Fundamentals of Video-nystagmography: Theory, Practice, and Clinical cases

Part 2: Spontaneous testing, Gaze testing, Head shaking, Hyperventilation

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Learner Outcomes

- After this course, participants will be able to describe spontaneous testing, identify influencing factors, and discuss the results as they relate to vestibular function.
- After this course, participants will be able to describe the results from provocative testing, including the headshaking test and hyperventilation testing.
- After this course, participants will be able to describe the characteristics of vestibular nystagmus and differentiate between its various types based on observed eye movements.

Outline of the talk

- Revision of previous session 
- Spontaneous testing 
- Influencing factors 
  - Gaze testing
  - Visual fixation
- Provocative testing 
  - Head shaking test
  - Hyperventilation testing
- Vestibular nystagmus 

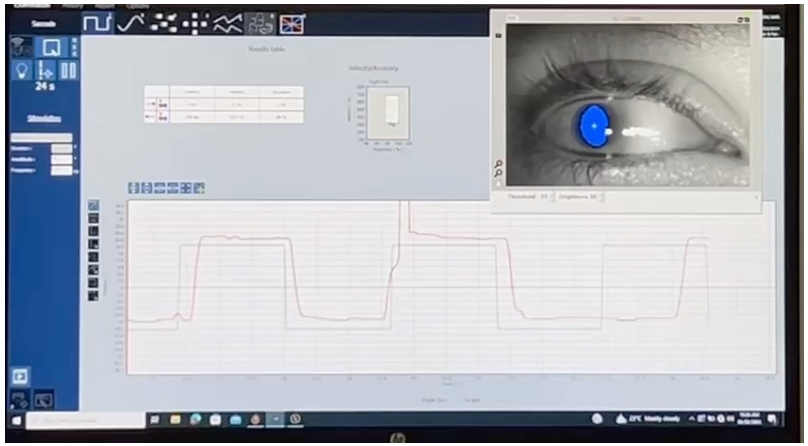
Functional types of eye movements control

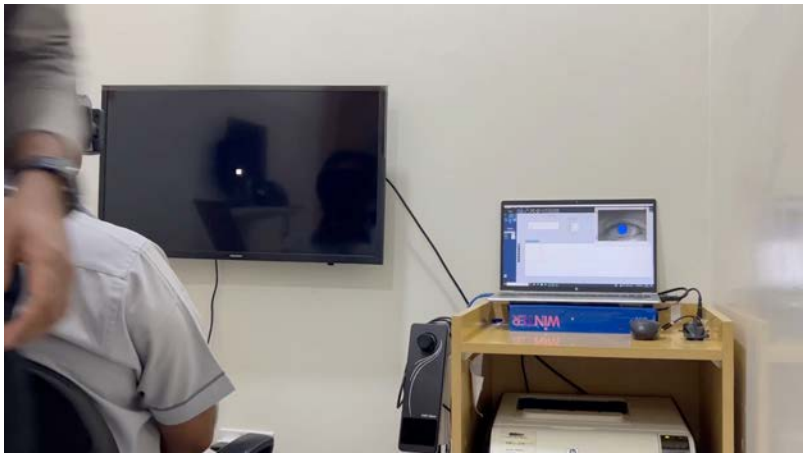
- Saccades
- Smooth pursuit
- Opto-kinetic reflex
- Vestibulo-ocular reflex
- Vergence
- Visual Fixation

Purpose of eye movements

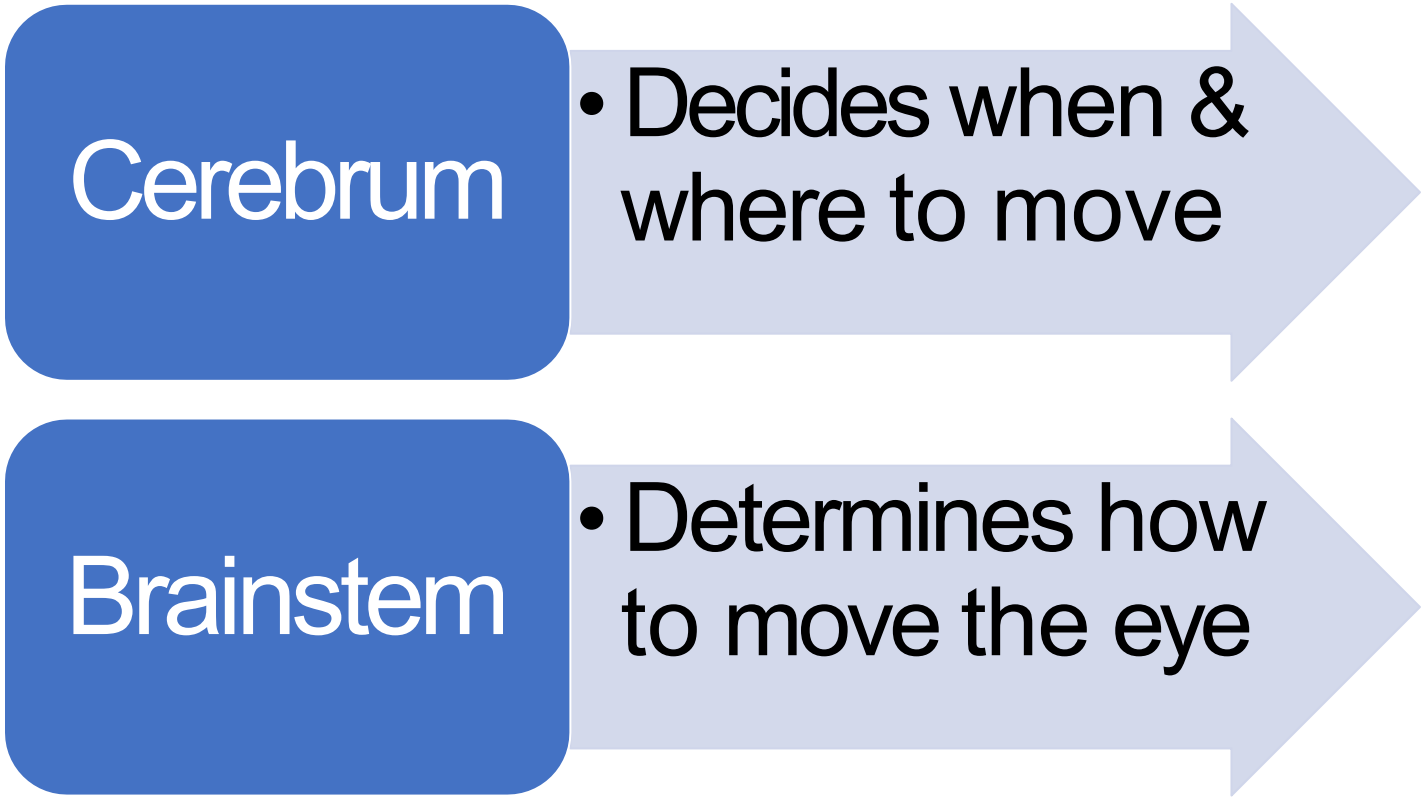
Saccades
Smooth pursuit
Opto-kinetic reflex
Vestibulo-ocular reflex
Vergence
Visual Fixation

Hold images of the visual world steady on the retina during brief head rotations or translations (linear movements)
Hold the image of a stationary object on the fovea by minimizing ocular drifts
Hold images of the visual world steady on the retina during sustained head rotation or translations
Hold the image of a small moving target on the fovea; or holds the image of a small target on the retina that is close to the head, during linear motion (translation); with optokinetic responses, aids gaze stabilization during sustained head rotation
Bring images of objects of interest onto the fovea
Move the eyes in opposite directions so that images of a single object are placed or held simultaneously on the fovea of each eye

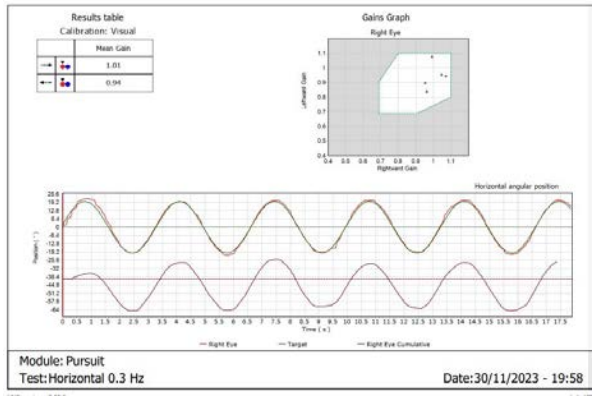




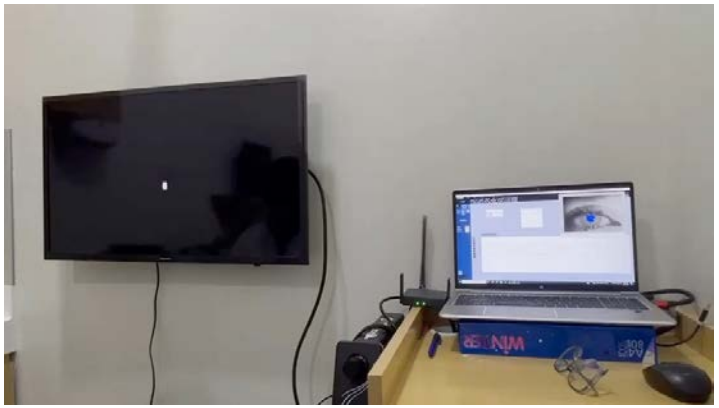
Horizontal saccade testing

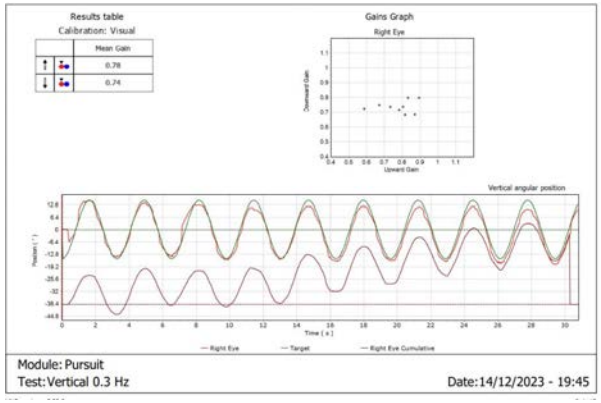

Cerebrum
• Decides when & where to move
Brainstem
• Determines how to move the eye

Horizontal smooth pursuit testing

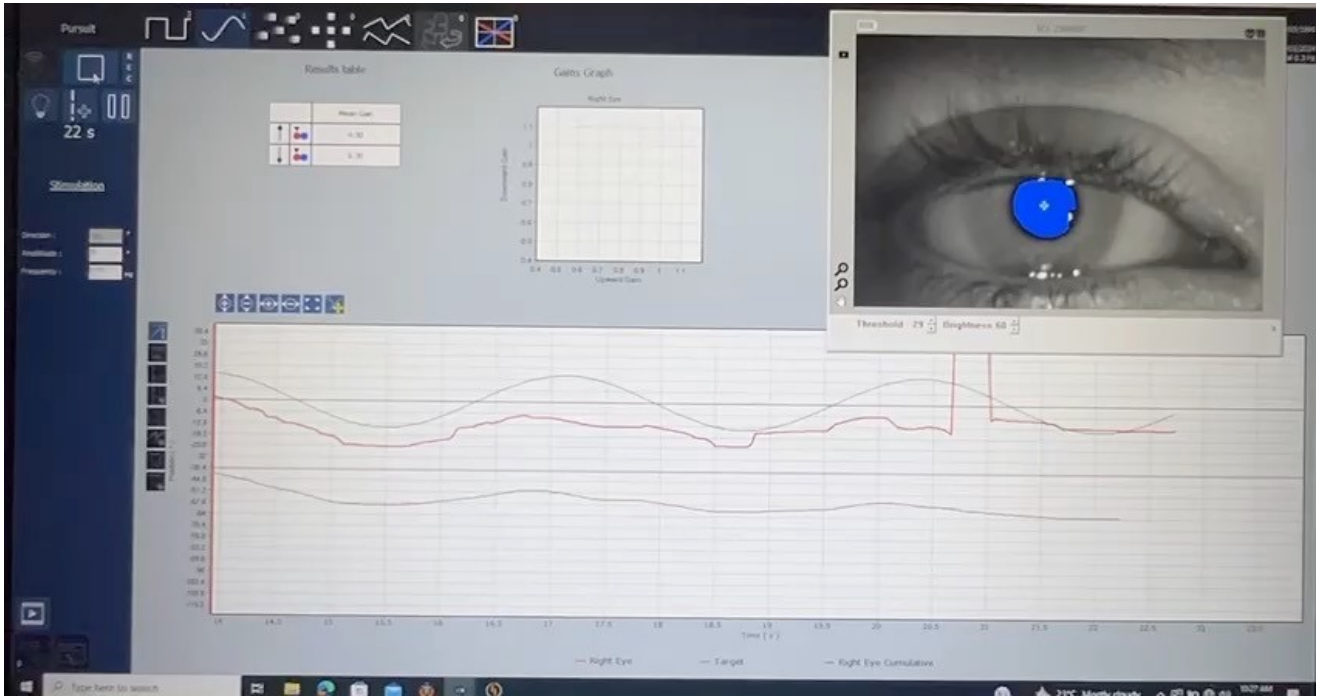


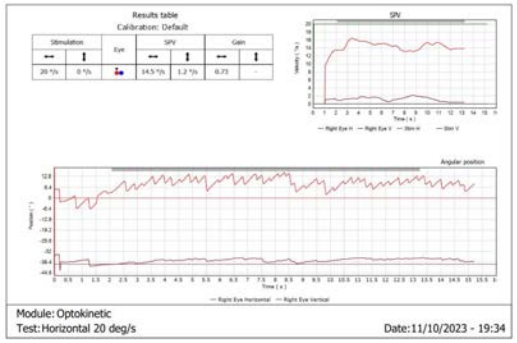
Vertical smooth pursuit testing

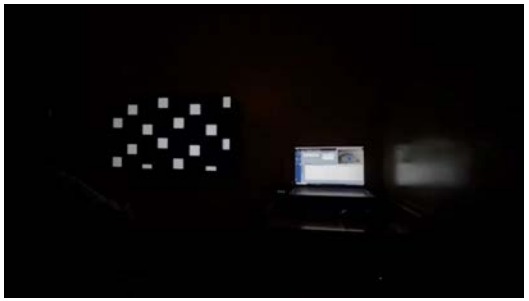


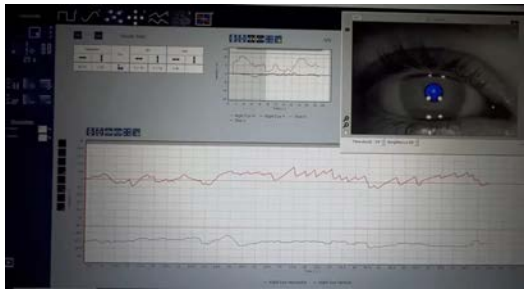


Vertical smooth pursuit testing







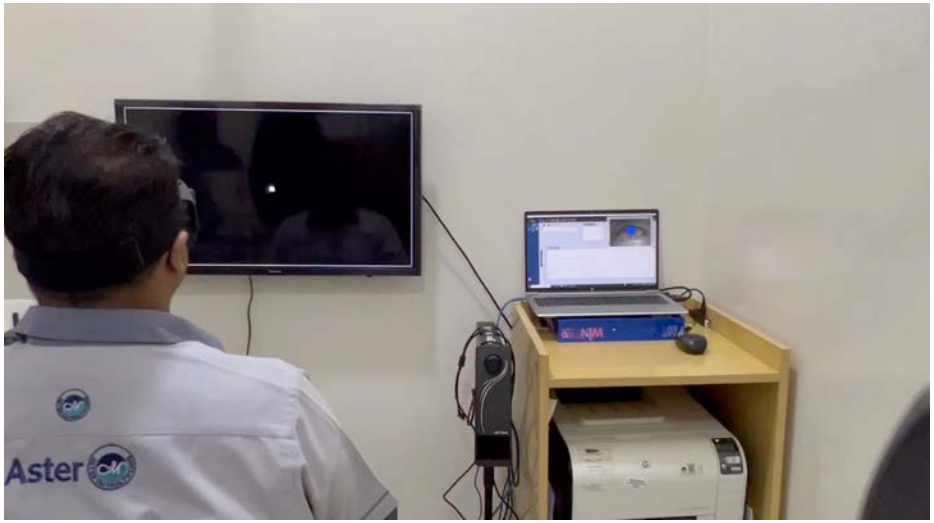


Optokinetic testing (Horizontal stimulation)

Spontaneous testing

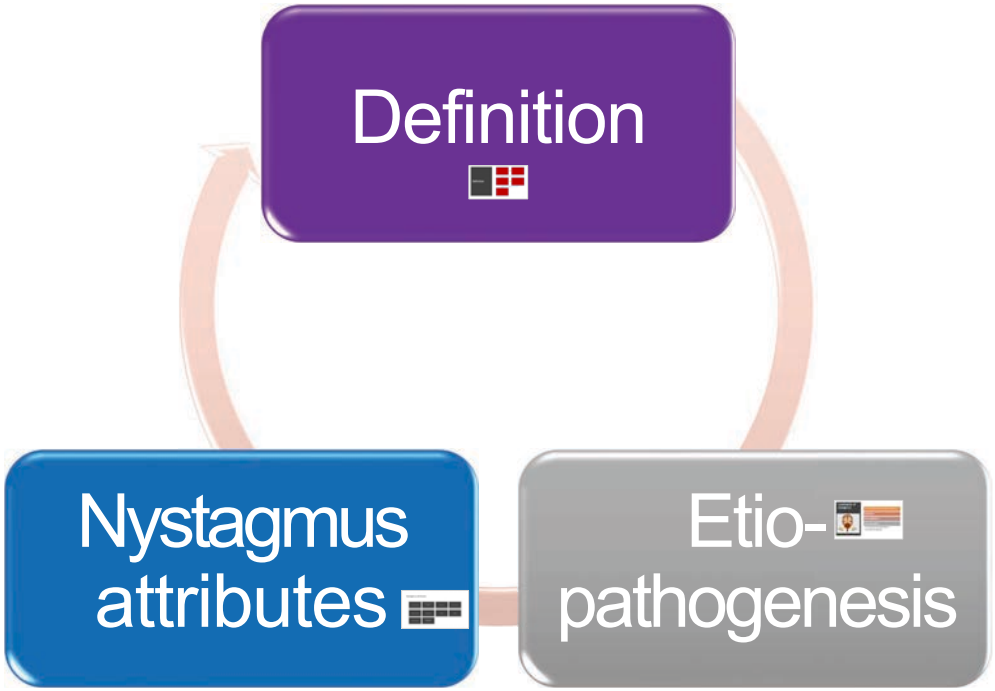
- Nystagmus
- Skew deviation
- Lids (Ptosis)
- Pupils (Asymmetry-Dilatation, Constriction)

SPN in light (With fixation)

