced baroreflex sensitivity, reduced blood flow, reduced blood volume

Atherosclerosis

Specific disorders

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

Aspecific contributing factors

- Anemia, chronic lung disease, congestive heart failure, dehydration,
- Long standing, hot weather, alcohol, prolonged recumbency, fatty meals, …

Specific disorders

- Orthostatic hypotension
- Postprandial hypotension
- Reflex syncopal syndromes
  - Vasovagal syncope and carotid sinus hypernsitivity
- Cardiac syncope
- Cerebrovascular syncope

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 diastolic blood pressure
- Measured 2- 3 minutes after standing up.
- Measure heart beat are not necessary but recommended
  - Bradycardics or autonomic system disorder

Orthostatic hypotension - Causes

- **Not only age-related**
- **Medications**
  - alpha-blockers, beto-blockers, diuretics, morphinics, neuroleptics, L-dopa, dopa-agonists
- **Autonomic failure**.
  - Primary: multiple system atrophy, parkinson disease or pure autonomic failure
  - Secondary: diabetic neuropathy or amyloid neuropathy
- **Volume depletion**
  - Medications (diuretics or SGLT2), hemorrage, diarrhea, febrile illness, hot weather, extensive burn, third sector, mineralocorticoids deficiency

Orthostatic hypotension - Management

- **Medications review and reduction**
- **Avoid potential situation increasing reduced blood flow and correct anemia**
- **Secure the standing up**
- If no lower limbs occlusive arteriopathy → **compression stockings**
- (Fludrocortisone): few EBM data available. Consider 0.1 mg 1-3/day by patients with Parkinson's or severe diabetes (Cochrane review). Pay attention to oedema and HTA (→ reduce to 0.05 mg 1-3/day)
- (Midodrine) : = a + , but several contraindications including HTA, cardiomyopathy, artheriopathy, retinopathy, glaucoma

Postprandial hypotension

Mechanism:

- An increase blood flow in splanchnic and superior mesentery artery and a rise in plasma insulin level

- Without corresponding rises in sympathetic nervous system activity

Clinical presentation: similar to orthostatic hypotension but with a temporal relationship to meals and without relation with orthostatism.

Management:

- Small and frequent meals including complex carbohydrates

- Review of medications

- Avoidance reduced blood flow situations

Specific disorders

- Orthostatic hypotension
- Postprandial hypotension
- Reflex syncopal syndromes
  - **Vasovagal syncope and carotid sinus hypersensitivity**
- Cardiac syncope
- Cerebrovascular syncope

Syncope - Reflex syncopal syndromes

- Carotid sinus syncope
- Vaso-vagal syncope secondary to
  - Acute Pain : visceral pain, trigeminal neuralgia
  - Cough
  - Defecation
  - Micturition
  - Intensive exercise
  - Anxiety

Vasovagal syncope - Mechanism

Hypothesis → abnormal Bezold-Jarish reflex.

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

→ an abnormal vagal stimulus leading to a bradycardia AND a decrease in sympathetic vessels tonicity.

→ bradycardia and a blood pressure decrease → hypotension and/or bradycardia → sudden decrease of the cerebral blood flow.

Vasovagal syncope – Clinical presentation

A prodrome or aura / loss of consciousness / postsyncopal phases

Precipitating factors : extreme emotional stress, anxiety, physical pain, warm, environment, air travel, long standing

Prodrome: 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 seconds or heart rate < 40 beats/min for at least 10 seconds.

