NEUROLOGY OF HUMAN BALANCE

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Human Balance

Vestibular System
Cerebellar System
Proprioceptive System
Visual & Ocular Motor Syst.
Extrapyramidal System
Cerebral Motor System

Central Integrator ?

? Vestibular Nucleus

?Tuberomammilary Nucleus

Vertigo Dizziness Balance Disorders

Mechanism :

Sensory Mismatch

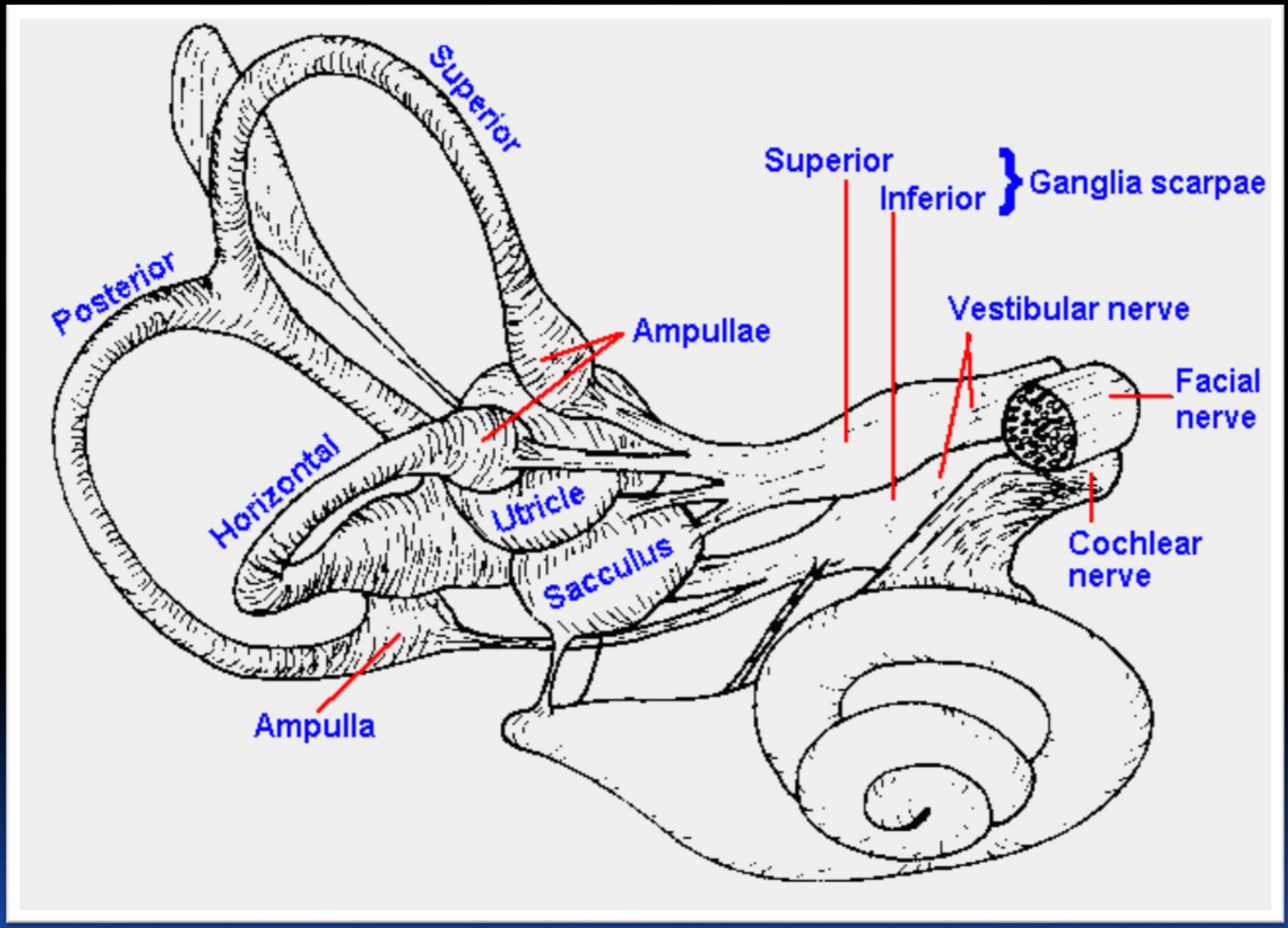
CNS Misinterpretation

Causes :