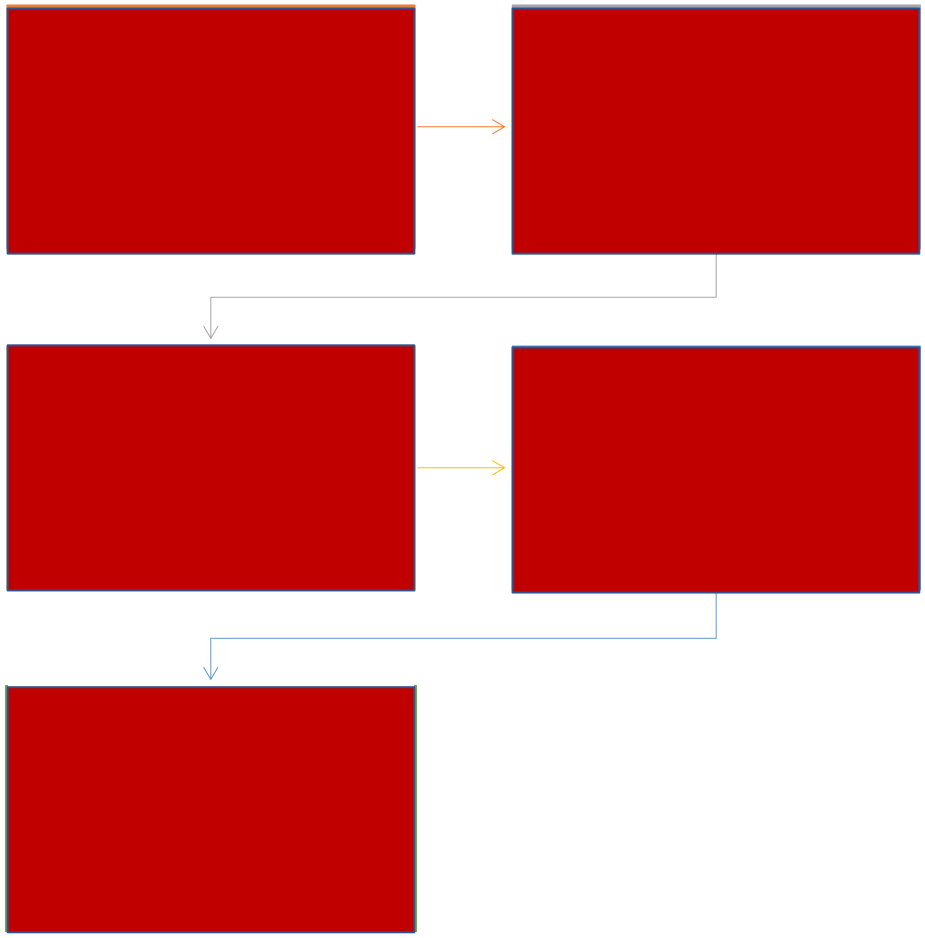


SPN in dark (Without fixation)

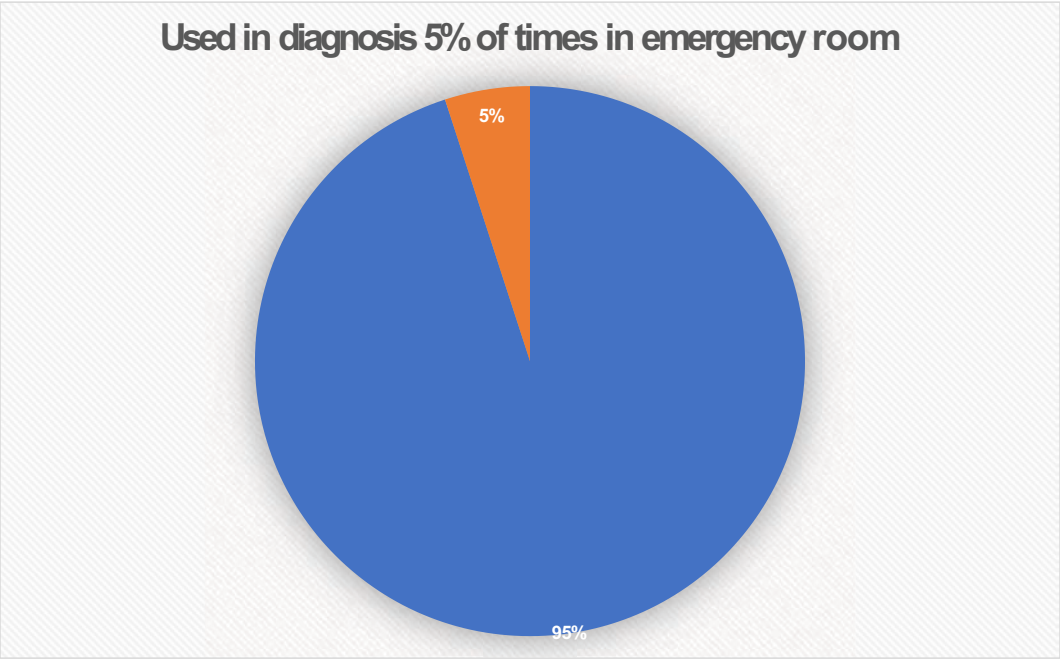
Nystagmus



Definition



Under-utilised in the diagnosis?



Nystagmus -present

Kaplan PW, Tusa RJ. Neurophysiologic and clinical correlations of epileptic nystagmus. Neurology. 1993; 43:2508–2514.

A common diagnostic challenge

Causes of nystagmus
Physiological to life-threatening

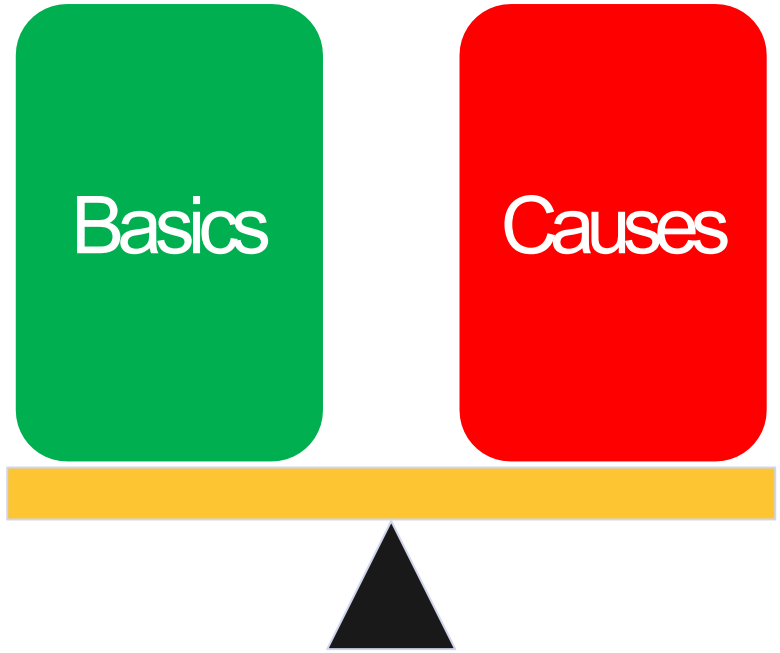
Central or peripheral?

Am I missing the central?

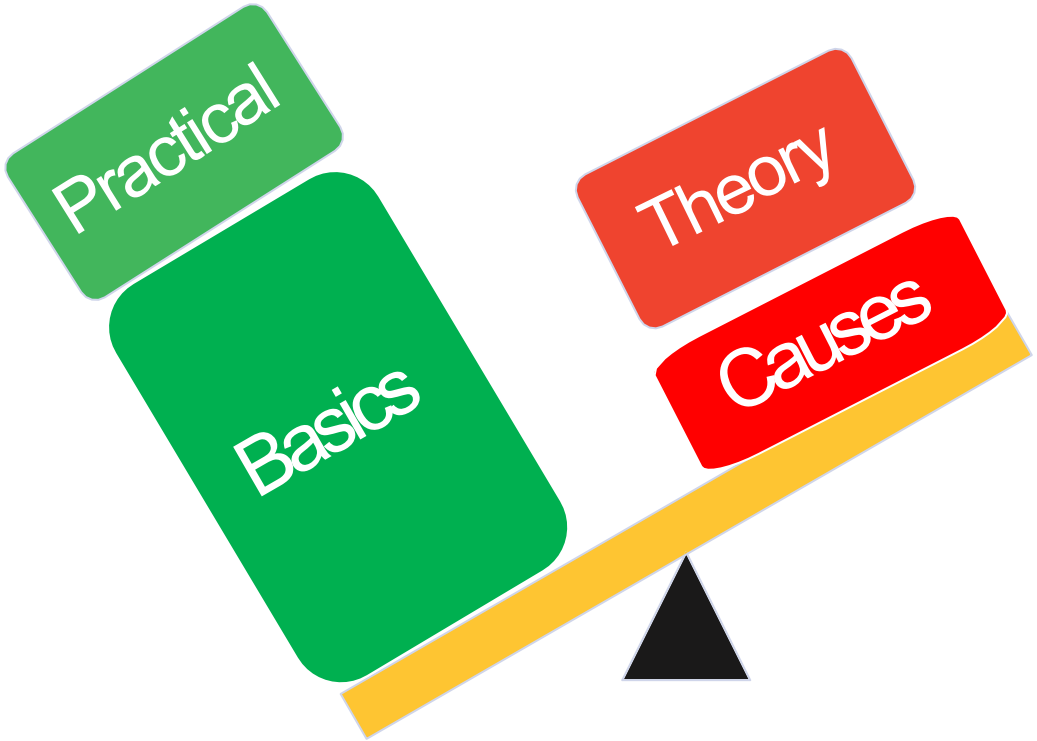
MRI brain- Normal

Creates anxiety and confusion

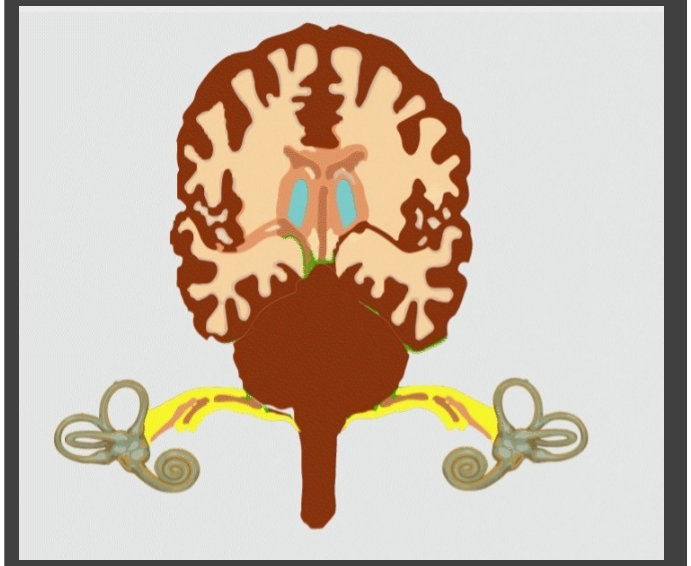
47 different types



47 different types



Localisation of nystagmus



The peripheral vestibular apparatus

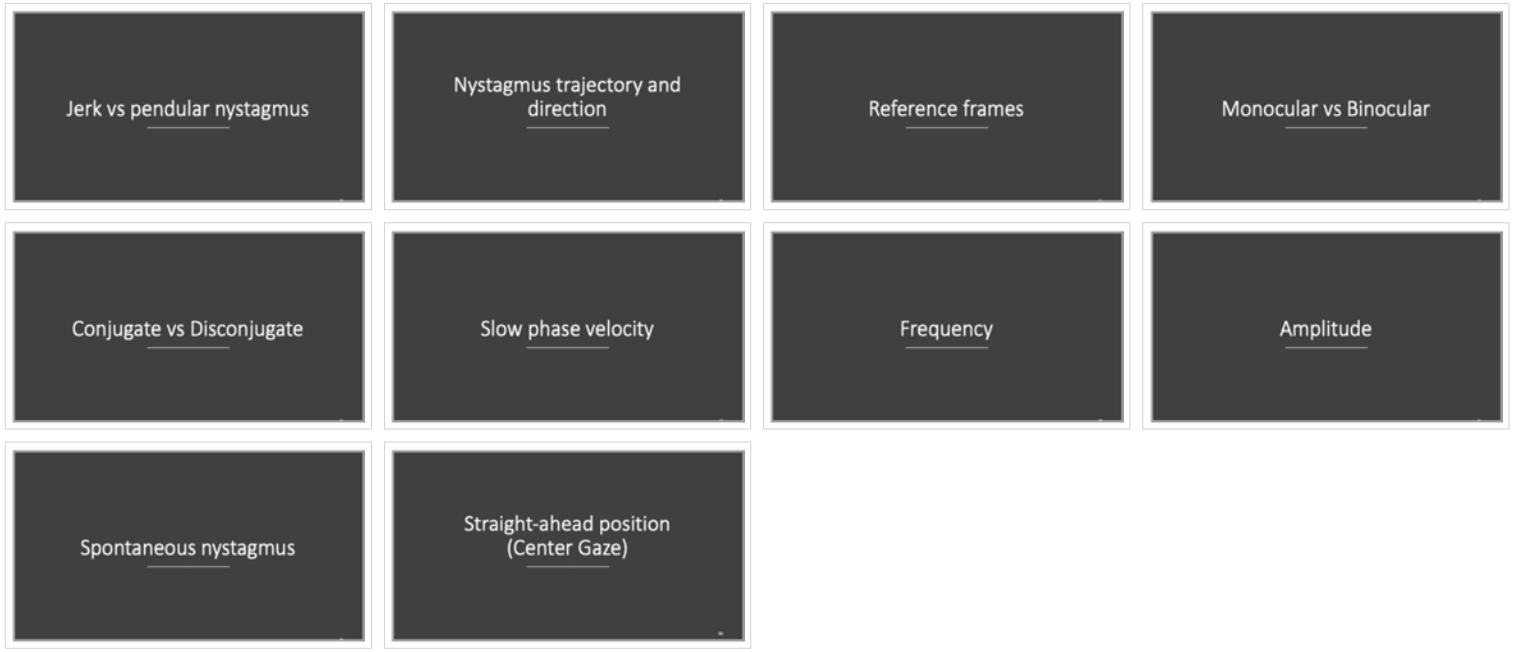
Brainstem

The cerebellum

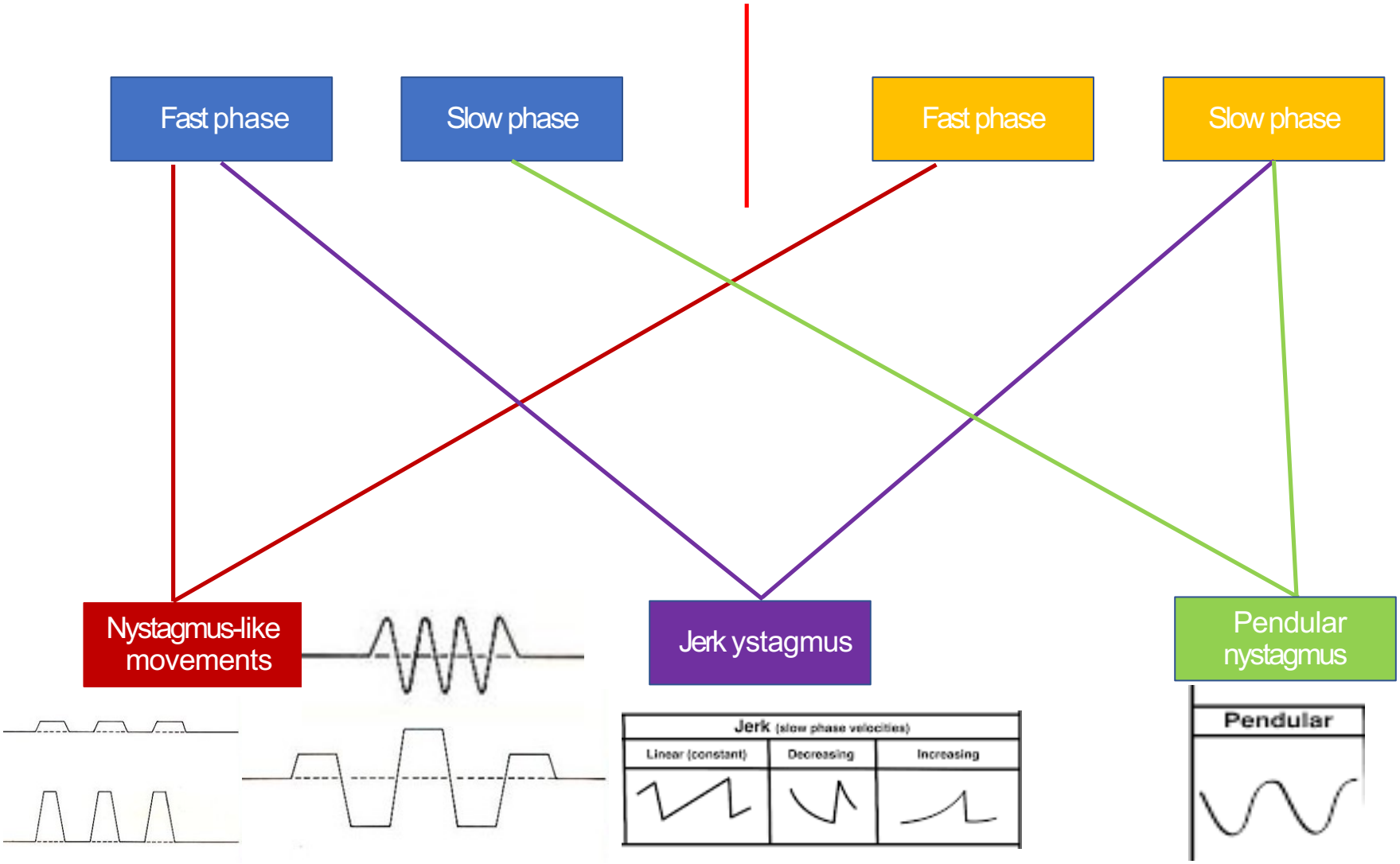
Less commonly

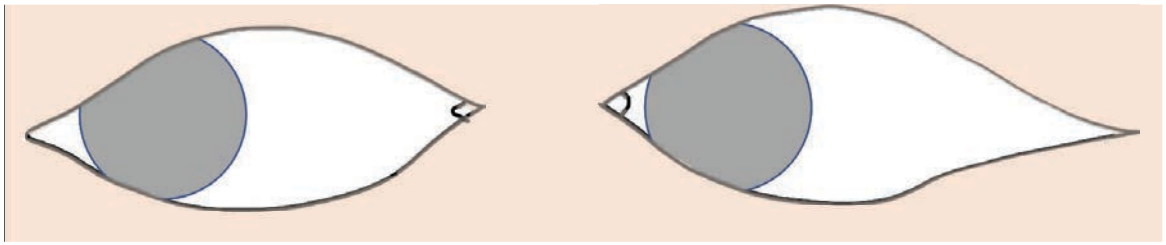
- The anterior visual pathways or
- Cerebral hemispheres.