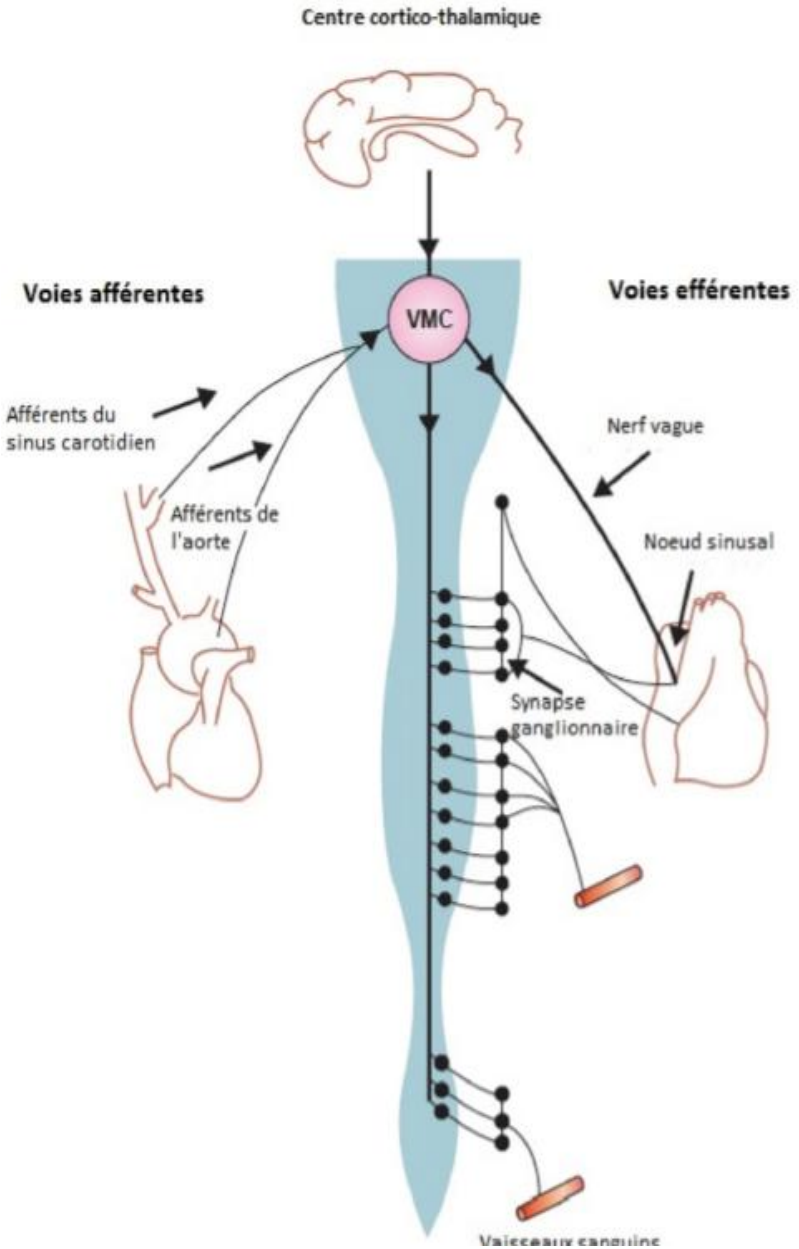
Tilt test

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, dehydration, any circumstances with volume depletion, …
- If no occlusive arteriopathy, → compression stockings
- If at least three seconds asystole → permanent cardiac pacing