½ Vestibular

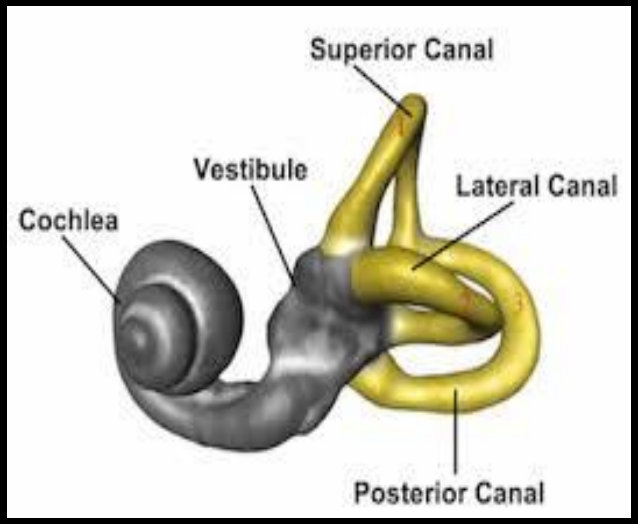
¼ Psychogenic

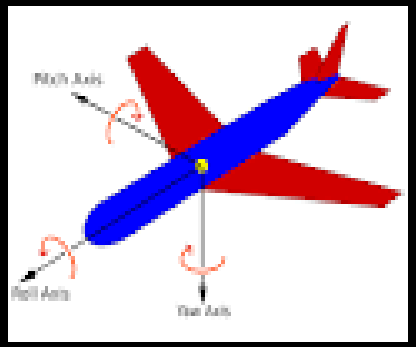
¼ Neurological & Mixed

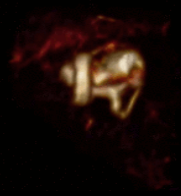


The human labyrinth

Anatomy of Balance

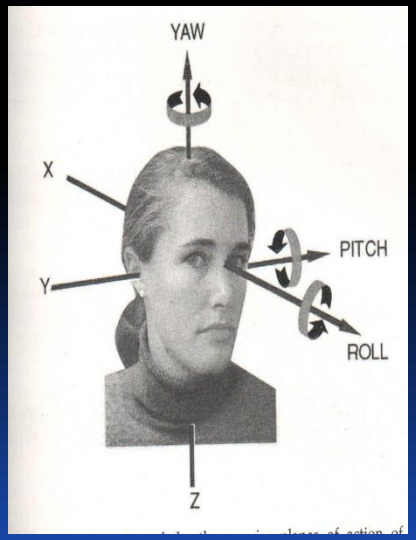


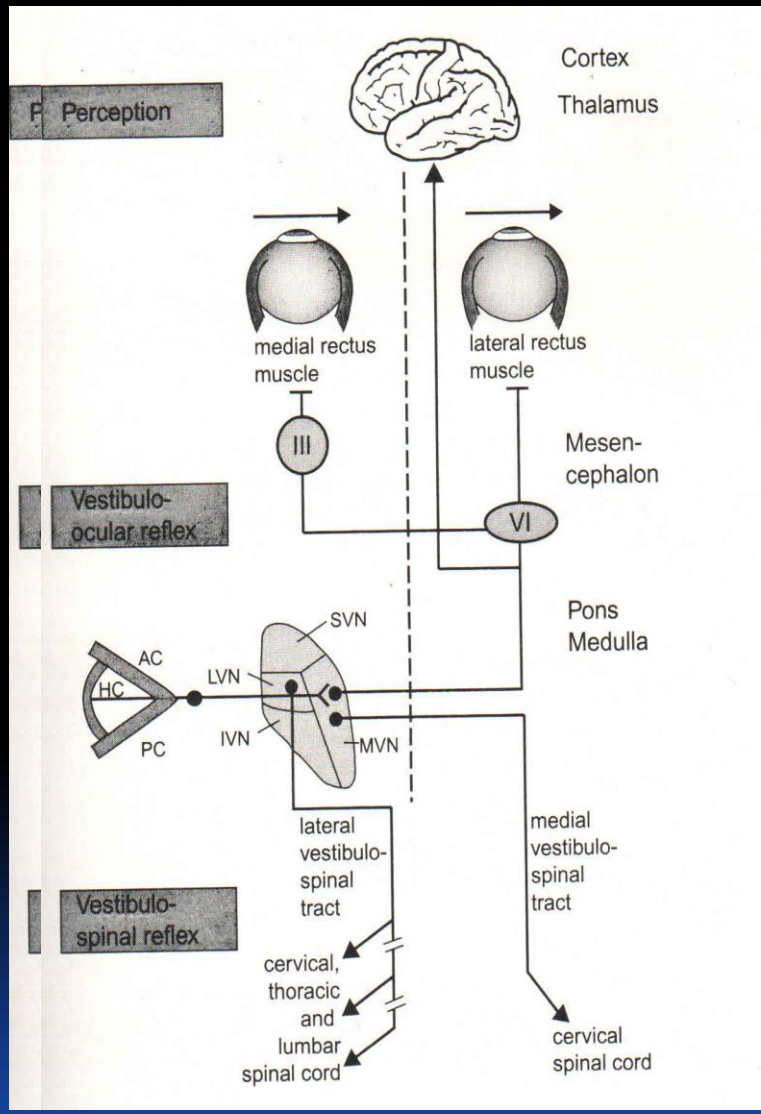




The semicircular canals are functionally paired and sense rotation