Nystagmus attributes



Jerk vs pendular nystagmus

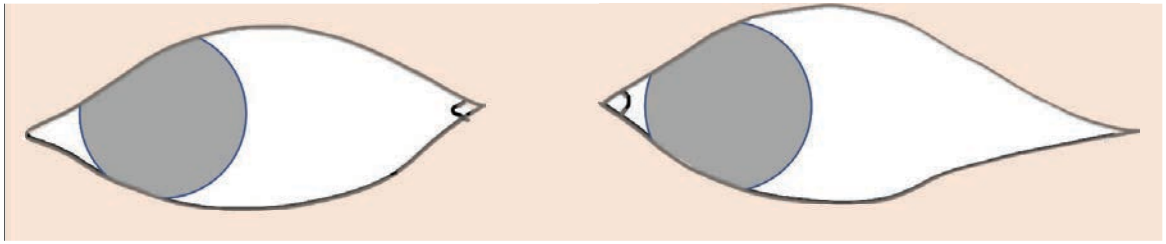

Fast phase
Slow phase
Fast phase
Slow phase
Nystagmus-like movements
Jerk ystagmus
Pendular nystagmus
Jerk (slow phase velocities)
Linear (constant)
Decreasing
Increasing
Pendular



Jerk nystagmus

Slow Component

Slow component – Genesis and Continuation
(Analysis – Anatomical localisation)



Fast component

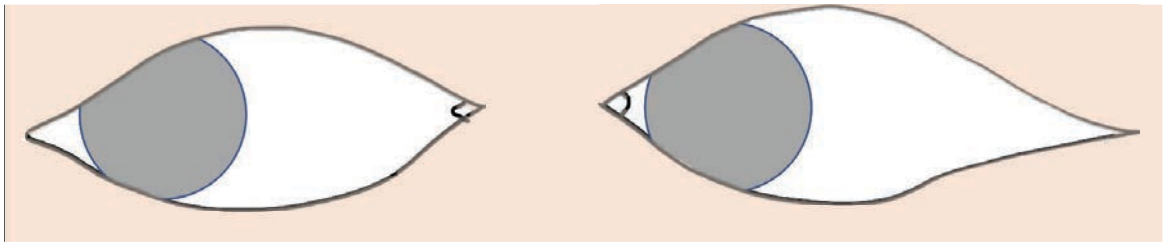
Slow Component

Jerk nystagmus

Slow component – Genesis and Continuation
(Analysis – Anatomical localisation)

Fast component- Corrective component

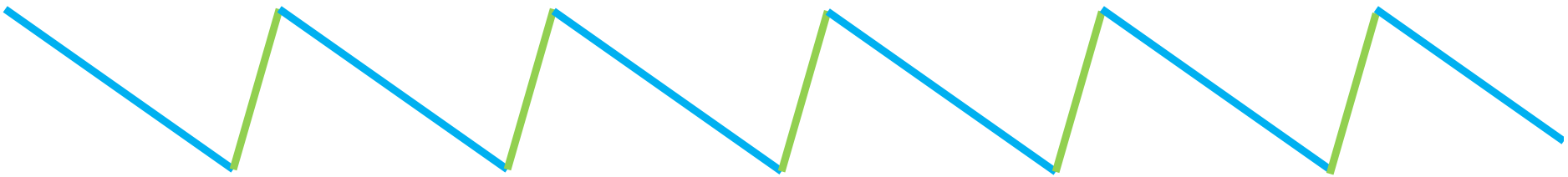
By convention, the direction of nystagmus is defined as the direction of the fast component

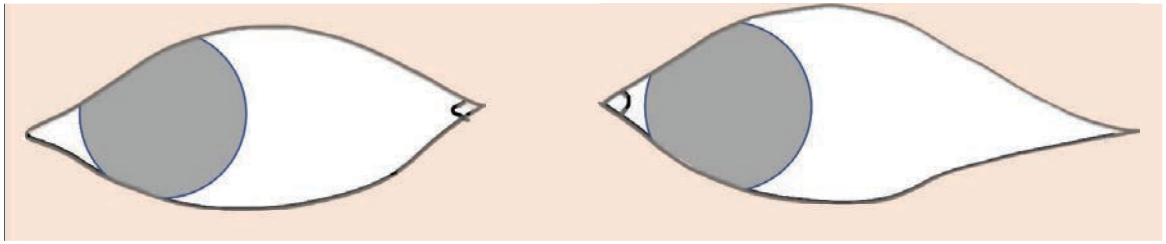


Jerk nystagmus

Fast component

Slow Component

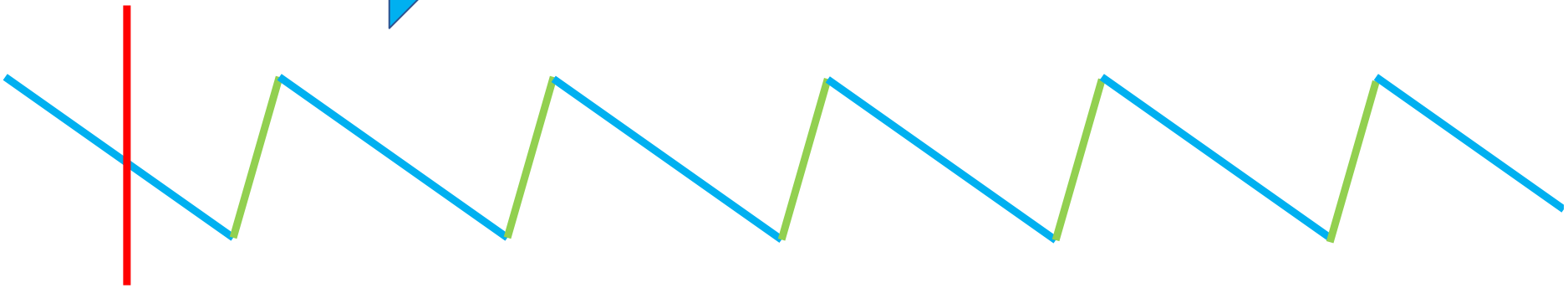


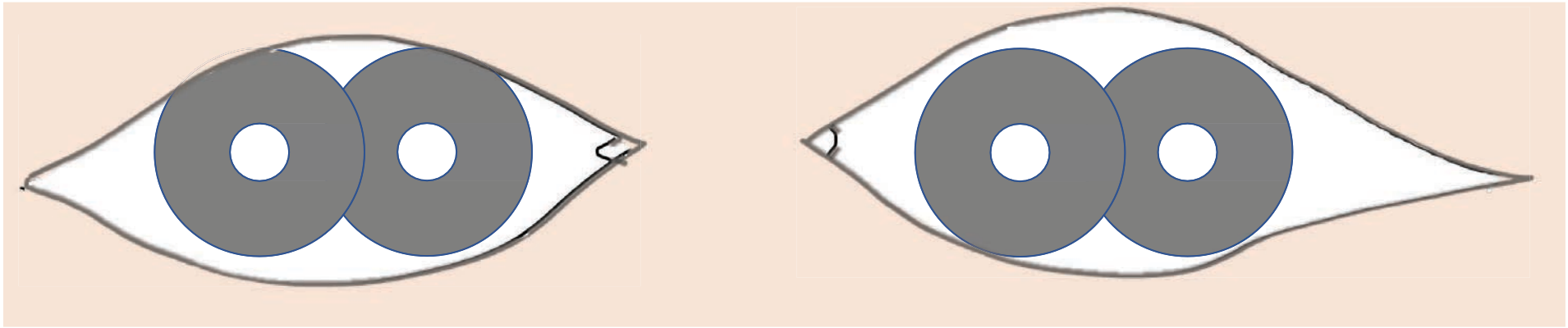


Jerk nystagmus

Fast component

Slow Component



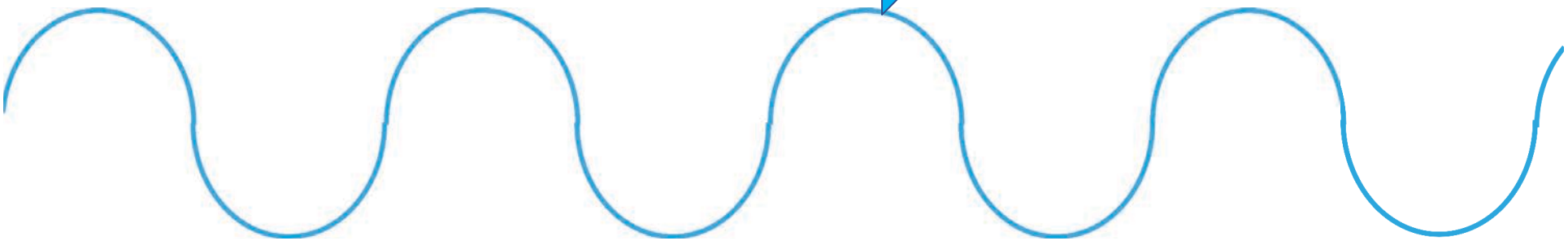


Pendular nystagmus

Central
Brainstem or cerebellar in origin

Slow component

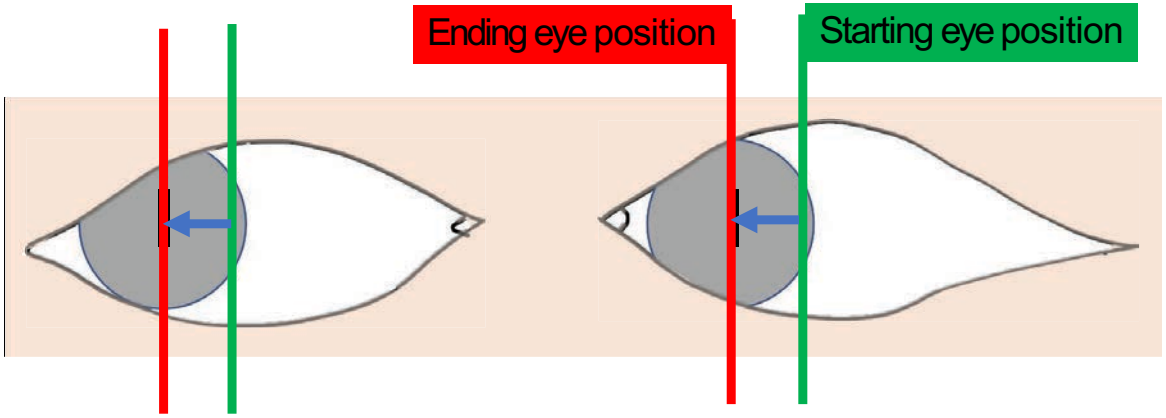
Slow Component



Nystagmus trajectory and direction

Nystagmus trajectory

- The path taken from starting eye position to ending eye position during one half cycle of movement is known as the **trajectory.**



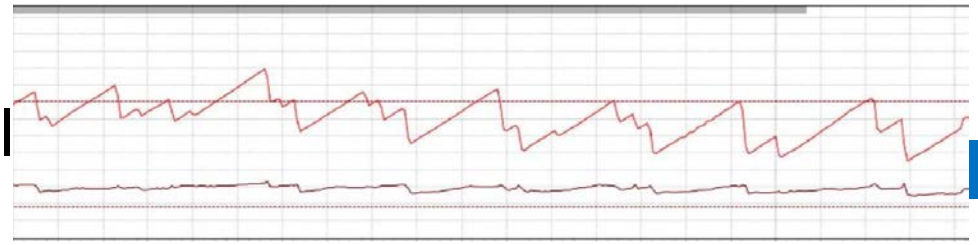
Trajectory

Horizontal

Vertical

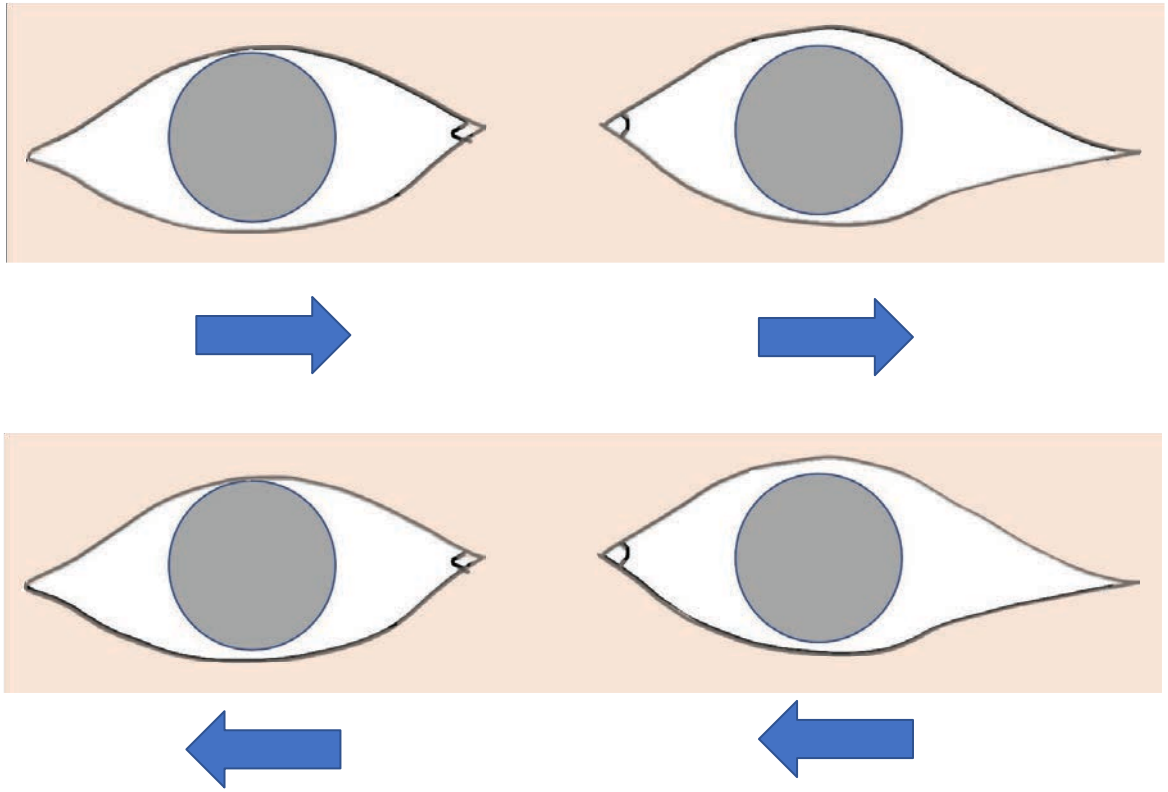
Torsional

Trajectory- Horizontal



Direction

Right

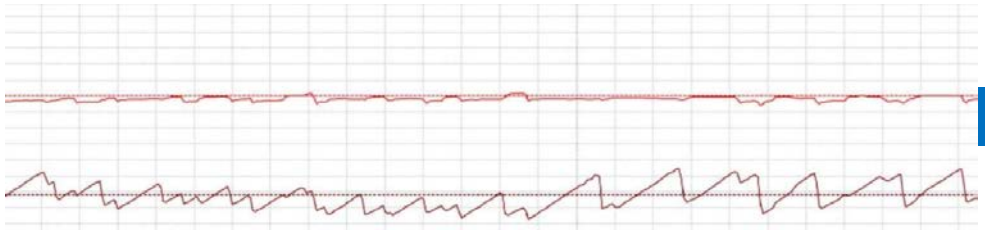


Left

Left beating

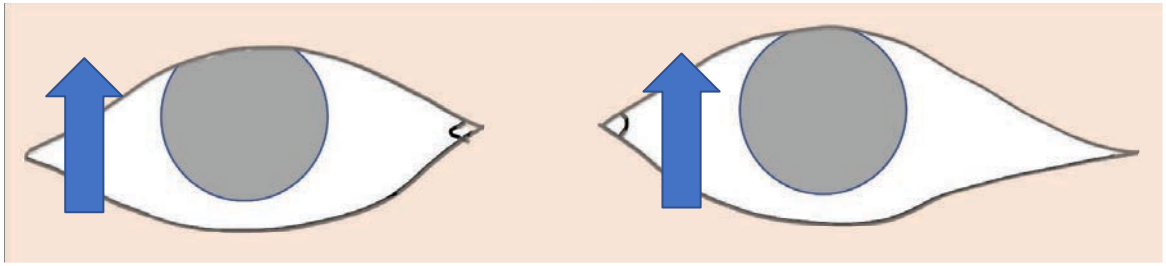
Right beating

Trajectory -Vertical

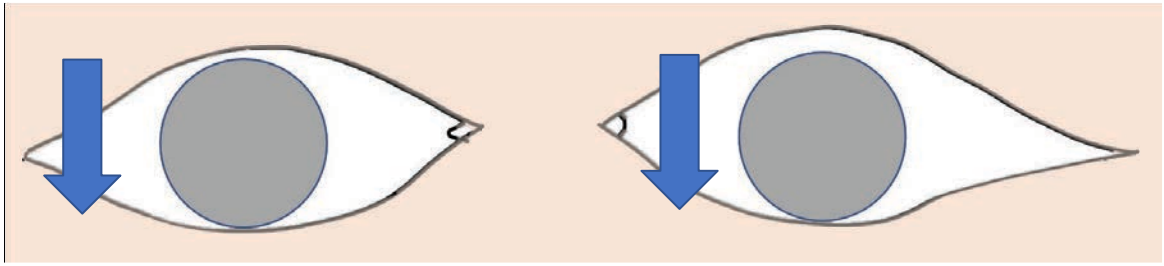


Direction

Up



Upbeating



Downbeating

Down

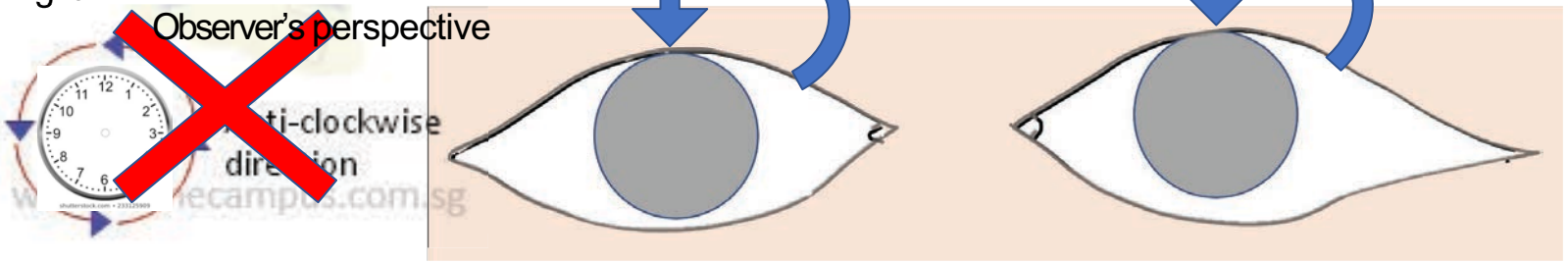
Trajectory- Torsional

Direction

Right

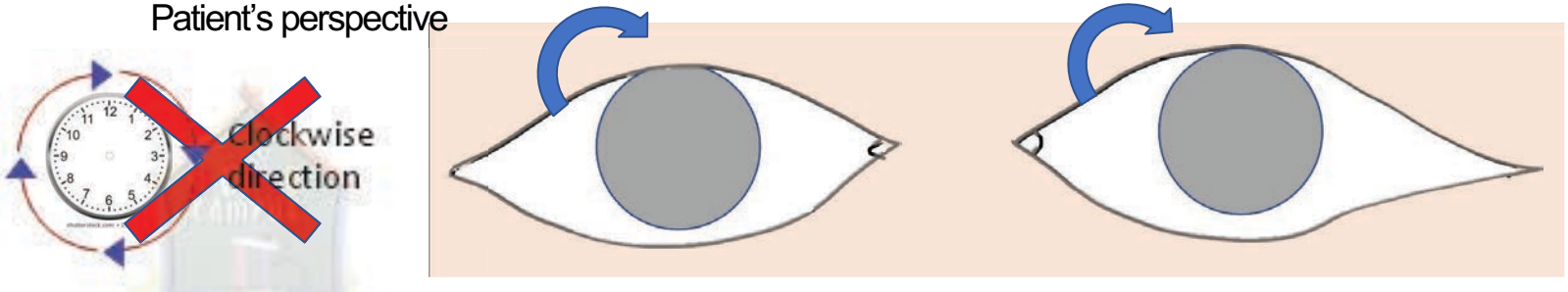
Left

Observer's perspective



Upper pole beating to right
Dextro-cyclo-rotation

Patient's perspective



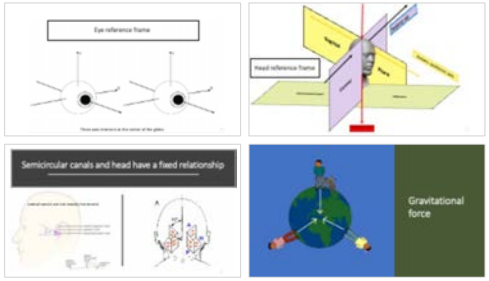
Upper pole beating to left
Levo-cyclo-rotation