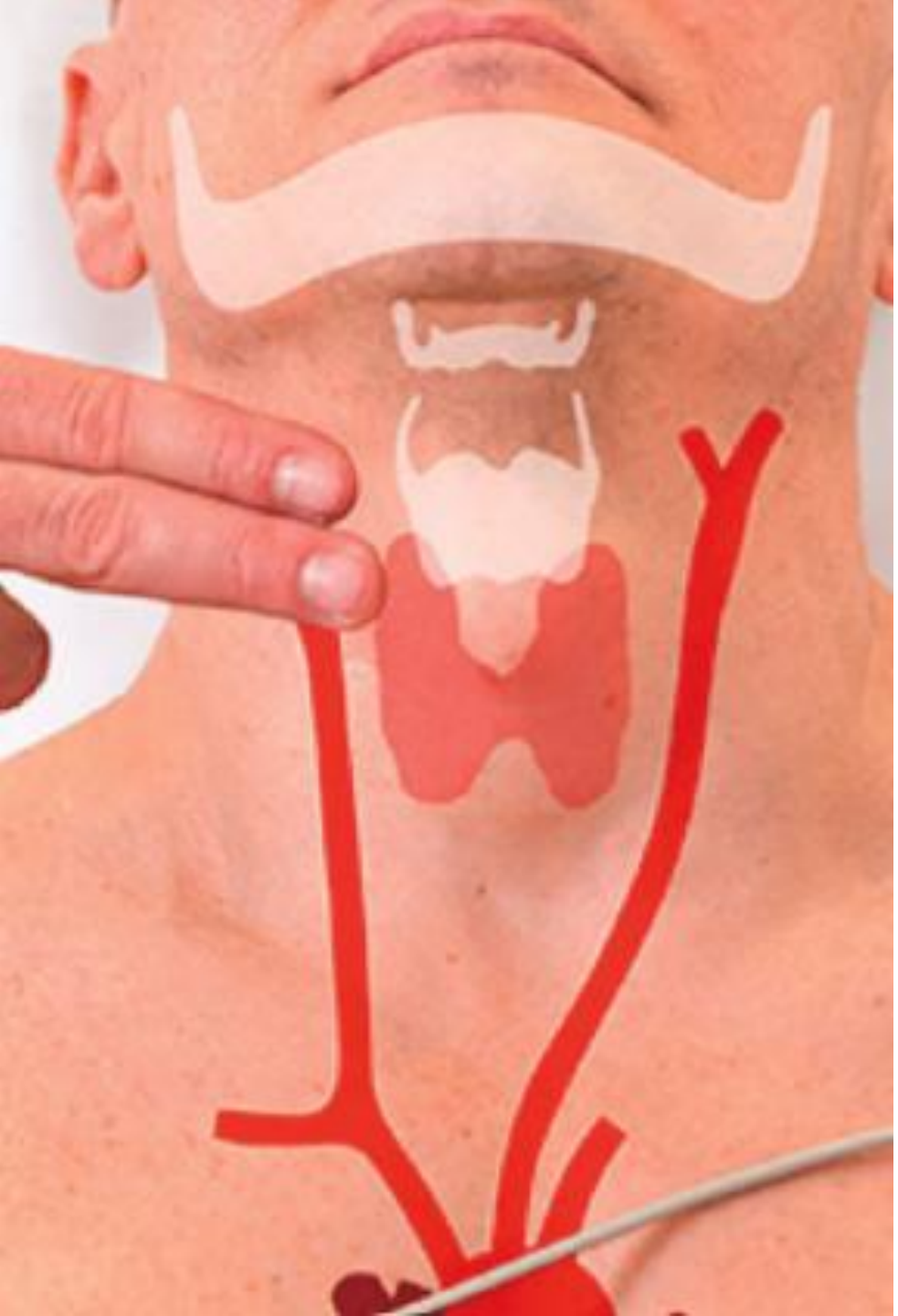


The carotid sinus syndrome or carotid sinus hypersentivity

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

Three forms:

- the cardio-inhibition form : **an at least three seconds asystole**
- the vasopressive form: **a decrease of at least 50 mm Hg** (systolic)
- the **mixed form**