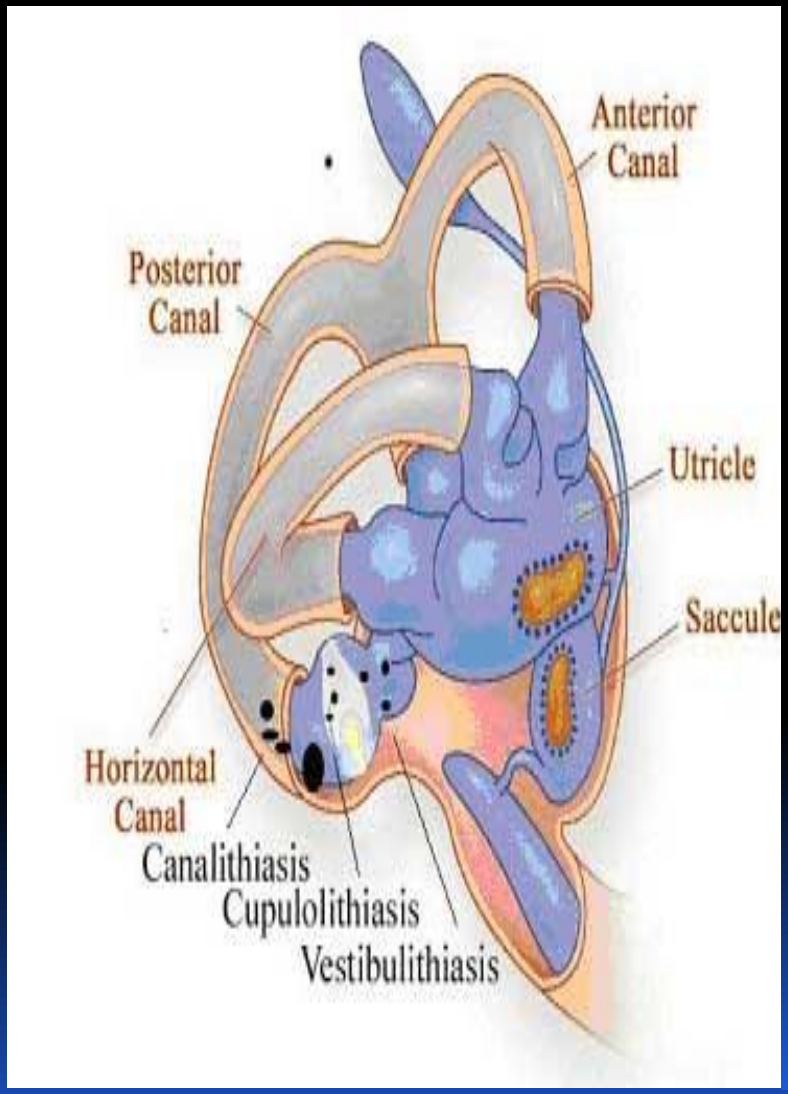
- Horizontal canals: rotation in the horizontal plane
- Left anterior and right posterior canals (LARP): rotation in the vertical plane skewed 45° anteriorly to the left.
- Right anterior and left posterior canals (RALP): rotation in the vertical plane skewed 45° anteriorly to the right.