Reference frames

Reference frames

Eyes

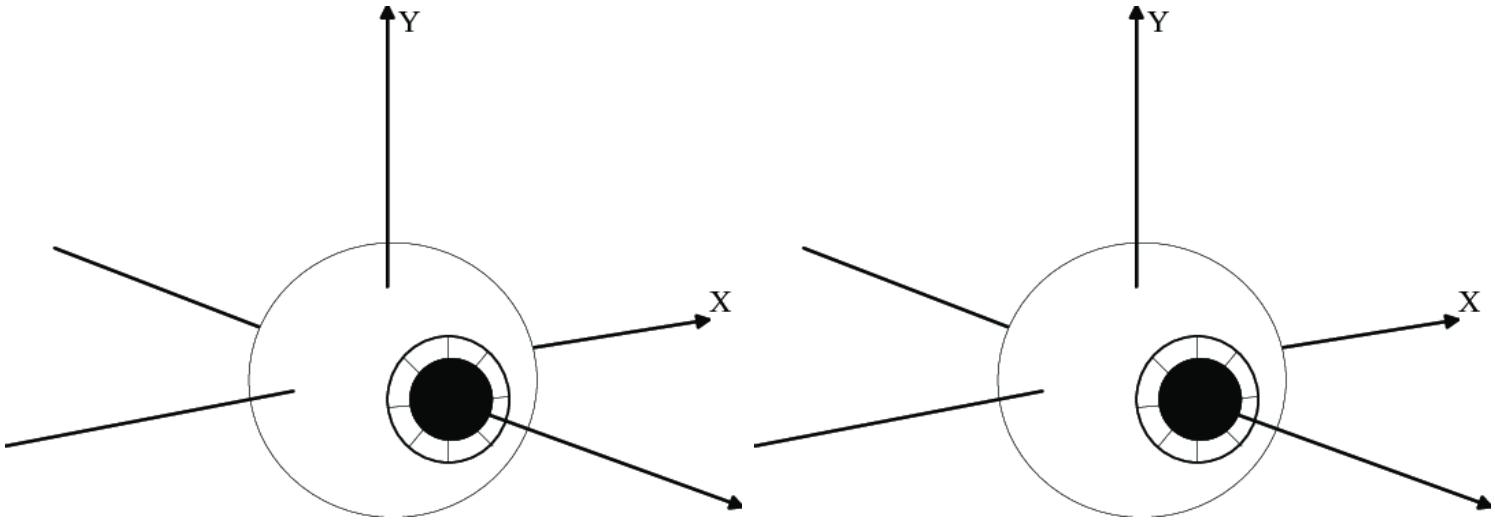
Canal



Head

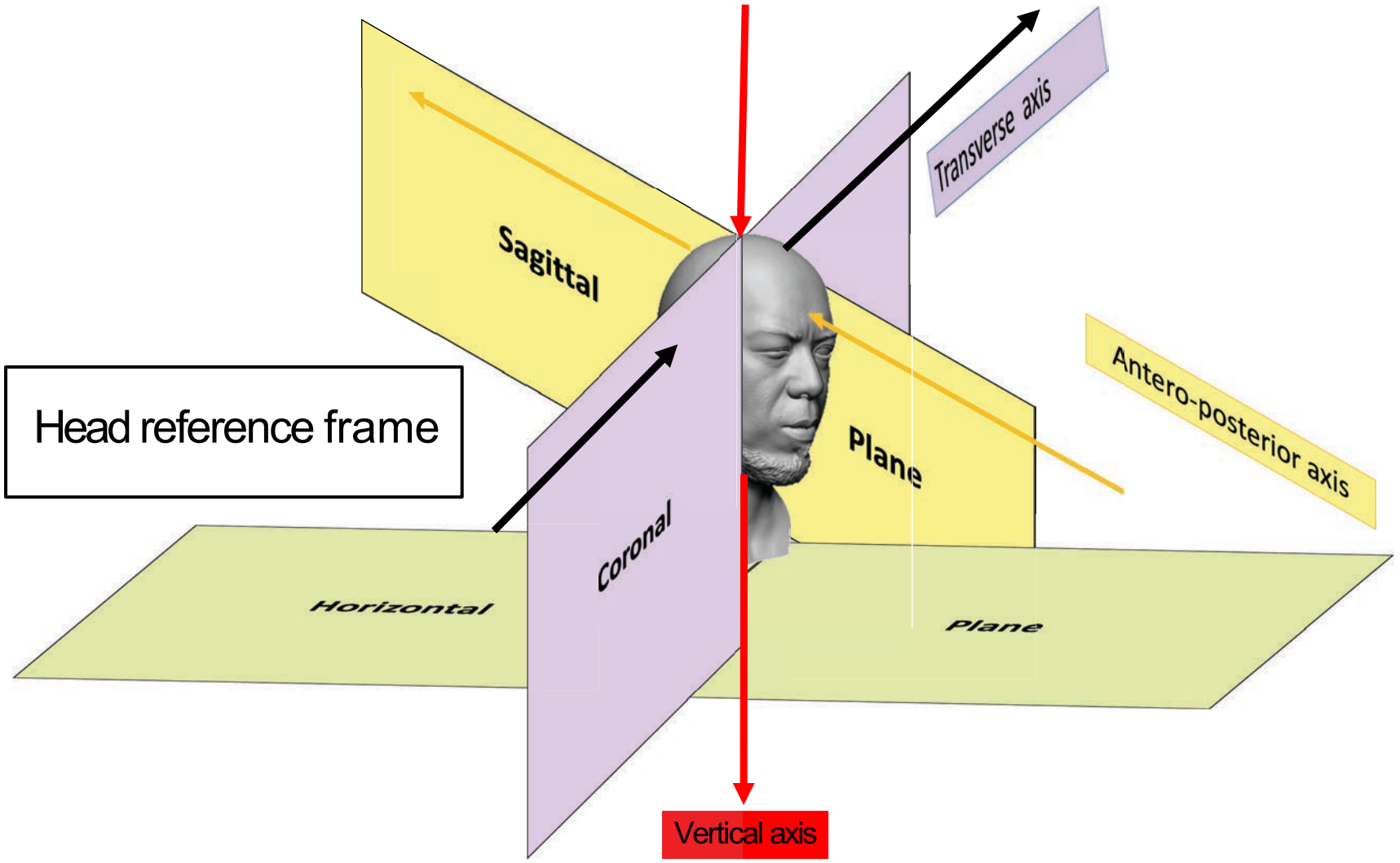
Earth

Eye reference frame

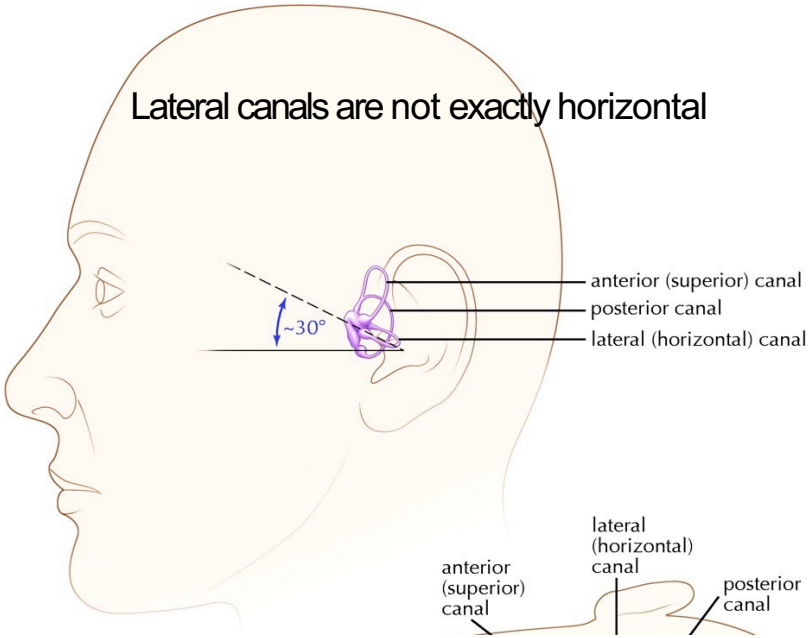


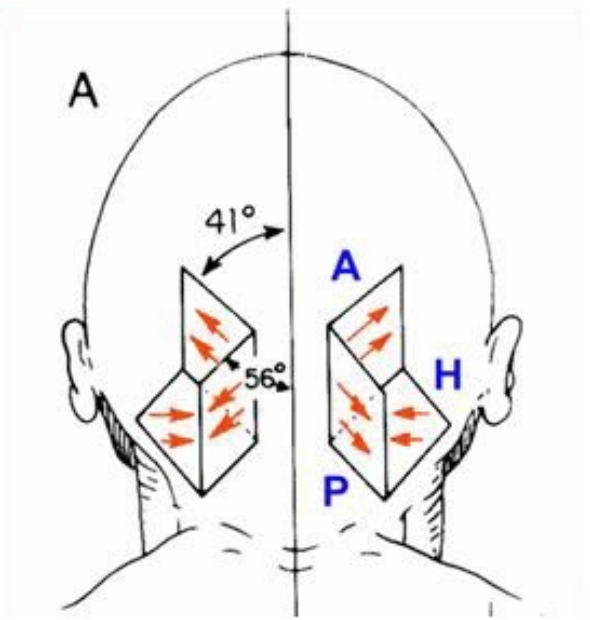
Three axes intersect at the center of the globe.