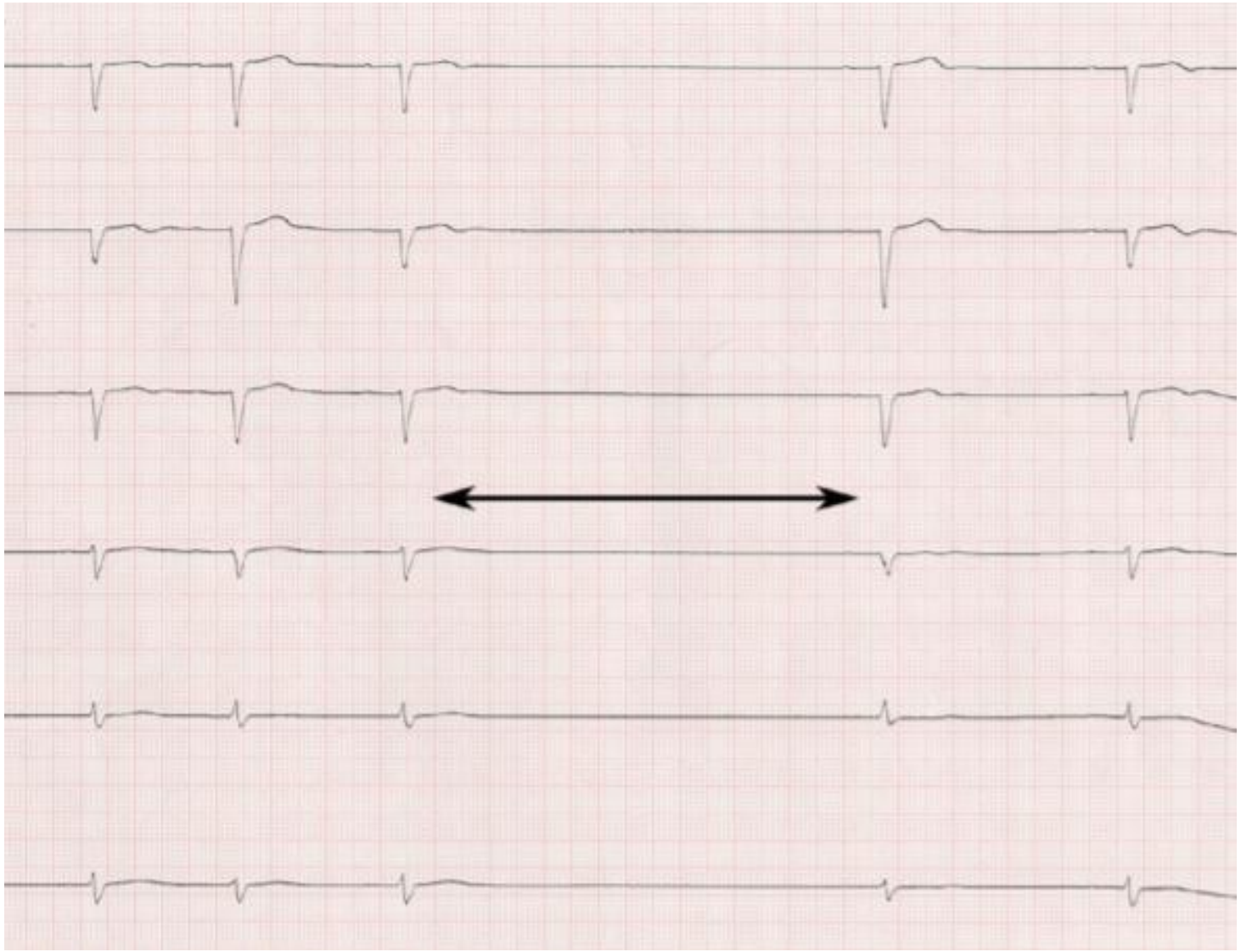
The carotid sinus syndrome or carotid sinus hypersentivity

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 seconds.

Complications include cardiac arythmia and neurologic disorders

Carotid sinus massage 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 has a carotid murmur (American Heart Association). Caution should be taken in cases of ventricular arythmia.



The carotid sinus syndrome or carotid sinus hypersensitivity

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 (rhythmic 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 → asystole > 3 seconds, rapid supraventricular or ventricular tachycardia;

An echocardiograph;

An exercise stress test;

An electrophysiologic study.

Cardiac syncope, ESC recommendations

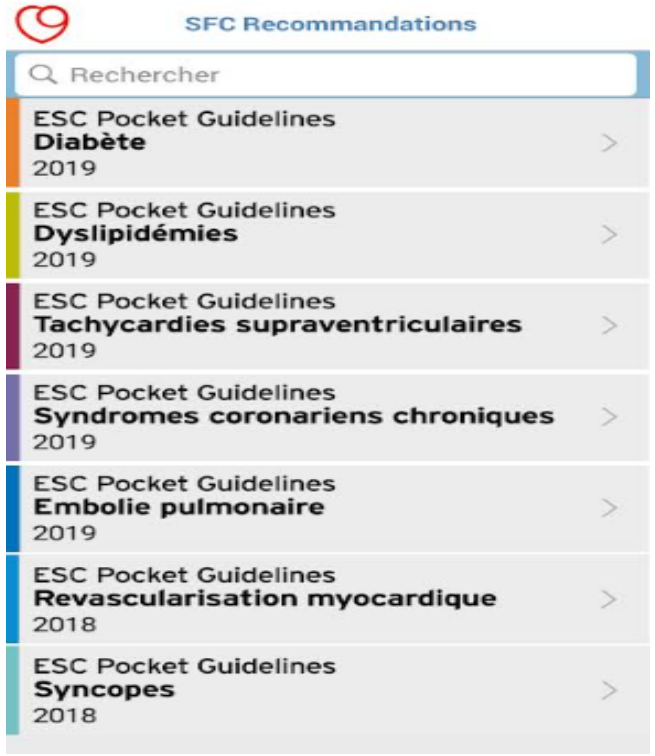
Recommendations of the European Society of Cardiology