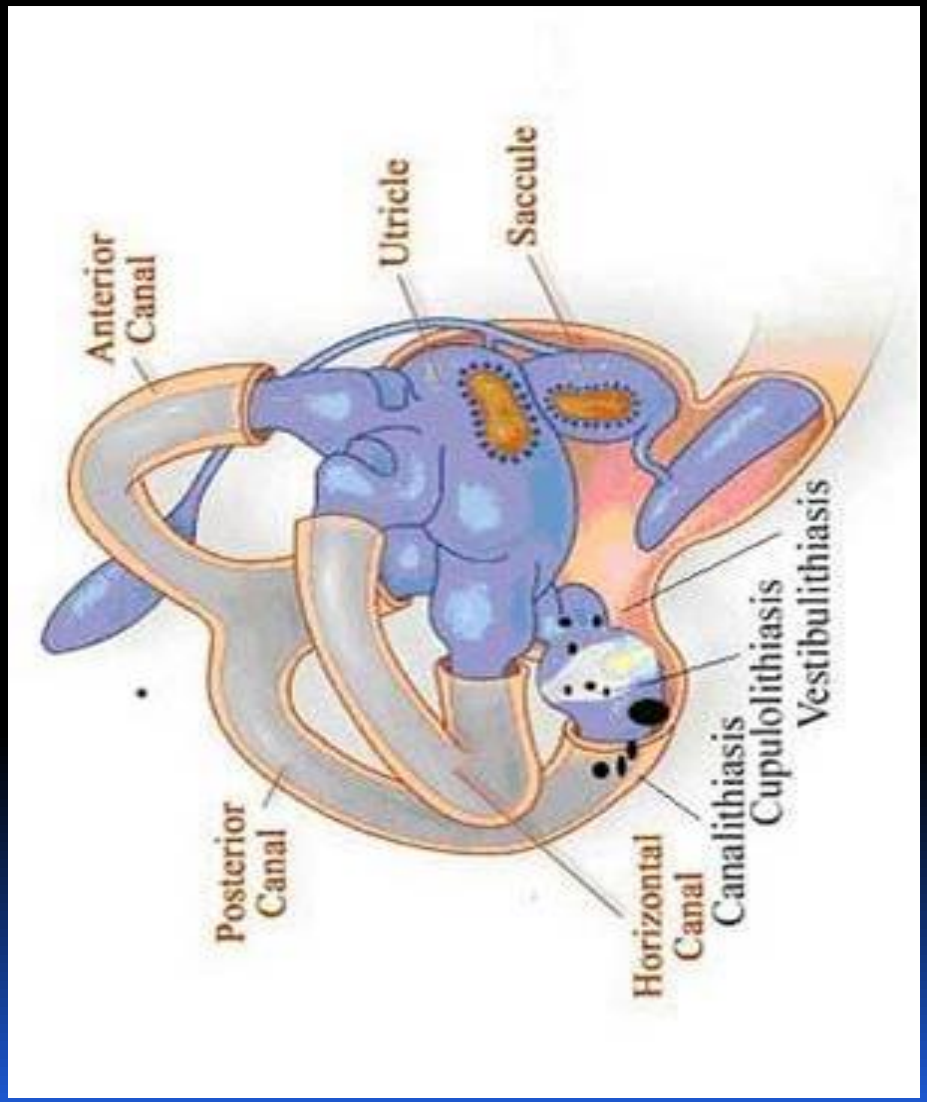


The Human Labyrinth In Different Body Positions

STANDING



LYING



Vestibular System : Physiology

- The vestibular system detects head motion and the effect of gravitational forces on the body. The vestibular system helps the brain in orienting the body during motion as well as in maintaining posture through interrelated networks
- There are two types of sensory epithelium in the vestibular system , the macula and the Crista ampullaris (Cupula) . Both contain rod shaped mechanoreceptors called hair cells. These receptors are embedded in a membrane of neuroepithelium and consists of a single large kinocilium and about 70 to100 sterocilia at its apical end.

Vestibular System:Physiology (2)

- Tip links connect the shorter sterocilia with adjacent taller ones. During head motion the sterocilia are tilted towards the kinocilium. The tip links get shifted resulting in opening of transduction channels which results in an influx of K^+ ions.The hair cells get depolarised and enables entry of Ca++ through channels at the base of the hair cells This causes neurotransmitter release into the vestibular synapses and increased firing of the vestibular afferent fibres.The bending of the sterocilia decreases the tip link tension and the channels are closed mechanically. This stops the influx of the Ca++ ions and the cells are hyperpolarised. The nerve fibres stop firing and the cycle is completed.