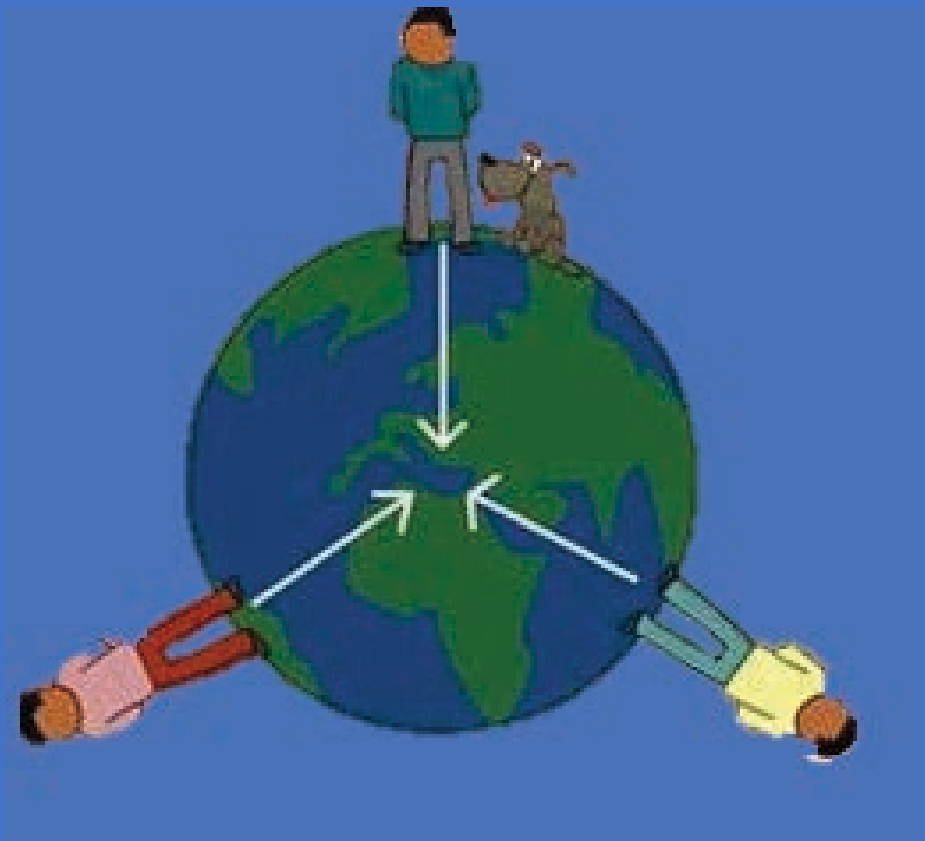
Head reference frame



Semicircular canals and head have a fixed relationship



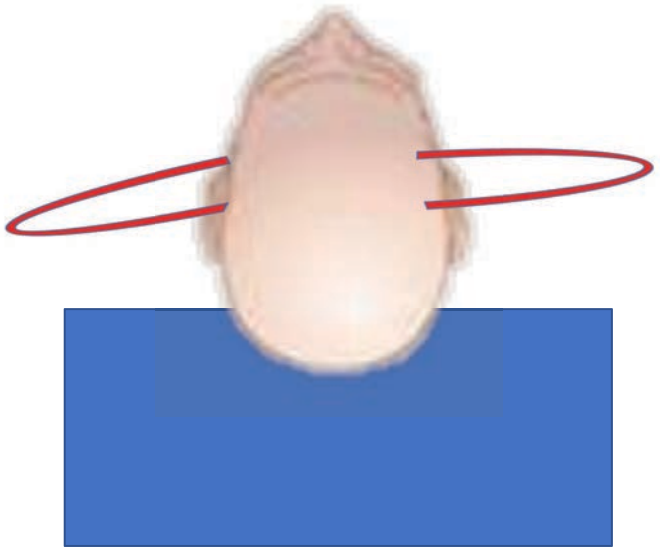




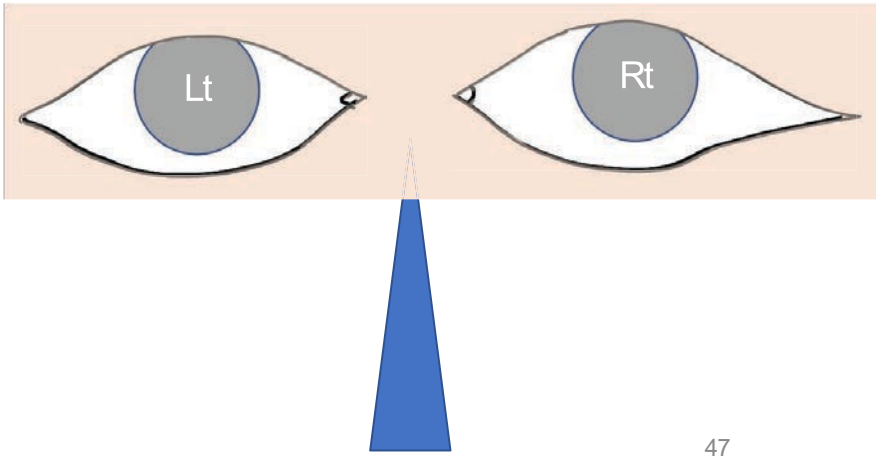
Gravitational force

Neutral position

Left

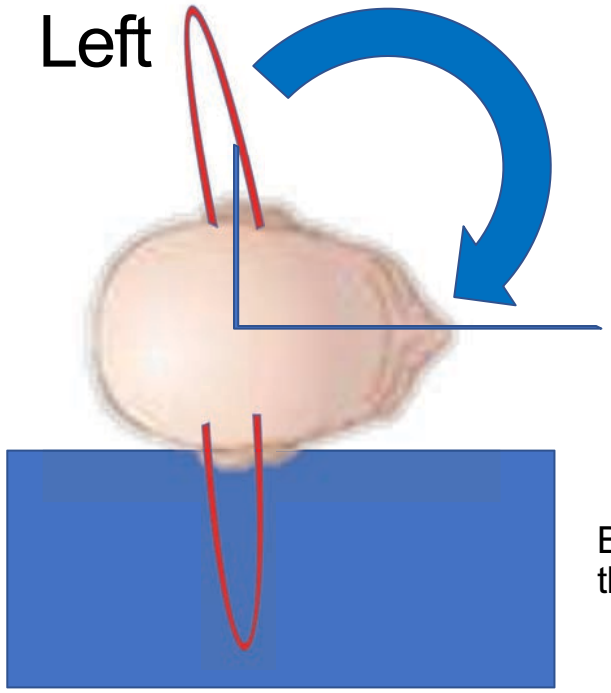


Right



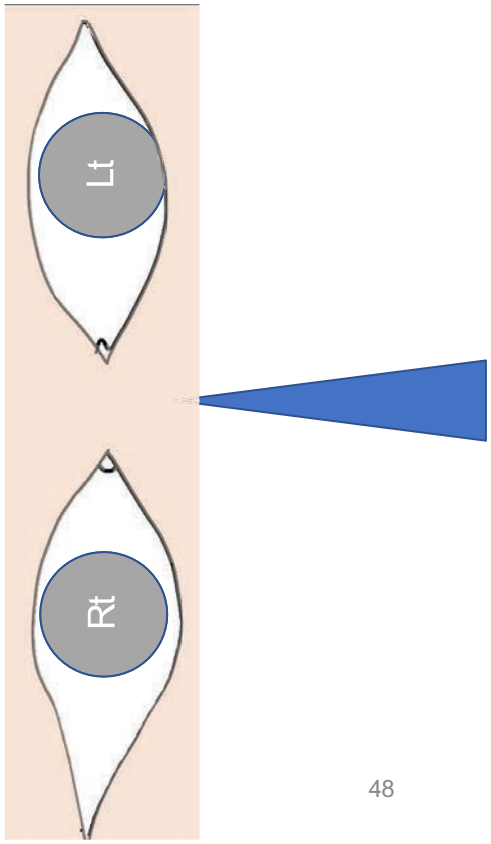
Ground

Pagnini-McCLure maneuver/ Supine head roll to right

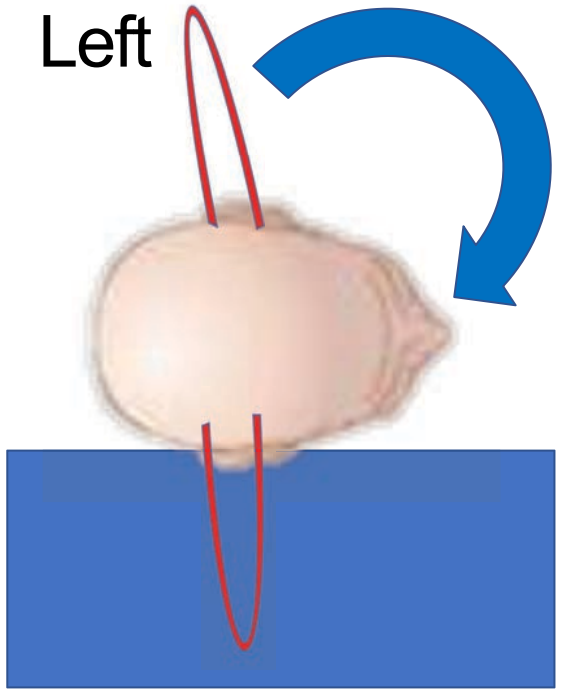


Beating towards the ground

Geotrophic nystagmus

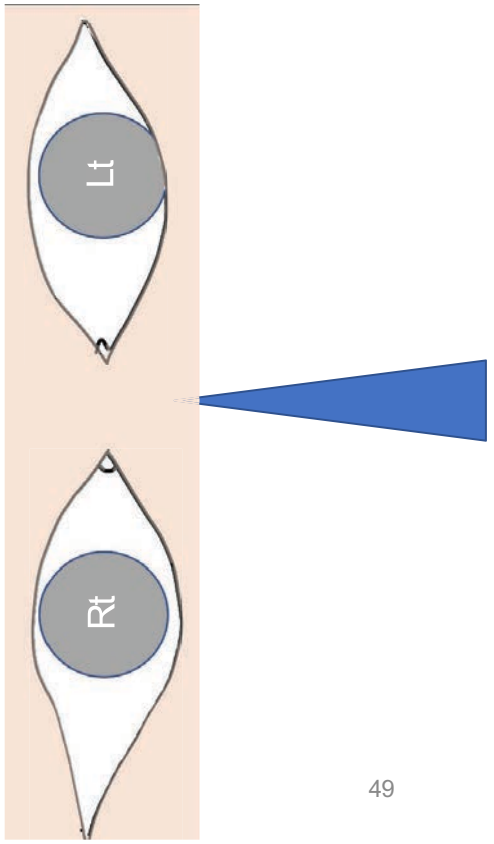


Pagnini-McCLure maneuver/ Supine head roll to right



Beating away
From the ground

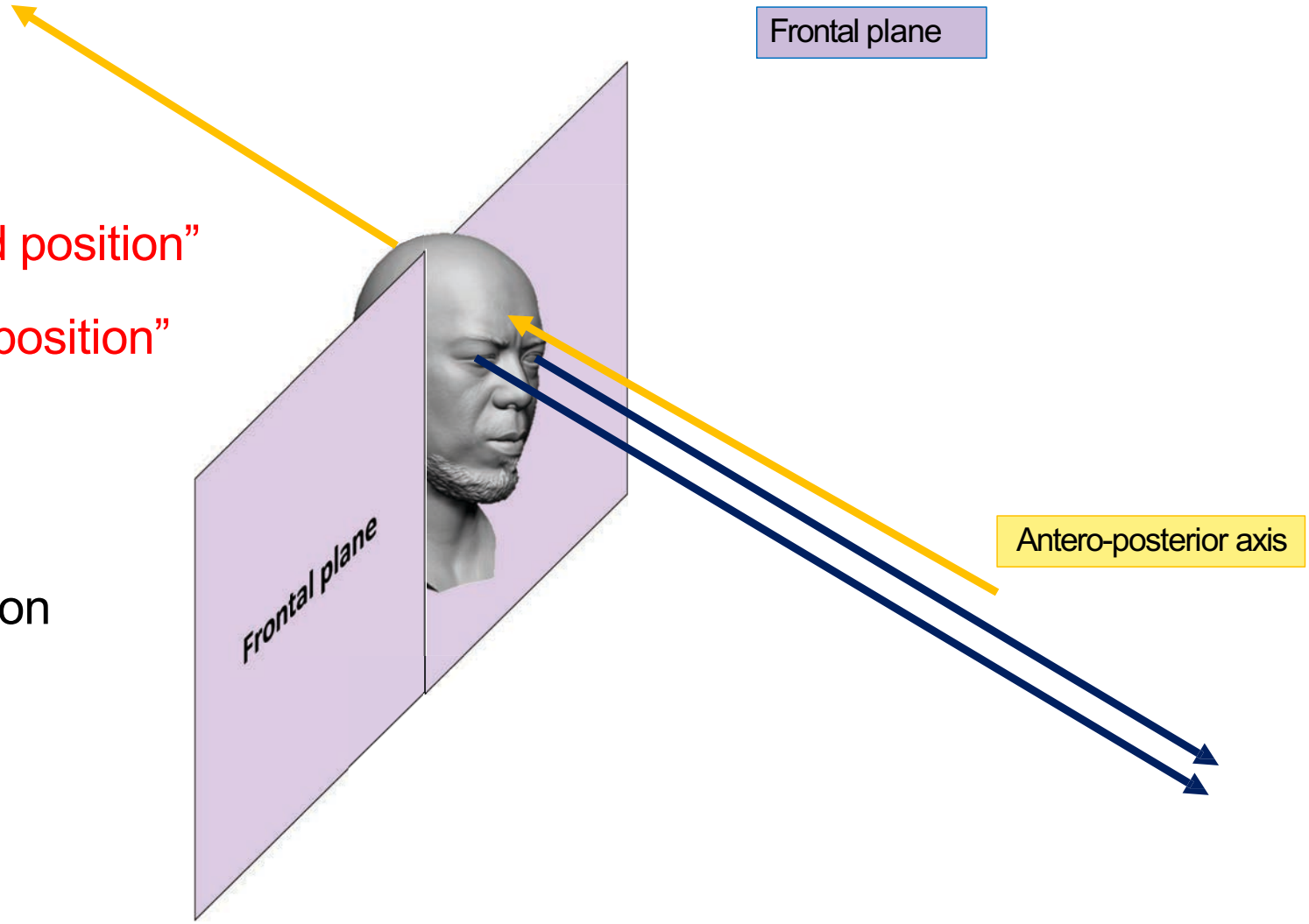
Apo-geotrophic
nystagmus



Straight-ahead position (Center Gaze)

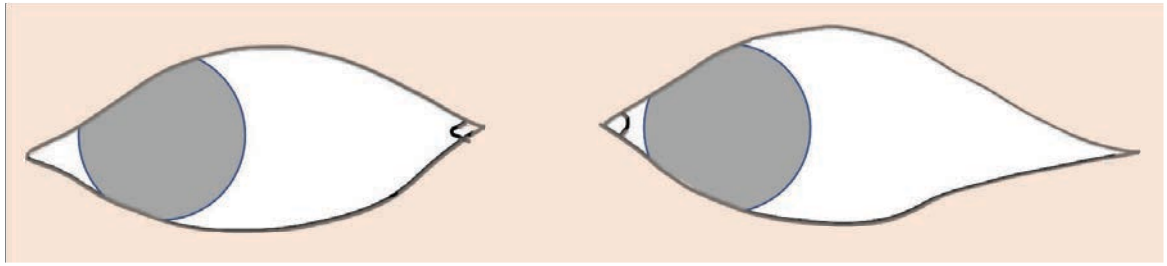
“Straight-ahead position”
or
“Center gaze position”

Primary position



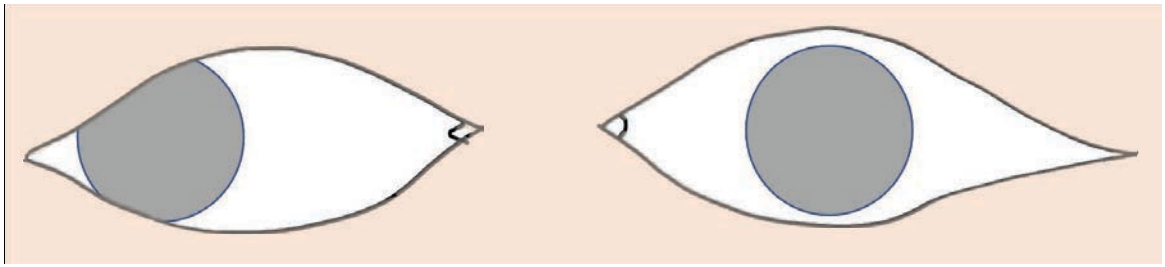
Monocular vs Binocular

Binocular



- **Acquired monocular blindness (nystagmus in blind eye)**
- Brainstem infarction (thalamus and upper midbrain)
- **Ictal nystagmus**
- **Internuclear and pseudointernuclear ophthalmoplegia**
- **Multiple sclerosis**
- **Nystagmus with monocular ophthalmoplegia (III,VI nerve palsy)**
- **Nystagmus with one eye absent**
- **Pseudonystagmus (lid fasciculations)**
- **Spasmus nutans**
- **Myasthenia gravis**
- **Restrictive orbitopathy**

Monocular



Abduction nystagmus

Adduction paresis

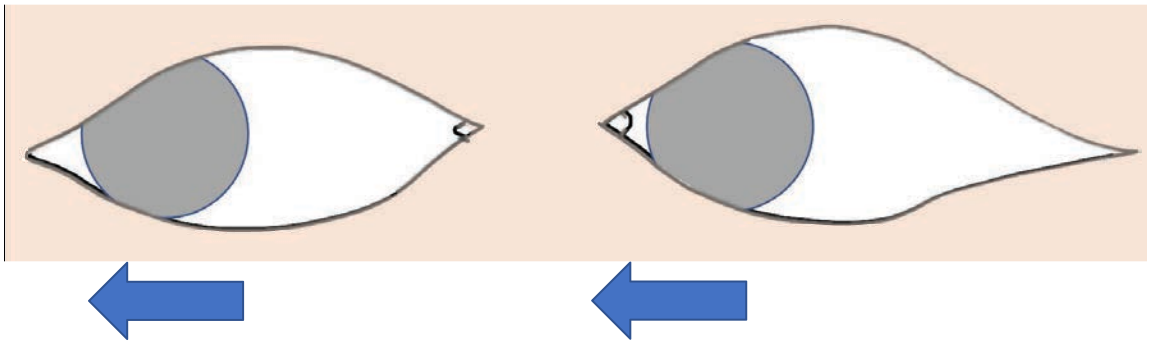
Conjugate vs Disconjugate

Nystagmus

Conjugate

In the same direction

With same velocity and amplitude



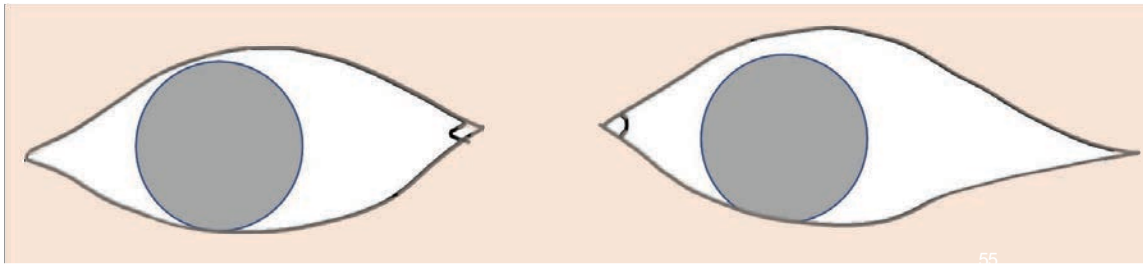
Dis-conjugate

In opposite direction

Disjunctive

With different velocity or amplitude

Dissociated

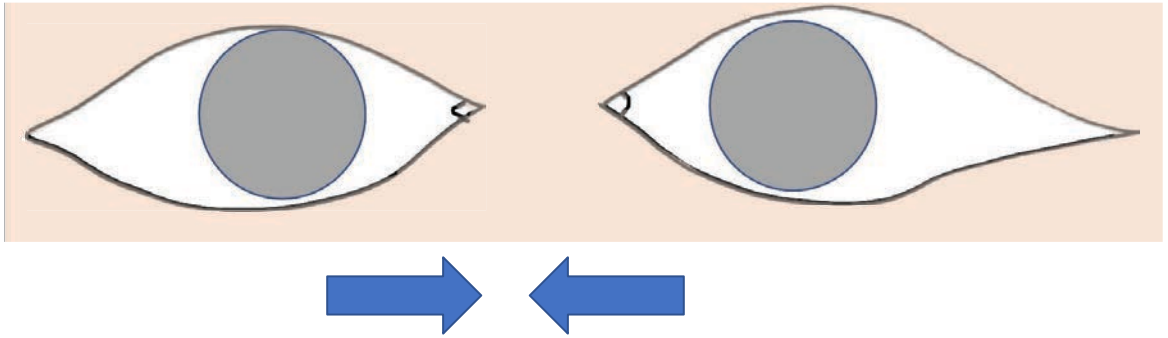


Dis-conjugate

Disjunctive

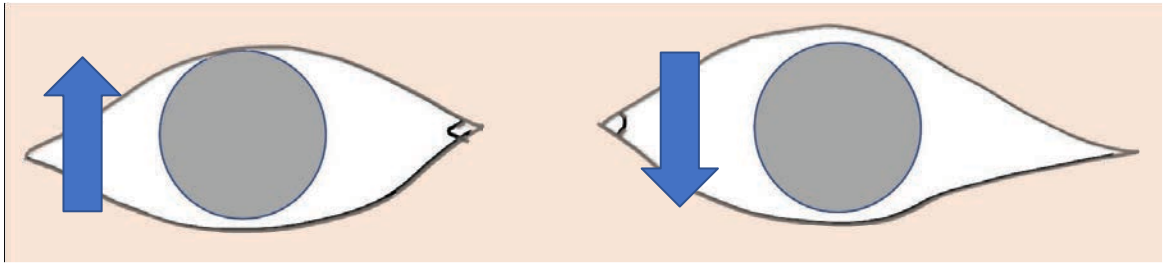
Horizontal

Convergence-divergence nystagmus



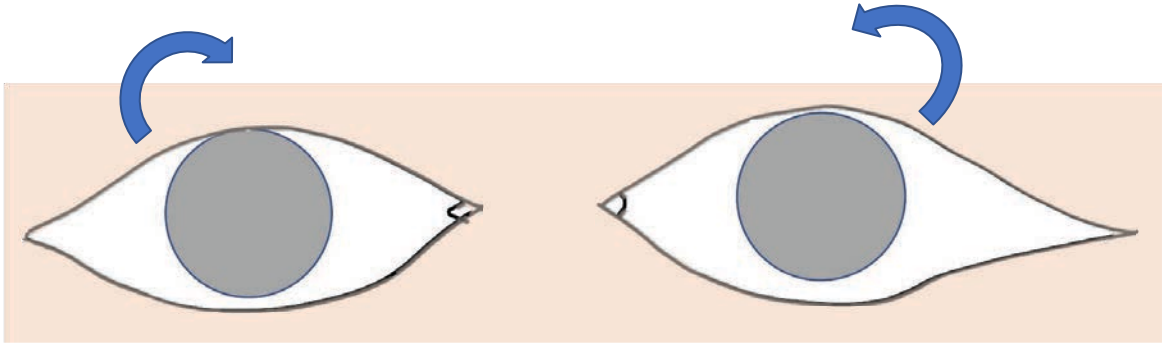
Vertical

Sea-saw nystagmus



Torsional

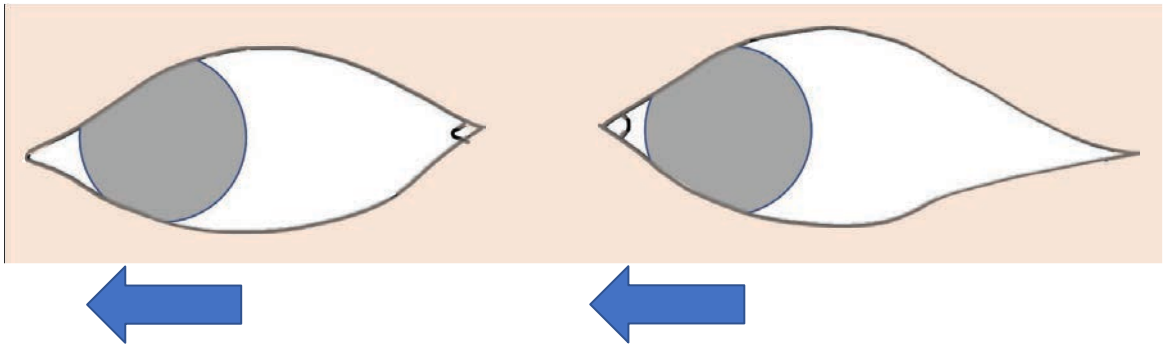
Cyclo-vergent



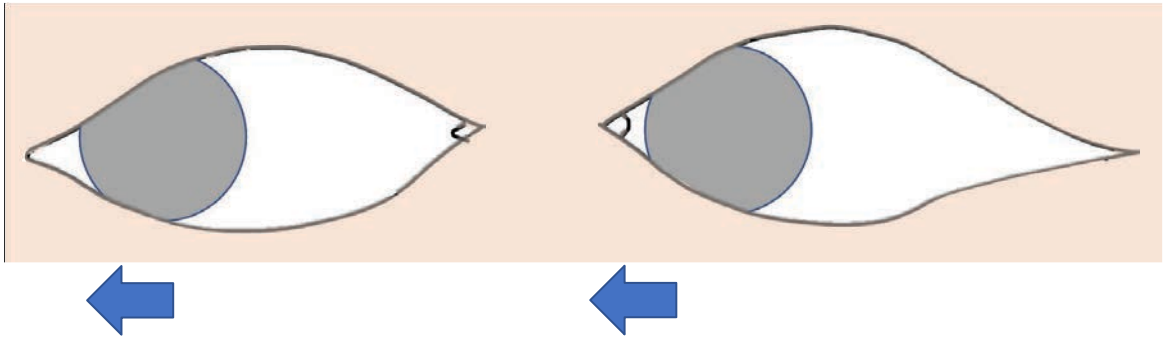
Slow phase velocity

Slow phase velocity

Fast



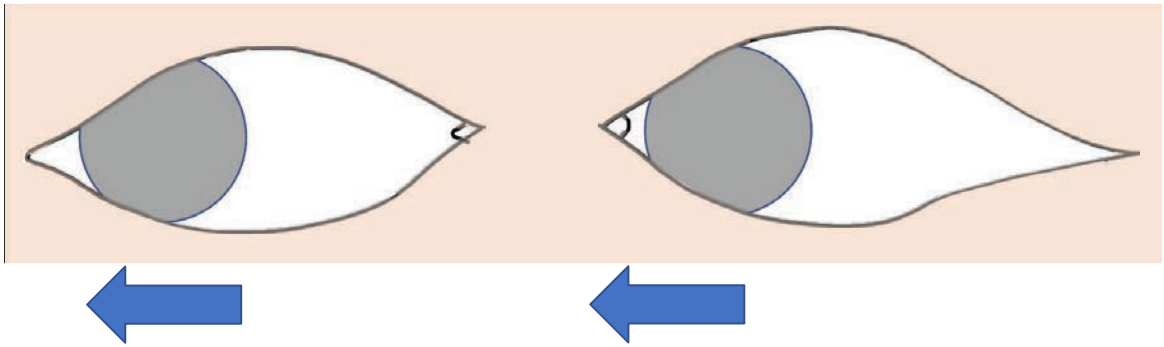
Slow



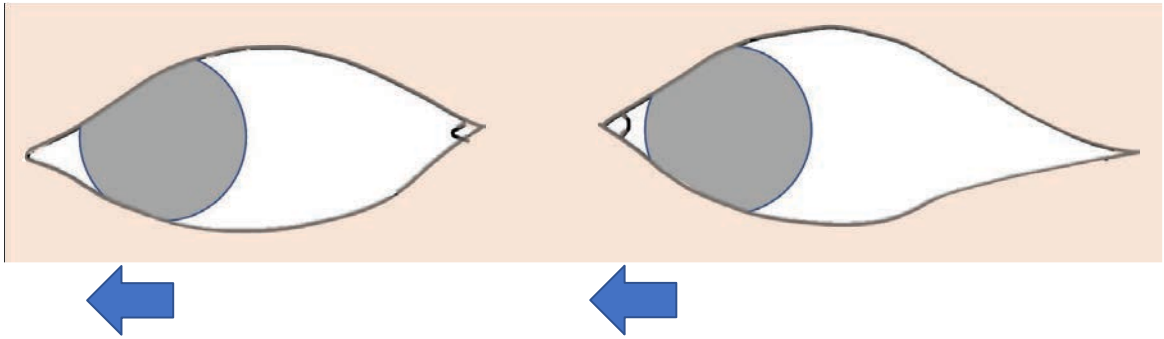
Frequency

Frequency (Beats per second)