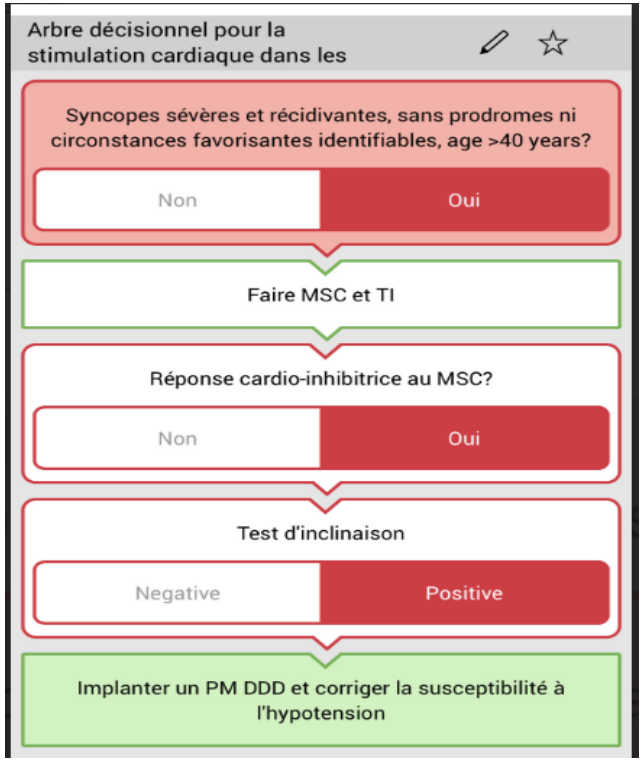
- Mechanisms
- Classification
- Algorithm of investigation and management

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

Cardiac syncope, ESC recommendations

Recommendations of the European Society of Cardiology





Cardiac Syncope and medications

As a reminder,

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

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	Trilifan®
Pimozide	Orap®
Pipampérone	Dipipéron®
Pipotiazine	Piportil®
Propériciazine	Neuleptil®
(hydro)quinidine	Sérécor®
Sotalol	Sotalex®
Spiramycine	Rovamycine®
Sulpiride	Dogmatil®, Synédil®
Sultopride	Barnétil®
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

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

Management: Specific to the cause and according to the patient profile

Syncope – Specific disorders

- Orthostatic hypotension
- Postprandial hypotension
- Reflex syncopal syndromes
  - Vasovagal syncope and carotid sinus hypernsitivity
- Cardiac syncope
- **Cerebrovascular syncope**

Cerebro-vascular syncope

Causes : Aortic/carotid dissection,

Subclavian derivation,

Transient ischemia, stroke,

Thrombo-embolic events

Diagnosis : MRI with gadolinium / CT scan with contrast

Investigation and management are disease-specific

Syncope - A challenge for the 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 the clinician

Management should include

- Fear of falling
- Walk and balance abilities
- Physiotherapy
- Environment
- Medications review : especially platelet aggregation inhibitors, anticoagulation therapy, sedative, diuretics….
- Ability to drive should be considered

- **Minimal screening CGA** -> and more if needed

Take Home Messages

Syncope and dizziness are **frequent and could have 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 challenges** for the clinician.

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

Whatever the cause, don't forget to do a comprehensive geriatric assessment

and avoid negative clinical consequences

This is the end of this course

I would like to thank you for your attention

Please, feel free to ask your questions

After your questions, please, take time to answer the satisfaction survey

Your comments are important to know your needs of instruction

You have 10 minutes to fill in the survey

Before leaving, don't forget to sign the attendance list and give us the survey

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