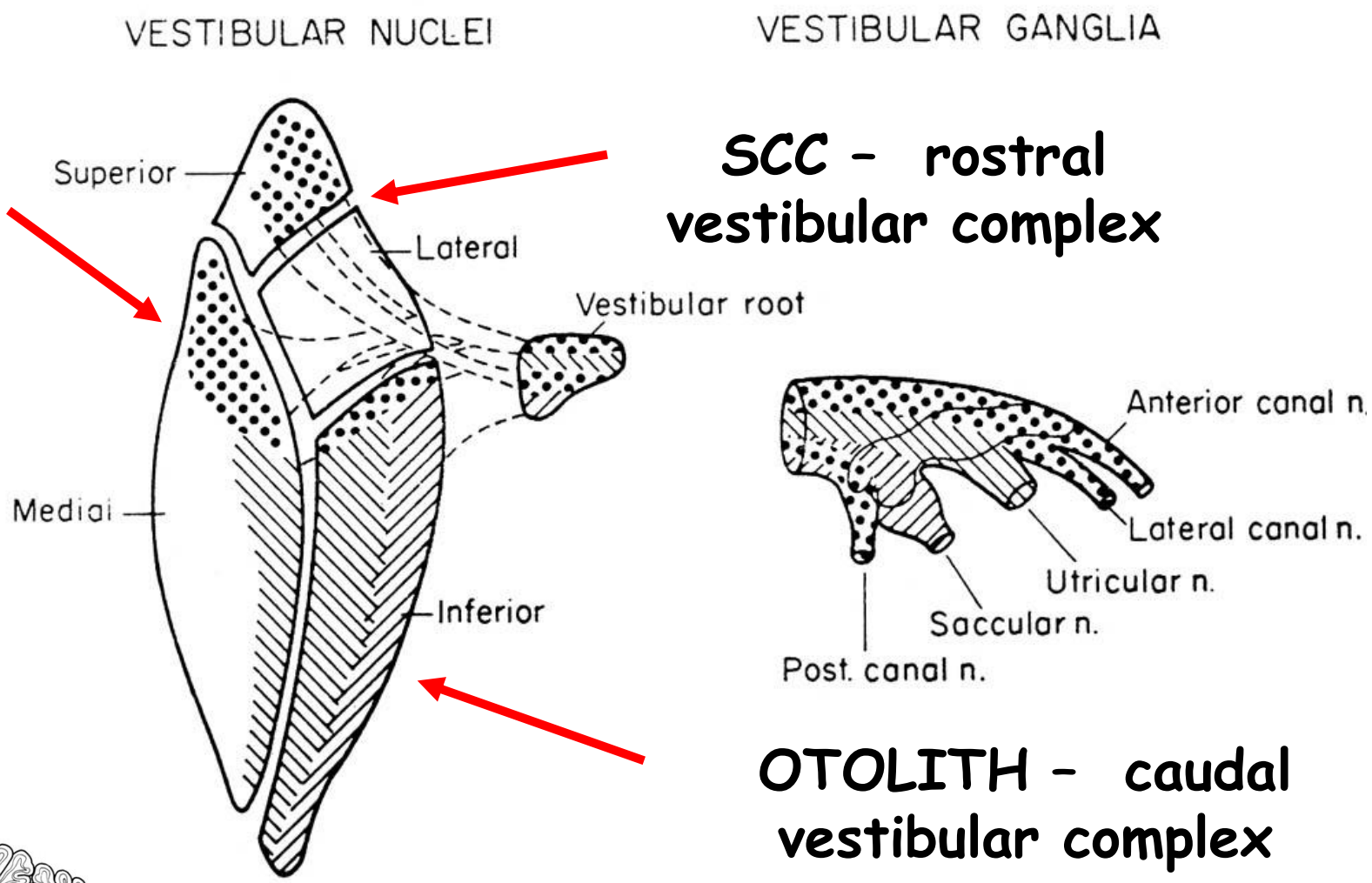
Vestibular System : Physiology (3)

- The utricle and saccule are concerned with static equilibrium .They respond to linear acceleration, tilting of the head and gravitational forces. Each contains a sensory structure called the macula. The utricular macula senses motion in the horizontal plane while the one in the saccule does so in the vertical plane. The interior of the macula has a gelatinous membrane with calcium carbonate crystals called otoliths or otoconia embedded in it. Vestibular receptor hair cells project from this membrane. .Linear movement or tilting of the head causes bending of the hair cells due to the inertial drag and shearing force between the otolithic membrane and the macular surface.

Vestibular System:Physiology(4)

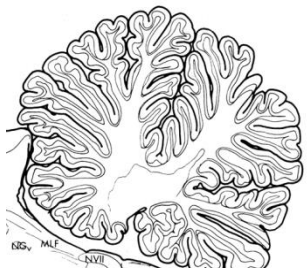
- Motion in one direction will stimulate one group of hair cells and inhibit the other. Some group of cells would remain neutral. This response pattern is essential in determining the position of the head to the central nervous system. When the head tilt stays beyond a few seconds, the bent hair cells and the depolarised membrane returns to normal in a process called adaptation. This allows the hair cells to respond to further changes in positions.
- The SCC are oriented at right angles to one another and sense angular acceleration. . The superior and posterior canals are at 45 degrees to the sagittal plane and the lateral canals are at 30 degrees to the axial plane. Functional pairing of canals exist. This arrangement allows a three dimensional vector for rotational acceleration