Higher frequency

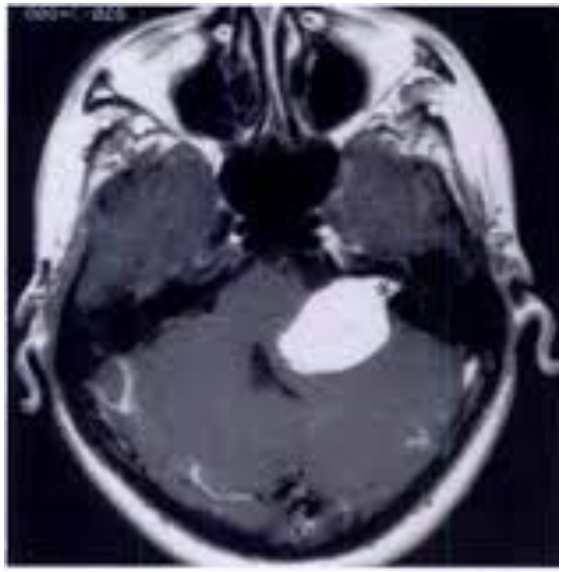


Lower frequency

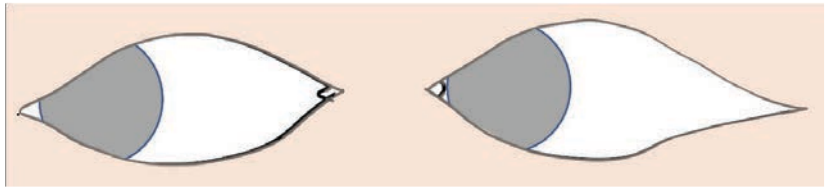


Amplitude

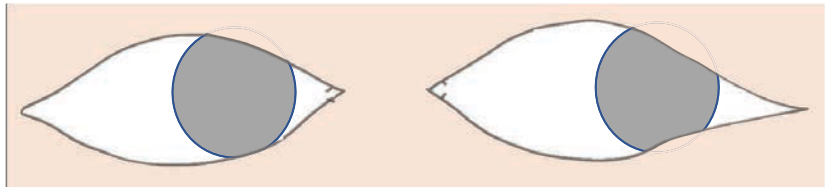
Fine and course nystagmus (Depends on amplitude)



Higher frequency
Short amplitude
Fine nystagmus



Low frequency
Large amplitude
Coarse nystagmus

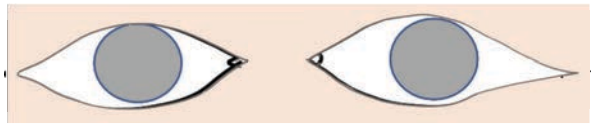


Brun's nystagmus

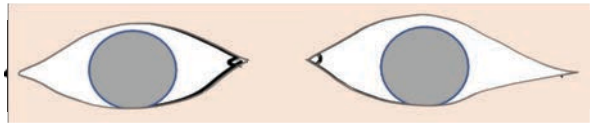
Spontaneous nystagmus

Spontaneous nystagmus:

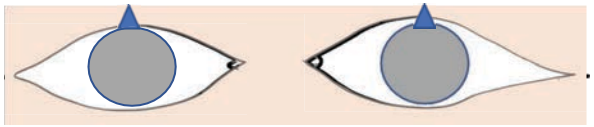
Spontaneous horizontal



Spontaneous vertical

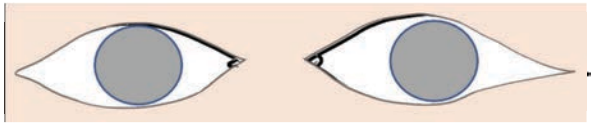


Spontaneous torsional



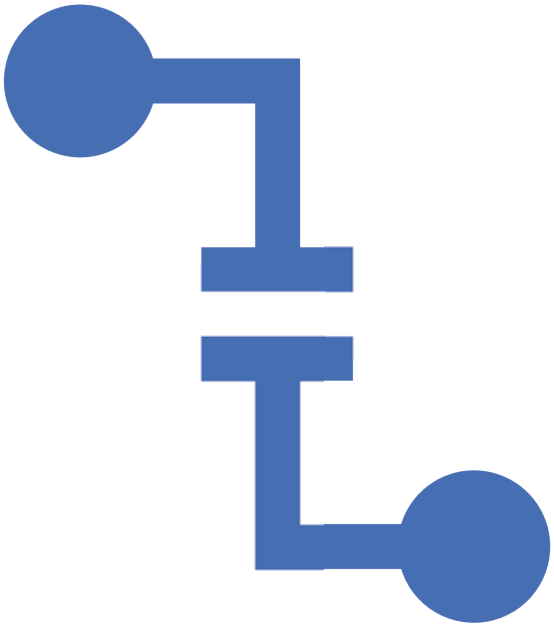
- **Straight-ahead (center) gaze position**
- **Head stationary**
- **Unprovoked**

Centre-gaze



Gaze-testing

- **No nystagmus**
- **Present**
- **Increased**
- **Decreased**



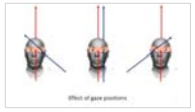
- Central mechanisms suppress the vertical and horizontal component as compared to the torsional component.

-Paul Brazis

Influencing factors

Influencing factors

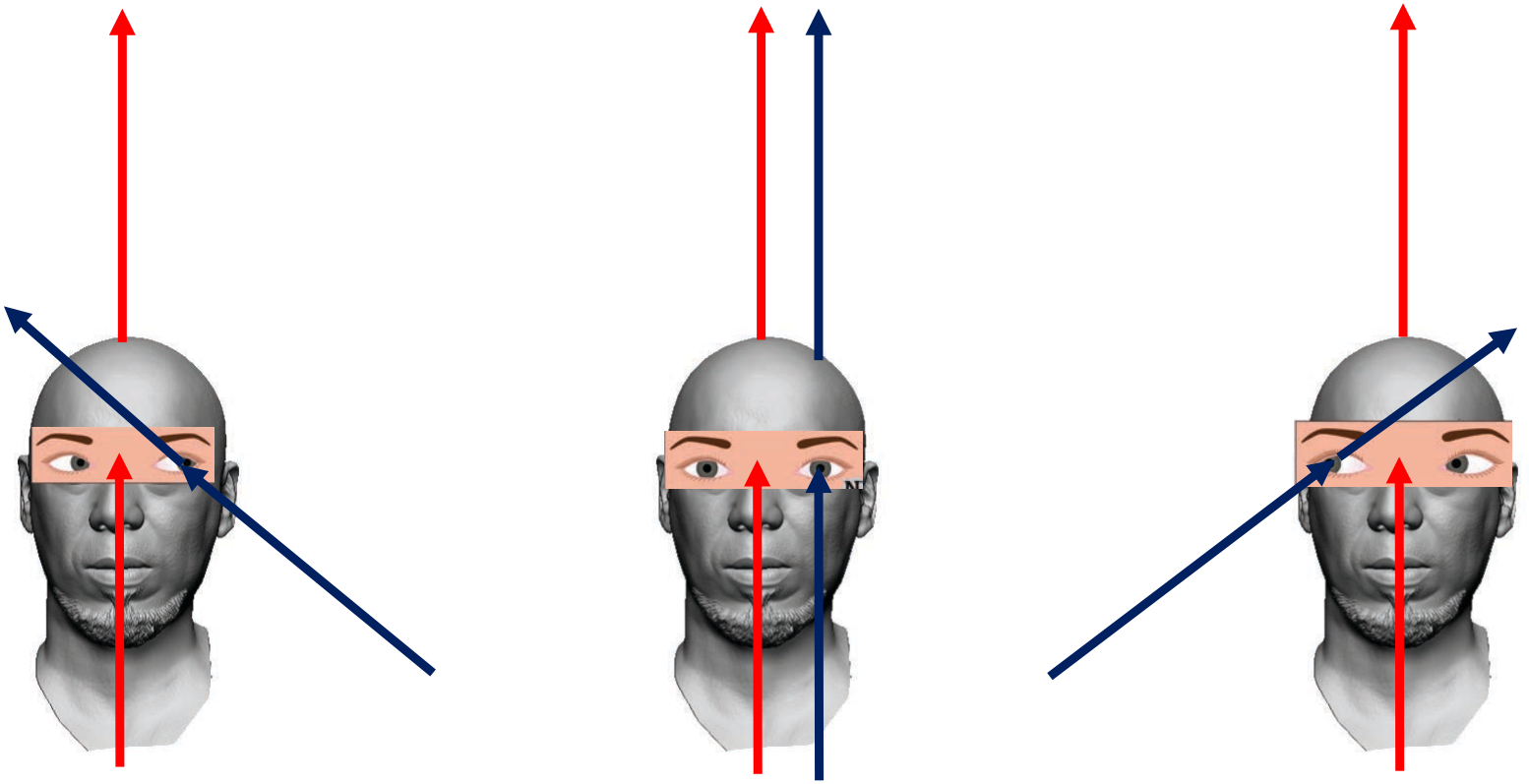
GAZE POSITIONS



VISUAL FIXATION

VERGENCE

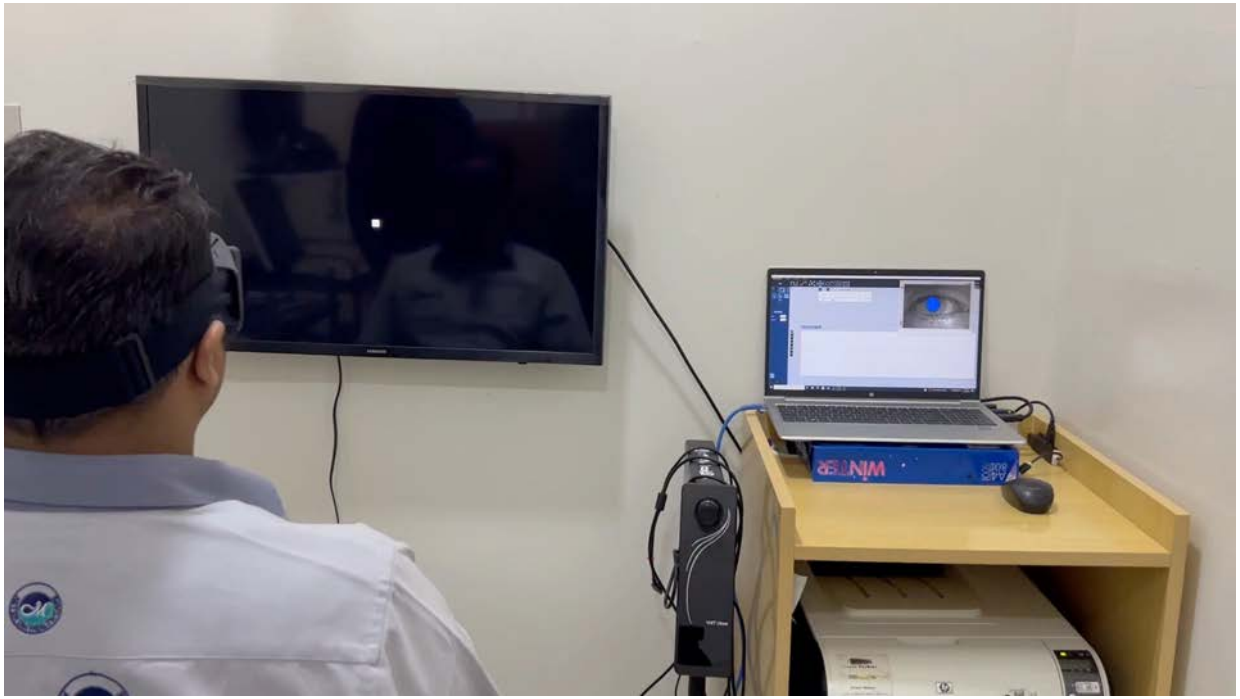
PATTERN RECOGNITION



Effect of gaze positions

Horizontal gaze testing

Vertical gaze testing



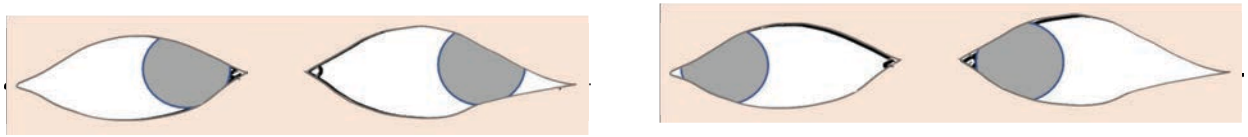
Without fixation

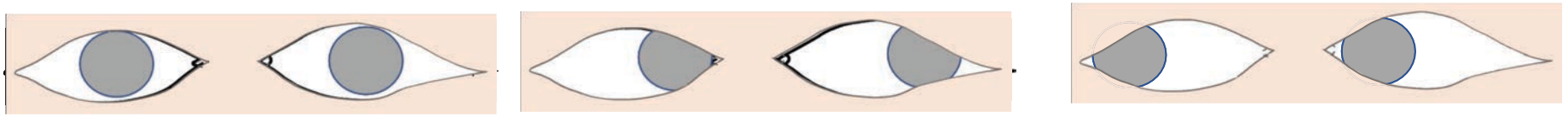
Gaze-evoked nystagmus

Centre-gaze

No nystagmus

Gaze-testing





Centre gaze + Gaze-evoked

Check for 90 seconds

Periodic alternating nystagmus

Central vs peripheral nystagmus

Direction of nystagmus

On right gaze

On left gaze

Peripheral

Unidirectional

Left beating

Left beating

Central

Bidirectional

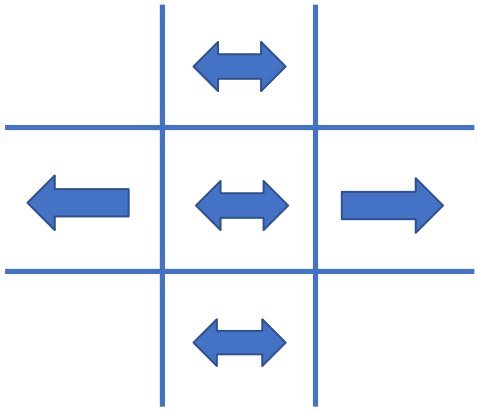
Cerebellar downbeat nystagmus syndrome

Right beating

Left beating

Dr Vishal Pawar, Personal dizziness registry. Aster clinic, International city, Dubai, 2019

Infantile nystagmus

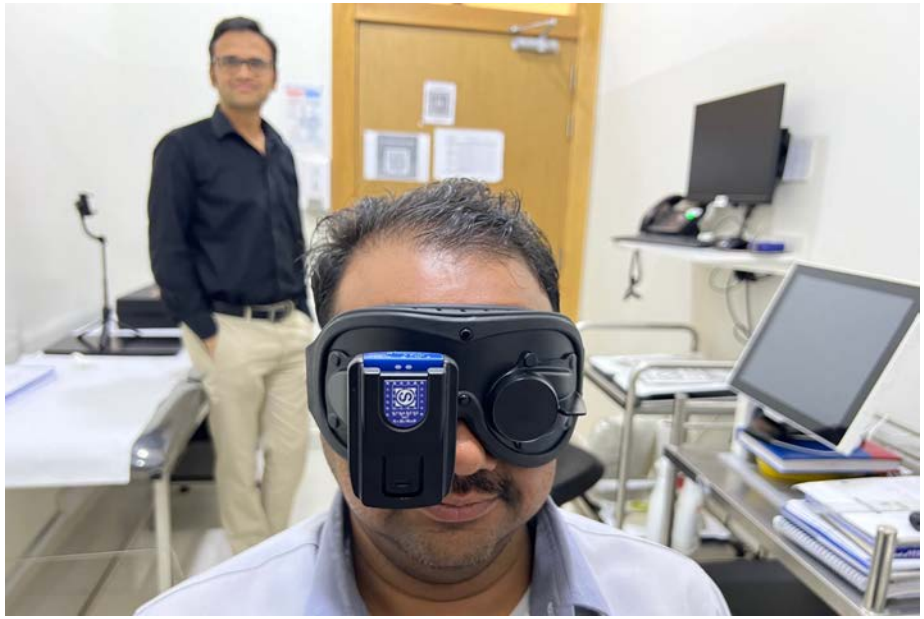


Dizziness registry, Dr Vishal Pawar, Aster clinic, Discovery Gardens Dubai. Informed consent obtained from the patient

Other methods I use to remove fixation

- VNG (Closing the visor)
- Seeing in dark
- Frenzel glassses
  - M-glasses
  - **Rohiwal glasses**

Removal of fixation



Signs

Central

- Visual fixation
- No inhibition

Peripheral

- Inhibits nystagmus

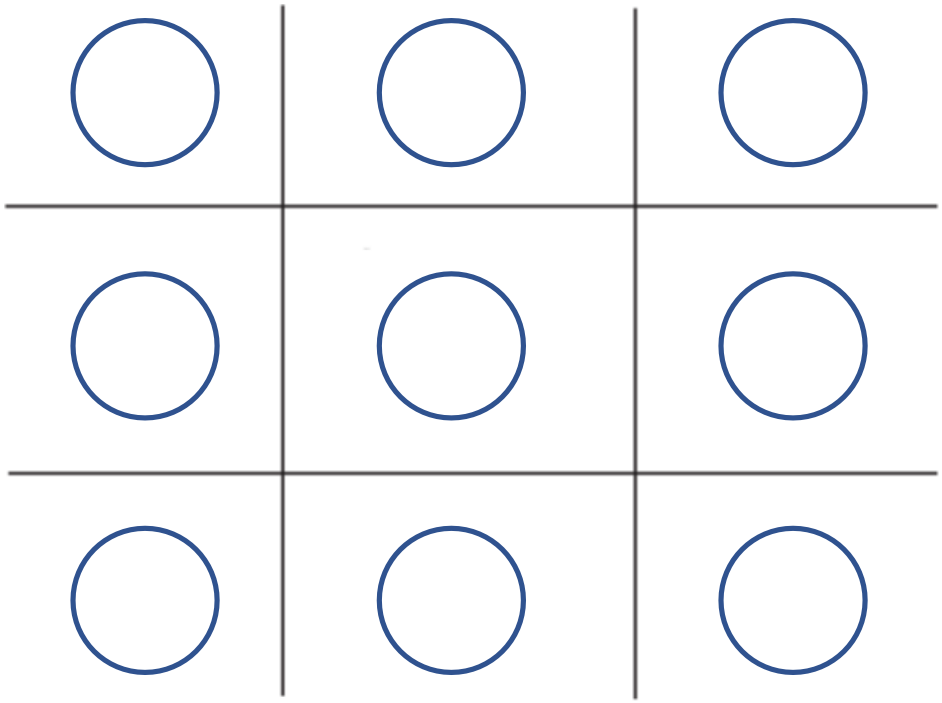
Effect of convergence

Suppresses infantile nystagmus.

May accentuate, suppress, or reverse the direction of vertical nystagmus.

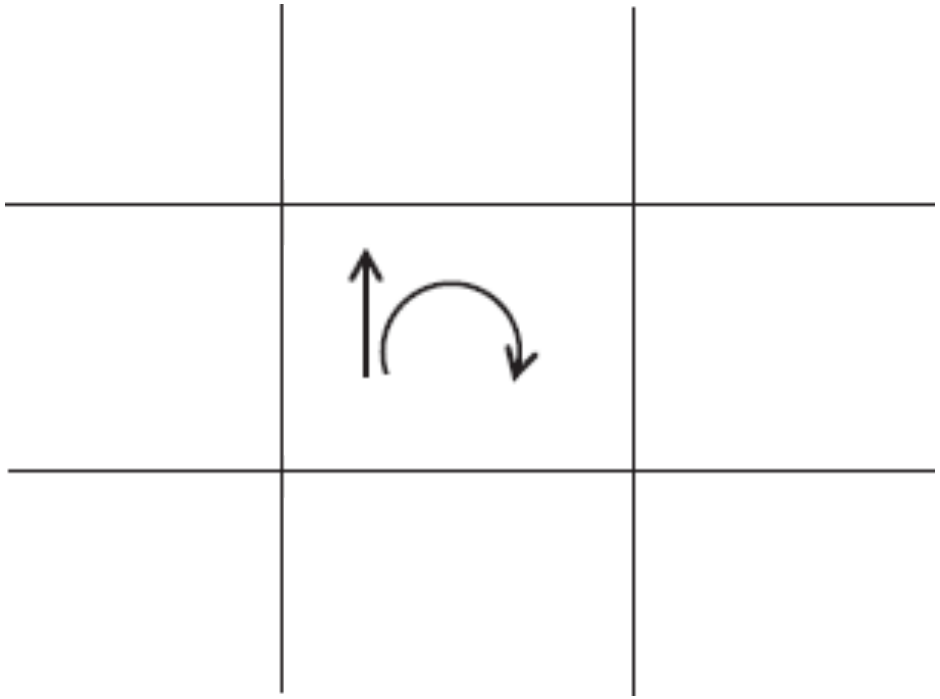
With fixation

No spontaneous nystagmus



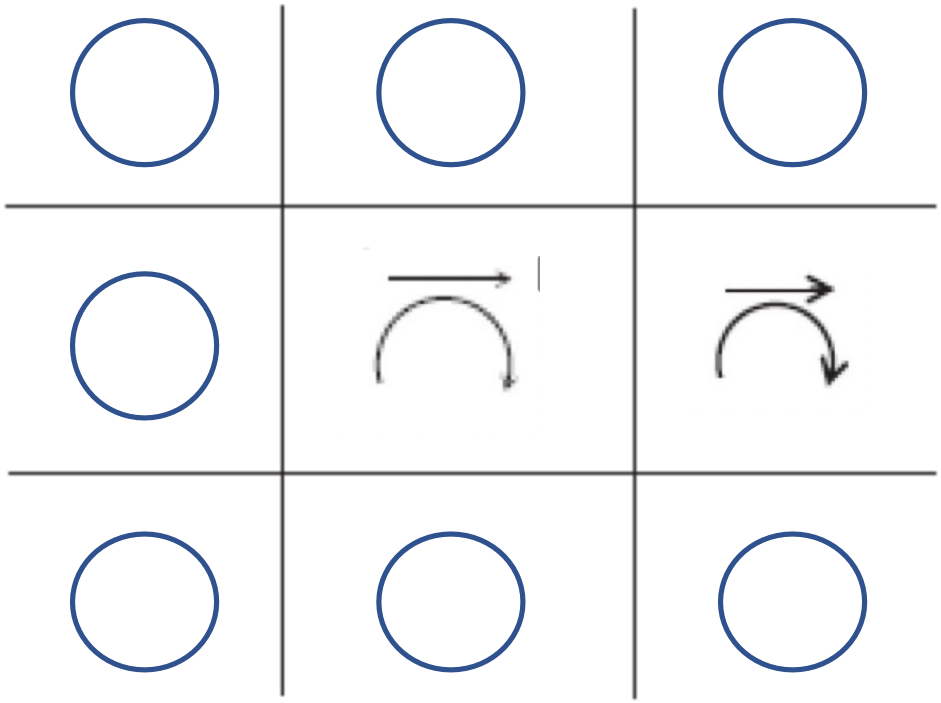
Positional testing

Left posterior canal BPPV



- No spontaneous nystagmus
- Torsional up beating in center gaze
- Up beating in right gaze
- Torsional on left gaze
- Increases in intensity on looking to fast phase (Follows Alexander's law)

With fixation

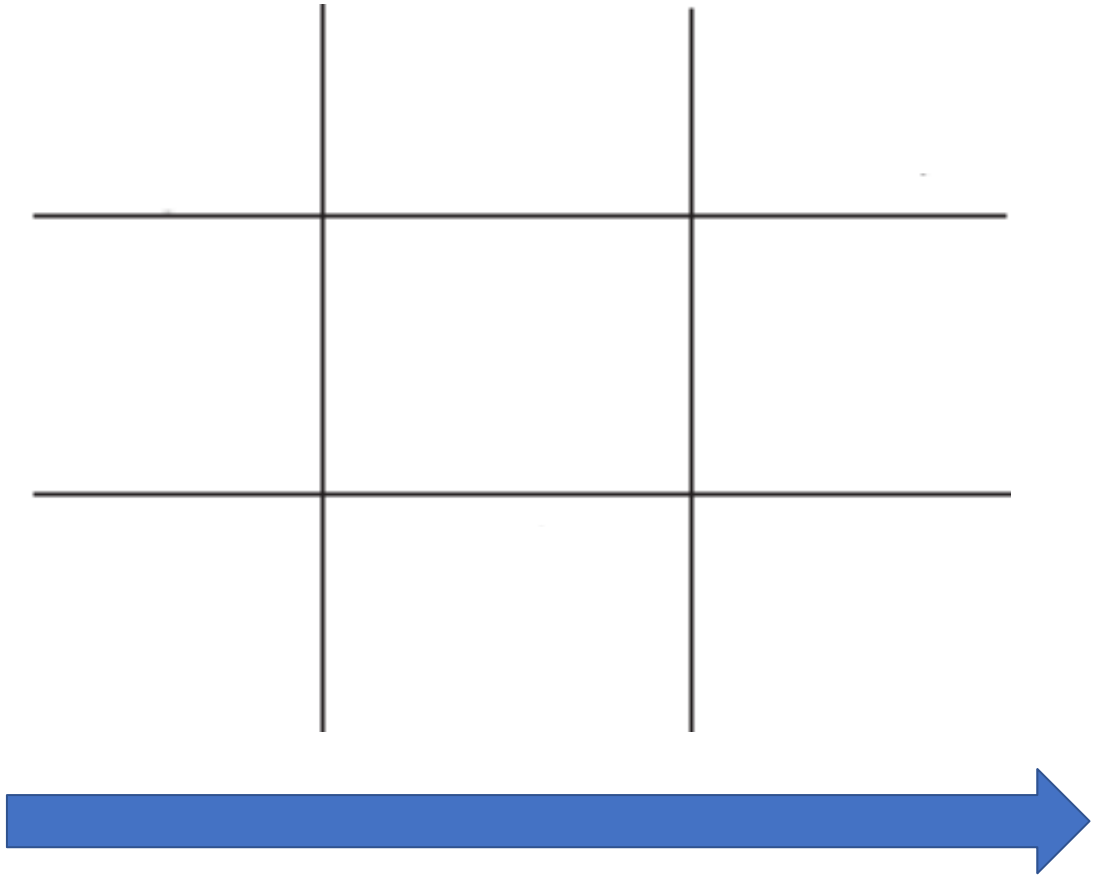


Greatest when gaze is directed toward the healthy ear

Is attenuated at central gaze

May be absent when gaze is directed toward the impaired ear

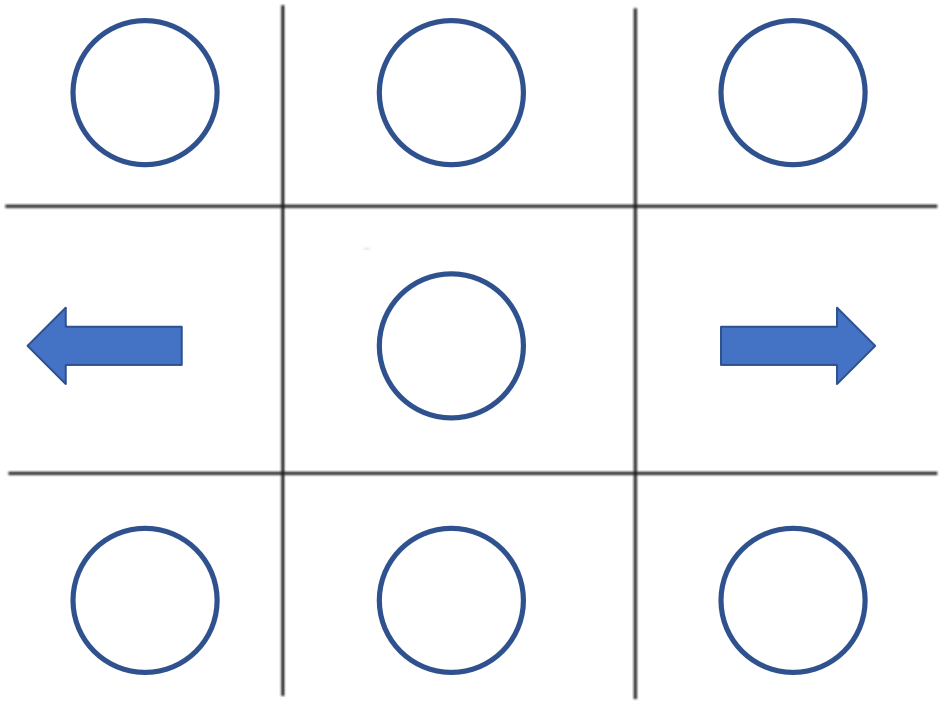
Without fixation



Unilateral vestibulopathy

- Enhancement without fixation
- Pronounced Horizontal torsional in center and vertical gazes
- Increases in intensity on looking to fast phase
- Decreases in intensity on looking to slow phase
- Follows Alexander's law
- Unidirectional

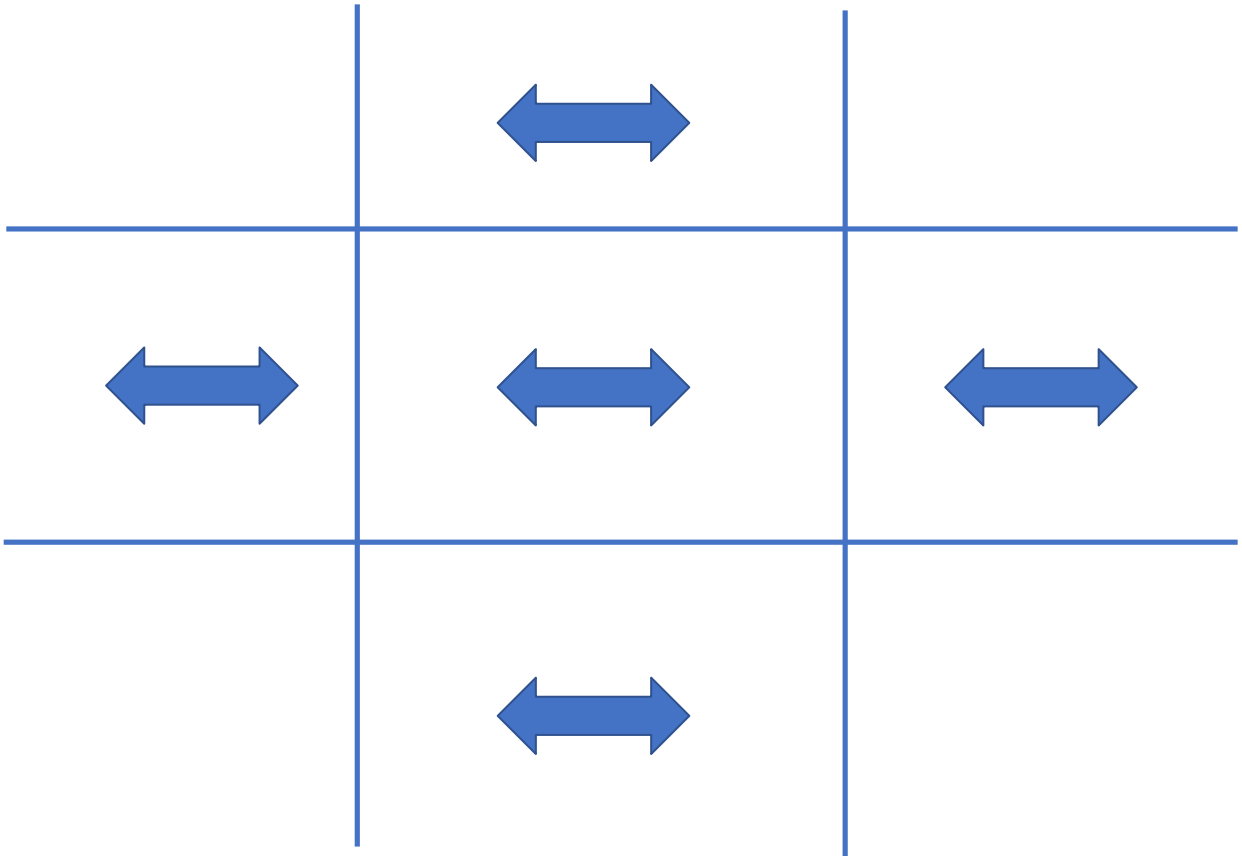
With fixation



No Center gaze nystagmus

Gaze-evoked nystagmus

With fixation



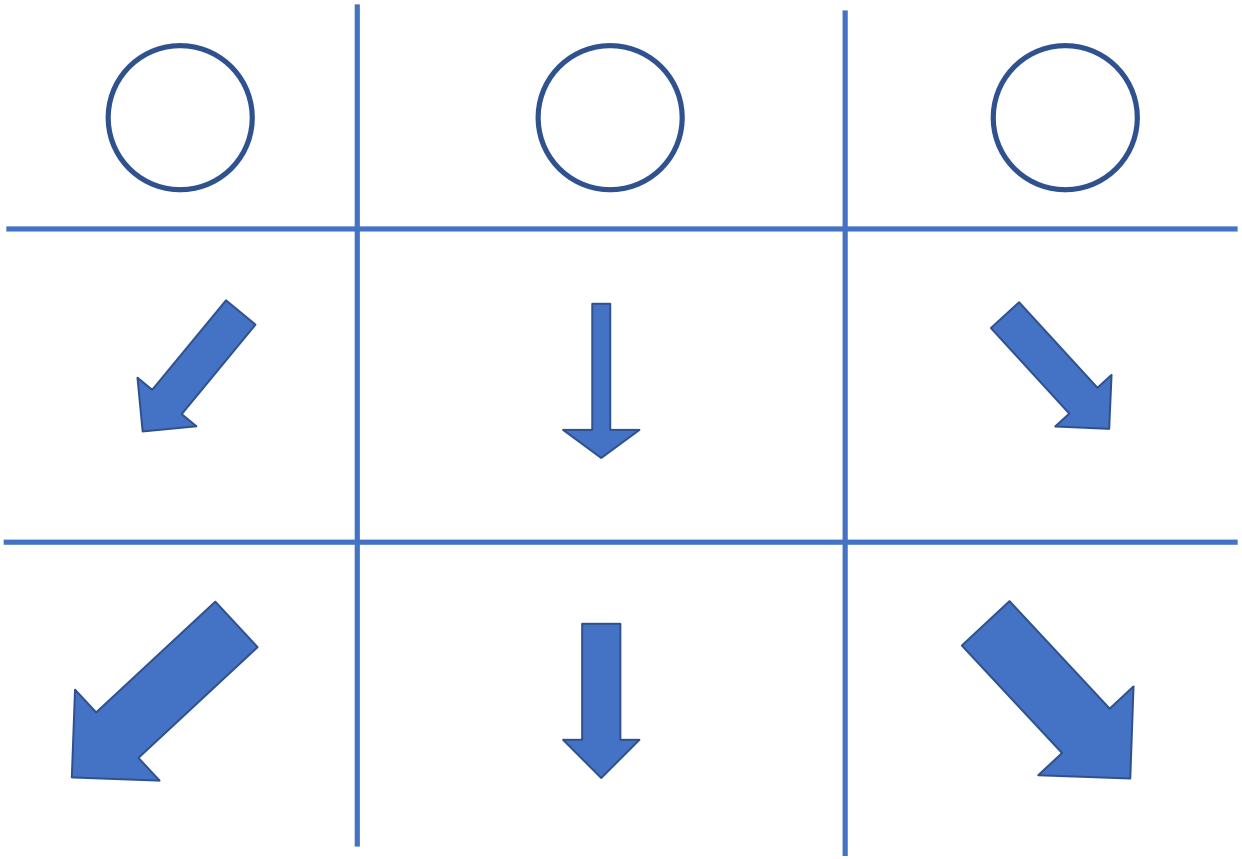
Infantile nystagmus

Horizontal in center gaze

Horizontal in lateral gazes

Horizontal in vertical gazes

With fixation



Central nystagmus

Down beating in center and down gaze

Side-pocket or oblique in right and left gazes and down gazes

Reference frame

All frames are equally valid

Selection of frame linked to mechanism of nystagmus

Specify the frame

Eyes

Head

Semicircular Canals

Earth

Central nystagmus

Rotational VOR

Posterior and lateral canal BPPV

Dizziness registry, Dr Vishal Pawar, Aster clinic, Dubai

Central nystagmus

- No suppression on fixation
- Gaze evoked nystagmus
- Pure variety of SPN in light

Provocative testing

Provocative testing

- Positional nystagmus
- Head-shaking induced nystagmus
- Cross-coupled nystagmus
- Sound-induced nystagmus
- Valsalva induced nystagmus
- Pressure induced nystagmus
- Vibration-induced nystagmus
- Hyperventilation induced nystagmus

Provocative testing

Central or peripheral

- Positional nystagmus
-  Head-shaking induced nystagmus
- Vibration-induced nystagmus
-  Hyperventilation induced nystagmus

Peripheral

- Sound-induced nystagmus
- Valsalva induced nystagmus
- Pressure induced nystagmus

Central

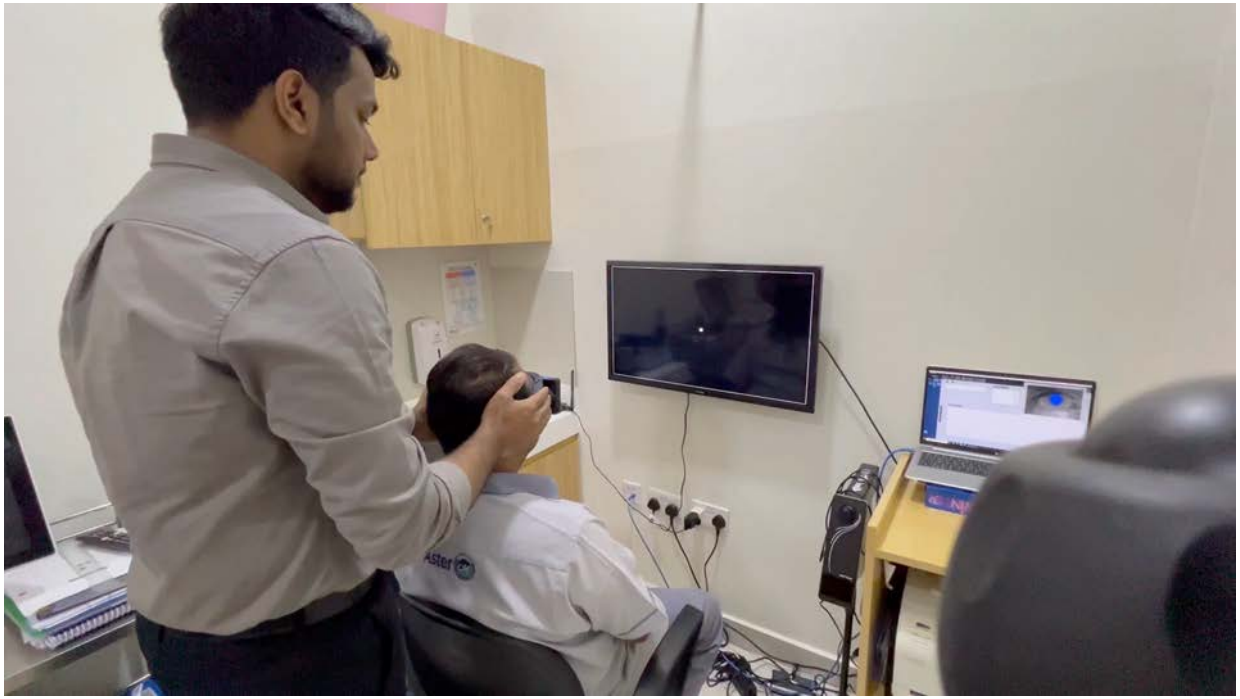
- Cross-coupled head shaking nystagmus

- Vestibular test battery
  - Gives findings
  - Not diagnosis
  - Aims to estimate the presence of a disorder rather than verify or quantify it
  - Indicates vestibular system abnormalities rather than lesion localization