Central labyrinthine projections



SCC – rostral vestibular complex

OTOLITH – caudal vestibular complex



SCC / Otolith

VESTIBULOCEREBELLUM

Vestibular Laws

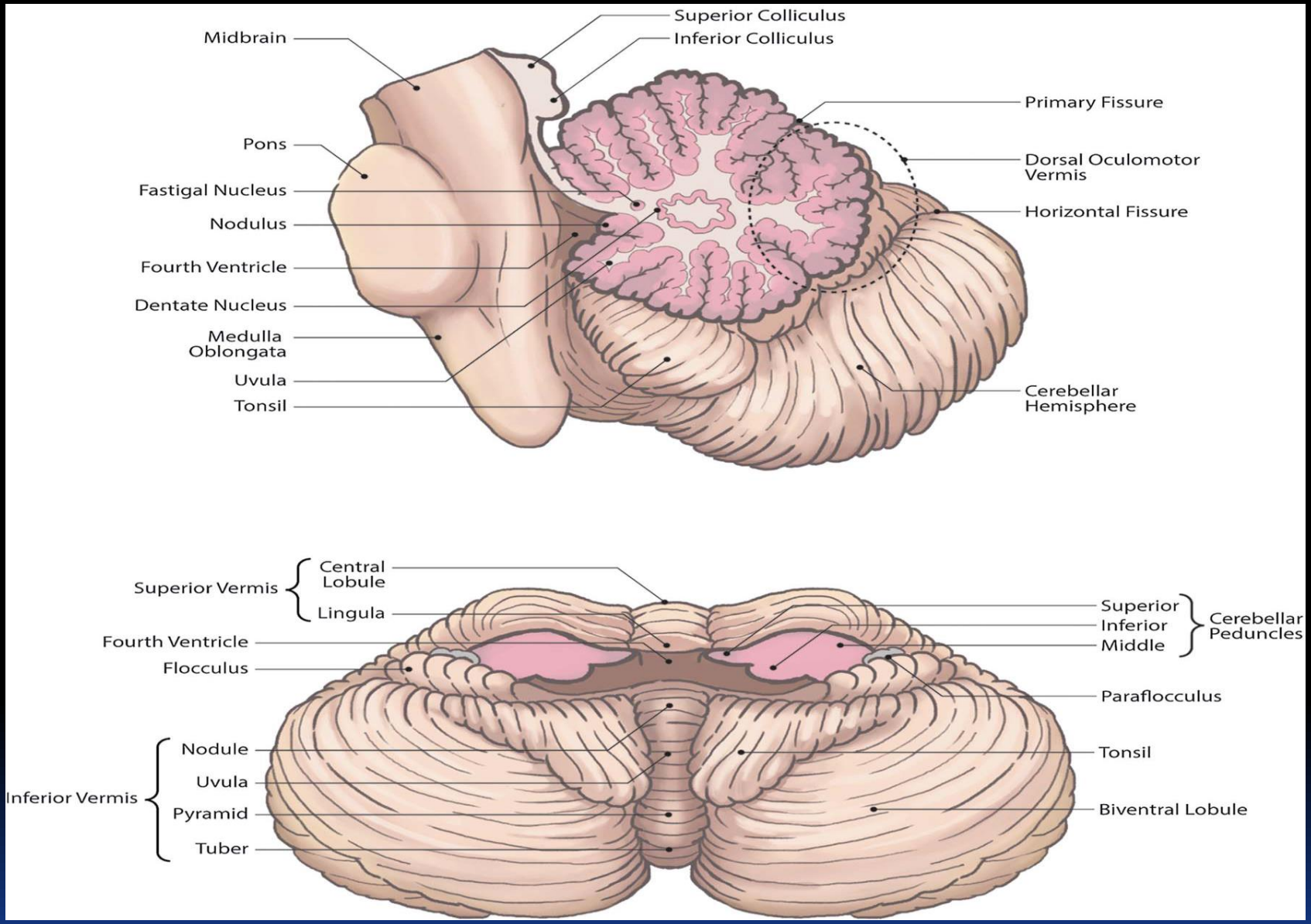
A. Ewald's 3 Laws :

- 1. A stimulation of the SCC causes a movement of the eyes in the plane of the SCC.
- 2. In the horizontal canal , an ampullopetal endolymph movement causes a greater stimulation than an ampullofugal one.
- 3. In the vertical SCC , the reverse is the true.

B. Alexender's Law :

- The spontaneus nystagmus of a patient with a vestibular lesion is more intense when he looks in the quick phase direction than when looking in the slow phase direction.

C. Anti – Alexender's Law : Central Lesion



Structures of the cerebellum subserving visuomotor function. The principal structures that play an important role in cerebellar visuomotor function are the ocular motor vermis (lobules VI and VII), caudal fastigial nuclei (or fastigial oculomotor region), ventral uvula, nodulus, flocculus, and paraflocculus. The ventral uvula and nodulus together form the caudal vermis The flocculus-paraflocculus and caudal vermis together constitute the vestibulocerebellum