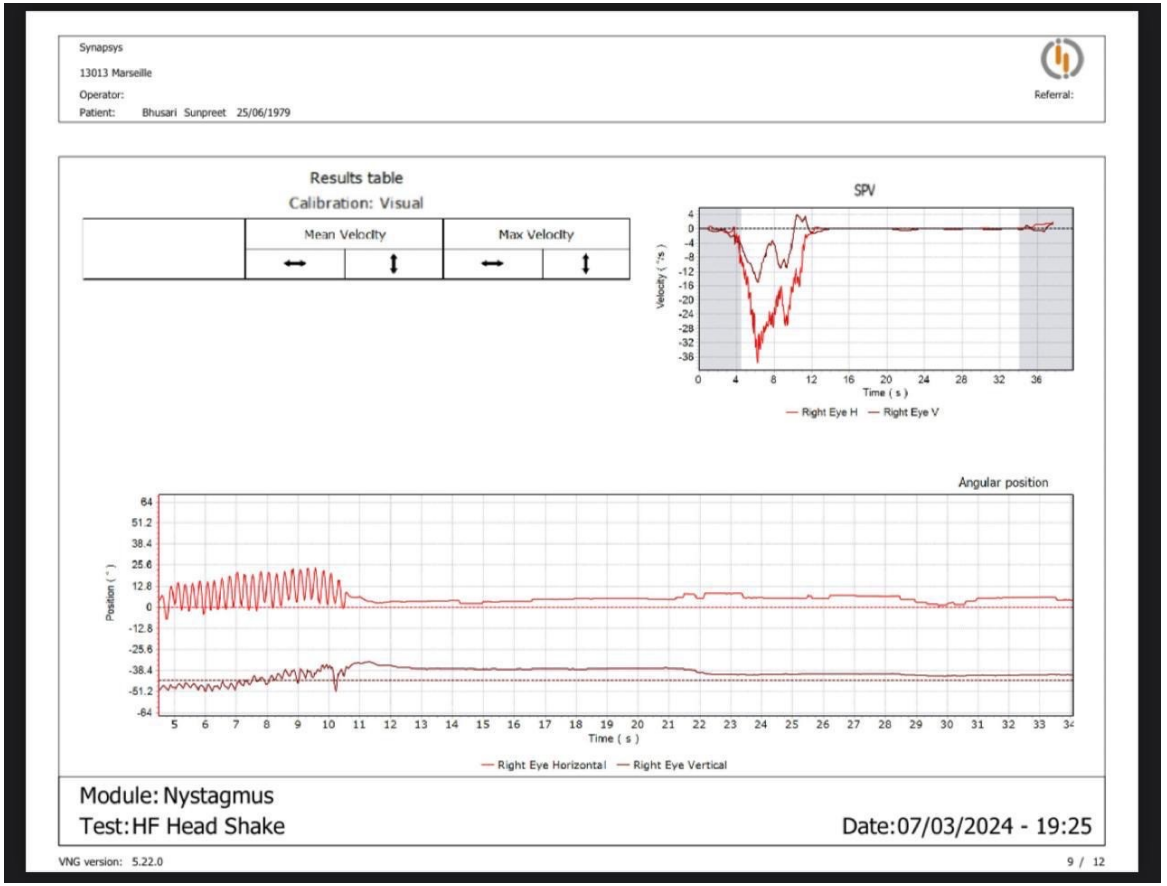
Head Shaking Test

Aspect	Details
Purpose	To screen for vestibular system abnormalities by inducing nystagmus through passive or active head shaking.
Method	The patient's head is rapidly moved left and right (yaw plane) by the examiner or by the patient themselves, usually for about 15-20 seconds, then suddenly stopped.
Observation	The examiner looks for nystagmus (involuntary eye movements), which is a sign of vestibular dysfunction.
Types of Nystagmus	**Post-head shaking nystagmus**: Nystagmus that occurs immediately after the cessation of head shaking. The direction and type of nystagmus can provide information about the affected side and nature of vestibular dysfunction.
Indications	Useful in diagnosing conditions like vestibular neuritis or Meniere's disease. Can help differentiate between central and peripheral causes of vertigo.

Head shaking test



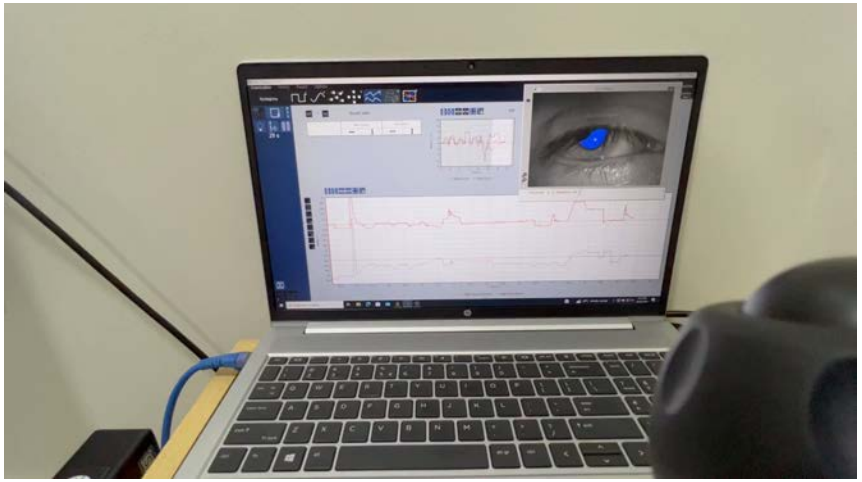
Head shaking test



Sensitivity and Specificity	Sensitivity (ability to detect the disorder when it is present) is moderate, and specificity (ability to confirm absence of the disorder when it is not present) is relatively high, but these values can vary depending on the patient population and the condition being tested.
Limitations	Sensitivity is lower in post-acute stages of vestibular impairment. Results can vary by age, diagnostic subgroup, and test position. Not useful for screening in cases of mild to moderate vestibular impairment.
Equipment	Typically requires no specialized equipment, though video nystagmography (VNG) or video-oculography (VOG) can be used to enhance detection of nystagmus.
Interpretation	A positive test (presence of nystagmus) suggests vestibular asymmetry or dysfunction. The direction of the nystagmus may indicate the affected side or type of dysfunction.
Clinical Use	Despite limitations, it's often used in combination with other vestibular tests for a more comprehensive evaluation. A negative test does not necessarily rule out vestibular dysfunction.

HVT is “the only test that unmasks unilateral vestibular disease without testing the dynamic properties of the vestibulo-ocular reflex”.

Prof Luigi Califano



Hyperventilation testing

Start of Hyperventilation:	• Rapid and deep breathing initiated.
Hypocapnia:	• Reduction in CO_2 levels in the blood due to excessive exhalation of CO_2.
Alkalosis:	• The decrease in CO_2 causes a rise in blood pH (alkalosis).
Biochemical Changes:	• Reduction in free plasma calcium levels (hypocalcemia).
Neuronal Effects:	• These biochemical changes lead to an excitatory effect on nervous tissue. • Altered function of voltage-dependent calcium channels in neurons.
Vestibular System Response:	• Excitatory effect on the vestibular system is observed. • Metabolic changes affect the vestibular nuclei and the balance of signals. • The function of partially compensated vestibular asymmetry is disrupted.
Central Compensation Modulation:	• Hyperventilation may interrupt central compensation for peripheral vestibular asymmetry, revealing or exacerbating nystagmus.
Manifestation of HVIN:	• Depending on the underlying vestibular status, HVIN can be: • **Paretic**: Enhancing existing spontaneous nystagmus. • **Excitatory**: Reducing, inhibiting, or reversing spontaneous nystagmus.
End of Hyperventilation:	• Return to normal breathing patterns may revert the temporary changes in nystagmus caused by hyperventilation.

Hyperventilation Test

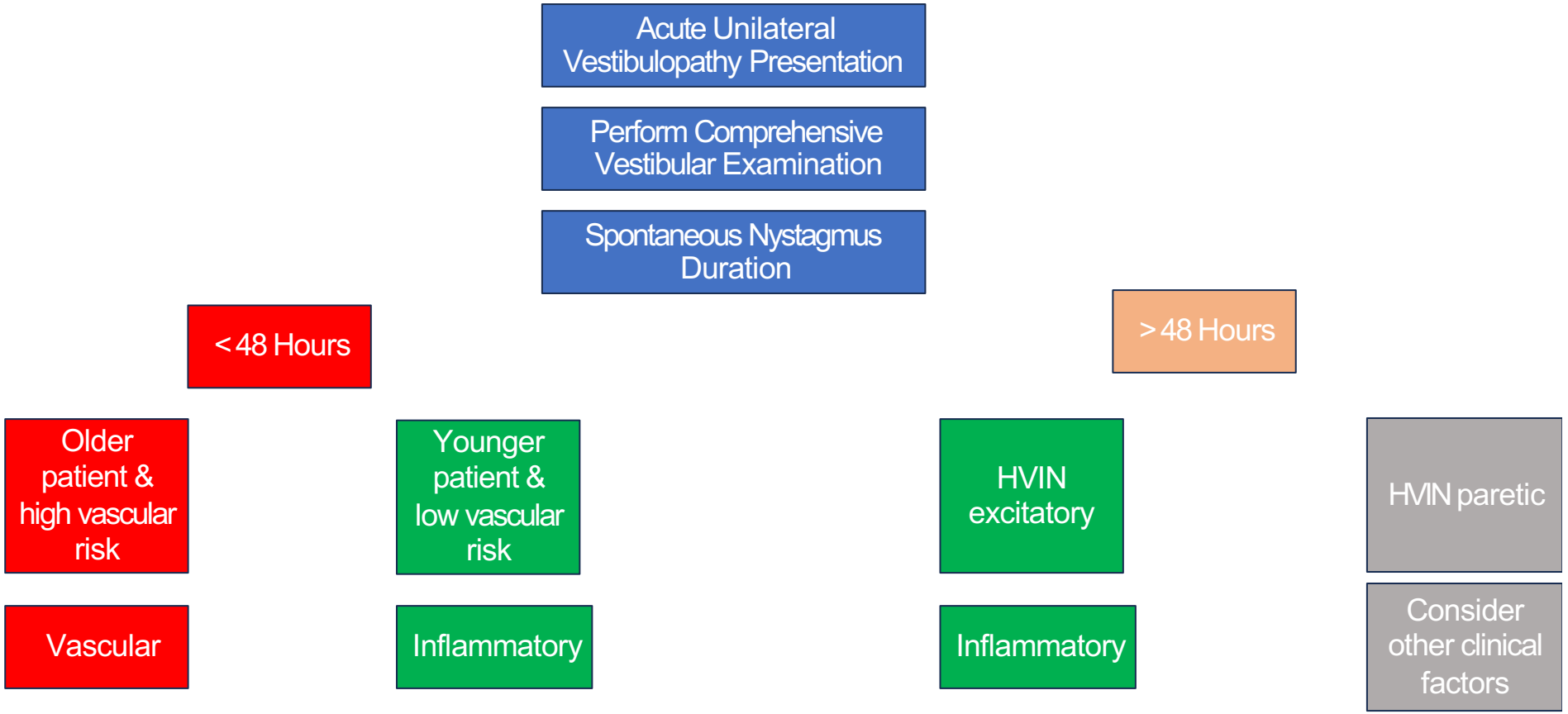
1. Utility ：The HVT is widely used in bedside examinations of vestibular patients, capable of revealing latent nystagmus in both central and peripheral vestibular diseases.

2. Incidence of HVIN ：In the largest study by Prof Luigi, HVIN was observed in 21.9% of vestibular patients, indicating its significant presence across various vestibular disorders.

3. Differential Diagnostic Value ：HVIN patterns and incidences help differentiate between vestibular neuritis and neuroma of the 8th cranial nerve, as well as in some central vestibular diseases.

4. Variation Across Conditions ：HVIN was more frequently detected in retro-cochlear vestibular diseases (e.g., acoustic neuroma). It can also be seen in end-organ vestibular diseases (e.g., Benign Paroxysmal Positional Vertigo - BPPV).

5. Patterns of HVIN ：In acute vestibular neuritis, three specific HVIN patterns were identified: paretic, excitatory, and strongly excitatory, with the excitatory patterns being time-dependent and transitioning to paretic patterns.



Can hyperventilation test and duration of spontaneous nystagmus help differentiate between vascular and inflammatory aetiology of acute unilateral vestibular deficit?

Luigi Califano, Giulia Locatelli, Maria Grazia Melillo

ACTA OTORHINOLARYNGOLOGICA ITALICA 2022;42:560-568

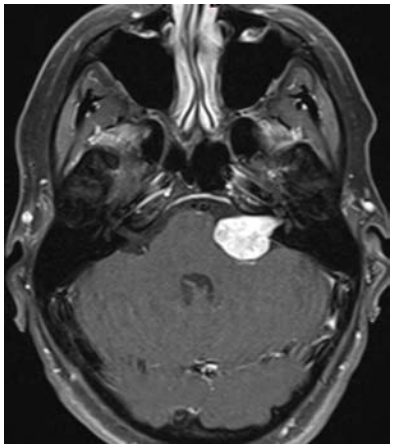
6. High Incidence in Specific Disorders : Notably high incidences of HVIN were found in conditions like pre-surgery acoustic neuroma (91.7%) and acute vestibular neuritis (72.2%).

7. Role in Central Vestibular Diseases : HVIN was frequently detected in vestibular migraine, demyelinating diseases like multiple sclerosis (75%) and cerebellar diseases (72.7%), indicating its utility beyond peripheral vestibular disorders.

8. Contribution to Comprehensive Evaluation : The findings from HVT should be integrated into a broader context of vestibular and auditory system examination for a comprehensive understanding of the patient's condition.

9. HVIN and functional dizziness: Dizziness without specific nystagmus is common

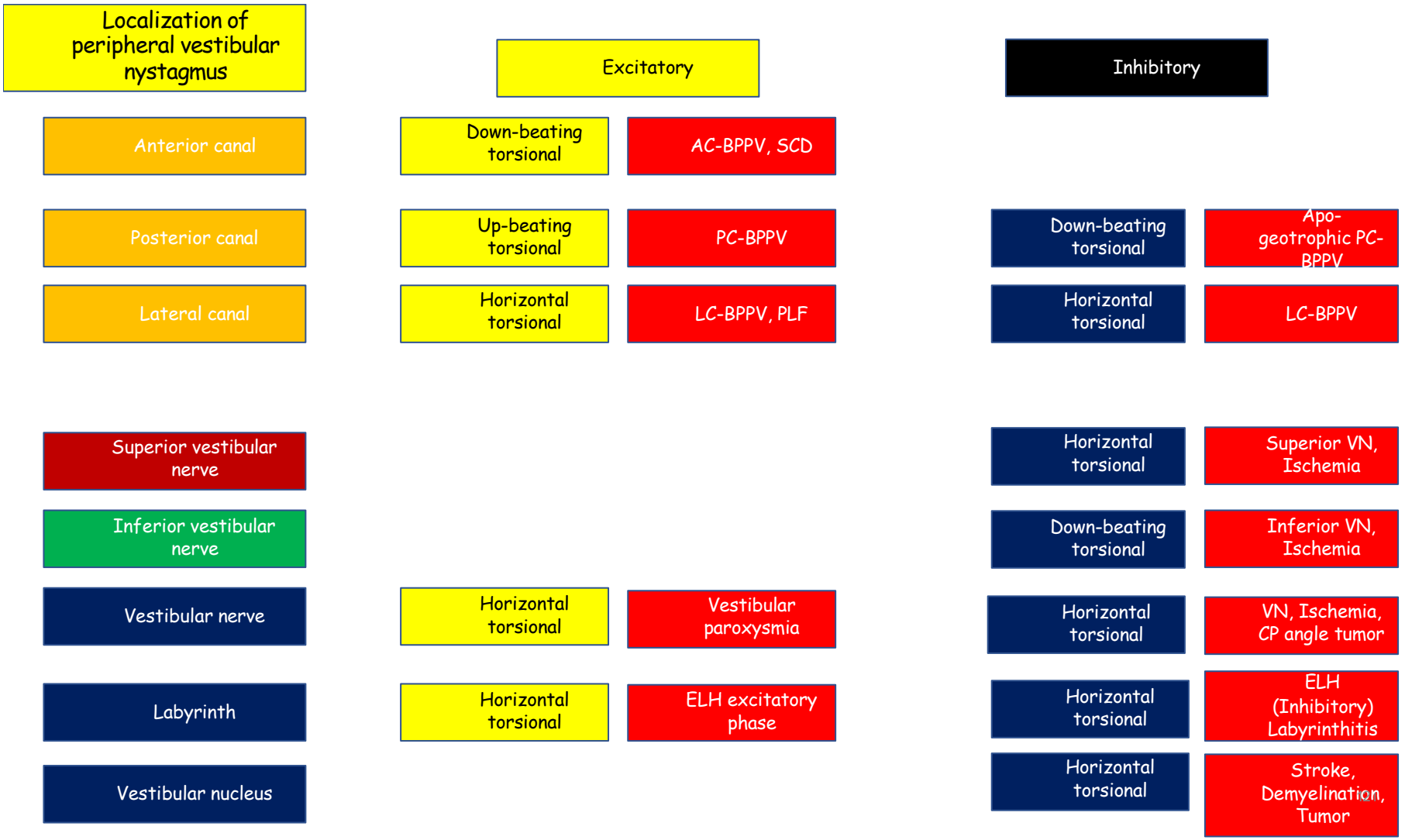
10. Clinical Implications and Recommendations : The presence of HVIN suggests the need for further diagnostic investigations, such as MRI with gadolinium, especially upon suspicion of acoustic neuroma or central neurological diseases, highlighting the test's clinical significance.



HVIN in various conditions

Acute Vestibular Neuritis	**Reinforced, inhibited, or reversed spontaneous nystagmus**,
Schwannoma of the Eighth Cranial Nerve	Indicative of demyelination or nerve fiber continuity.
'Third Window' Syndromes	Hyperventilation-induced intracranial hypotension can cause excitement in the superior canal, resulting in specific nystagmus patterns.
Multiple Sclerosis	Inhibit or evoke different types of nystagmus
Cerebellar Diseases	Hyperventilation may uncover downbeat nystagmus in cerebellar diseases
Other Vestibular Diseases	The hyperventilation test does not yield significant data in conditions like BPPV, Meniere's disease, or vascular diseases but is seldom observed in chronic subjective dizziness.

Peripheral vestibular nystagmus


Localization of peripheral vestibular nystagmus
Excitatory
Inhibitory
Anterior canal
Down-beating torsional
AC-BPPV, SCD
Posterior canal
Up-beating torsional
PC-BPPV
Down-beating torsional
Apo-geotrophic PC-BPPV
Lateral canal
Horizontal torsional
LC-BPPV, PLF
Horizontal torsional
LC-BPPV
Superior vestibular nerve
Horizontal torsional
Superior VN, Ischemia
Inferior vestibular nerve
Down-beating torsional
Inferior VN, Ischemia
Vestibular nerve
Horizontal torsional
Vestibular paroxysmia
Horizontal torsional
VN, Ischemia, CP angle tumor
Labyrinth
Horizontal torsional
ELH excitatory phase
Horizontal torsional
ELH (Inhibitory) Labyrinthitis
Vestibular nucleus
Horizontal torsional
Stroke, Demyelination, Tumor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SPEAKER

Dr. Vishal Pawar

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Thank you

neurovishal@gmail.com

+971553377067