Interaction of the Vestibular System and Cerebellum in Control of Balance

- The cerebellum acts as a regulator to the vestibular system and modifies its output like a processor when required. It does so by sending inhibitory outputs. The flocculonodular lobe and the vermis are mainly involved and are termed the vestibulo-cerebellum. The ipsilateral cerebellum is connected to the bilateral vestibular nuclei. It sends connections to the ipsilateral vestibular nucleus and fastigial nucleus which in turn is connected to the contralateral vestibular nuclei by the juxtarestiform body (inferior cerebellar peduncle).This connection is very important in maintaining balance by postural reflexes and in modulating behaviour.
- Different parts of the vestibulo-cerebellum subserve different functions. The flocculus adjusts the vestibulo-ocular reflex, the nodule modulates the duration of the vestibulo- ocular reflex and the superior vermis influences the vestibulospinal reflex.It does so by encoding vestibular signals and proprioceptive signals together

Interaction of the Vestibular System and Cerebellum in Control of Balance (2)

- The cerebellum has a major regulatory influence over voluntary limb movement , eye movement, balance , behaviour and even some higher cognitive functions. This theory is based on the extensive connections the cerebellum has with other relevant parts. Animal experiments give the same information. Cerebellar dysfunction is involved with problems of gait and balance. There is increased postural sway, increased or decreased responses to postural changes, inability to maintain equilibrium during motion of other body parts and abnormal oscillation of the body called titubation. The cerebellar gait or drunken gait is characterised by a wide base , veering to one side or the other, poor interjoint coordination and disjointed foot trajectories

Cerebellar Functional Organisation for the Control of Balance and Locomotion

- Observations in experimental animals provides strong evidence that the vermis and the flocculonodular lobe plays a key role in balance and smooth locomotion
- They help in maintaining the extensor tone which is vital for maintaining balance and posture. They also regulate the synchronicity of flexor and extensors by virtue of the wide connective network it has with the vestibular and reticular nuclei. The medial cerebellar region also receives sensory inputs from the limbs and sets up locomotor patterns
- Damage to the intermediate lobe leads to much less handicap compared to medial lobe affection. Upright posture and gait are affected very minimally in intermediate lobe lesions in experimental animals

Cerebellar Functional Organisation for the Control of Balance and Locomotion(2)

- Lateral lobe lesions affect gait minimally in normal walking conditions. In situations where more precise movements are needed and visual aid is required, the lateral cerebellum plays an important role. The lateral cerebellum plays a key role in visually guided movements as is evident from it's afferent and efferent connections with the visual cortex
- The medial cerebellum is more involved in the maintenance of posture and gait whereas the lateral cerebellum helps in more precision movements particularly the ones with strong visual cues

Cerebellar Control of Balance and Gait

- Cerebellar lesions involving the anterior lobe has increased sway in the antero-posterior direction while vestibulo-cerebellar lesions cause omnidirectional movement. Anterior lobe damage causes increased sway(of low frequency and high amplitude) without any directional preponderance. Lateral cerebellar lesions cause only mild postural sway
- Cerebellar lesions in humans cause hypermetric postural responses to surface displacement. Such patients show poor adaptation to changes during quiet standing or step initiation.

Cerebellar Control of Balance and Gait

- Human locomotion is bipedal and not quadrupedal like animals.It is less stable as it has fewer contact points and a narrower base compared to animals. Human gait thus needs greater descending cerebral control to maintain balance. Humans have greater influence from the lateral cerebellum which in turn has stronger cerebellar connections.Decomposition of movements is more seen in humans where a co-ordinated movement involving multiple joints is broken up in patients with cerebellar lesions. Multijoint movement is broken up into single joint movements involving the hip, knee and ankle joints

Three step mechanism for smooth locomotion

- Firstly the pattern of movement must be generated in the motor cortex. This also involves synergistic contraction and relaxation of flexor and extensor muscle groups
- Secondly the centre of mass should shift along with locomotion to maintain balance and the upright posture
- Thirdly the animal must be able to adapt to changes if there is a change in the movement pattern because of goal directed behaviour and locomotion
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Three step mechanism for smooth locomotion (2)

- *The cerebellum has a vital role to play in each of these three steps. Although the basic movement pattern is initiated in the motor cortex and then through the brain stem and spinal cord, it is the cerebellum which fine tunes motor activity. It maintains the smooth synergy between different joints to enable coordinated motor movement and also the balance between left and right sides*

  Anatomical Basis :

- *1. Cortico-pontine-ponto cerebellar – cerebello- dentate – dentato rubral – rubro thalamic –thalamo cortical circuit.*
- *2. Cerebellar afferents : Ventral and Dorsal spino-cecrbellar tracts.*

Cortical Control of Balance

- The cerebral cortex has also a role to play in the maintenance of balance. Till date no single vestibular cortex has been demarcated and there are different hypotheses. The lateral parieto-temporal cortex, the insular cortex, the right parietal opercular area have all been hypothesised. Kahane has also identified a lateral cortical temporo-parietal area which he has termed as the vestibular cortex. There are widespread connections with the thalamus and hippocampus and the latter is believed to play a vital role in memory guided movements. Some of these ideas have been corroborated by PET and f MRI studies.

Cortical Control of Balance(2)

- ***Cortico-cerebellar interconnection for smooth execution of movements is carried through the :***

- **Cerebello – Dentate – Dentato-Rubral(crossed) – Rubro-thalamic – Thalamo-cortical ciscuits** in one hand and the **Cortico – Pontine and Ponto – Cerebellar pathways (crossed)** on the other.

Vestibulo Ocular Reflex

- The vestibulo-ocular reflex (VOR) stabilise images of objects on the retina during head movement . This is achieved by a three neuron reflex arc. This involves the peripheral vestibular organs in the form of the semicircular canals, the vestibular nuclei and the external ocular muscles through their nuclei. The reflex enables conjugate eye turning in a direction opposite to the head motion.
- When the head turns right, the endolymph flow deflects the cupula to the left which causes depolarisation of hair cells to the right and hyper polarisation of the hair cells to the left.This fires the right vestibular nerve and impulses are sent to the vestibular nuclei and the cerebellum ipsilaterally.Impulses are sent to the right oculomotor nuclei and to the left abducens nuclei via the medial longitudinal fasciculus. This causes ipsilateral medial reclus contraction and contralateral lateral rectus contraction causing the eyes to turn in opposite direction to the head motion

Vestibulo-Spinal Reflex

- The vestibule-spinal reflex involves connections between the macula, crista ampullaris.visual motor system,the brain stem, cerebellum and the axial and proximal limb muscles.The lateral and medial vestibulospinal tracts are both involved. The lateral vestibulospinal tract plays a more significant role . In response to signals from the macula the lateral vestibular nucleus sends efferents via the lateral vestibulospinal tract to contract the ipsilateral axial and extensor muscles monosynaptically and inhibit the contralateral extensors bisynaptically.

  Angular acceleration activates the semicircular ducts and the medial vestibular nuclei are activated. Efferents travel by the medial vestibulospinal tract and activates the cervical muscle motoneurones bilaterally so that the neck can move in synchrony with the head motion.

Summary Of Vestibular Physiology

- The vestibulo-ocular reflex (VOR) ensures stability of vision during head motion by moving the eyes contralateral to the head to stabilize the retinal image; vestibulospinal reflexes help keeping the head and body upright. The semicircular canals (SCCs) sense angular acceleration to detect head rotation; the otolith organs sense linear acceleration to detect both head translation and the position of the head relative to the gravitational pull. The SCCs are arranged in pairs with two canals on each side working together in a push-pull fashion. Pairing occurs between the right and left lateral, the right anterior and left posterior, and the left anterior and right posterior SCC. If one partner is excited, the other is inhibited and vice versa, while under steady conditions the primary vestibular afferents have a tonic discharge that is exactly balanced between the corresponding canals. During rotation the head velocity corresponds to the difference in firing rate between SCC pairs. An idea about this pairing in a 3D geometrical concept along with their relative position with the otolith organs inside the inner ear would help one to understand certain patterns of nystagmus and ocular misalignment like skew deviation.
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The Basal Ganglia and Control of Balance

- 1.Storing and automatic execution of motor plans
- 2.Motor flexibility, adaptive behavior to
- environmental changes
- 3.Somatosensory integration
- 4. Muscle tone regulation
- 5. Gain control of automatic postural responses
- 6. Cognition, motivation and emotional aspects of behavior

CLINICAL COUTERPARTS :

- Gait akinesia, freezing ;Postural "inflexibility"
- Stooped posture ;Contraversive pushing ;Axial stiffness
- Exaggerated destabilizing responses ;Diminished stabilizing responses ; Co-contraction9. Impaired scaling of postural responses under conditions of uncertainty ; FALLS

Physiology of Human Balance
Concluding Remarks

The vestibular system coordinating head and eye movements and activating postural muscles that maintain balance, provide the proper orientation of the head and body in space. The vestibular nuclear complex is the primary processor of afferent vestibular input from the hair cells of the labyrinth and semicircular ducts. Under normal conditions the tonicity of the two vestibular nuclei are equal. Diseases of the end organs or central diseases disturb this tonicity between the two nuclei and cause dizziness and vertigo. The cerebellum functions as an adaptive processor that readjusts this balance as necessary. However the exact anatomical pathways interconnecting the cerebellum, vestibular nuclei and the cortical structures are yet to be elicitated. Efferent projections from the vestibular neurons, cerebellum, and the cortex control responses in the ocular and motor system that are essential for the body positions to maintain equilibrium with the gravity and environment.

Assessment Of Balance Disorders

- **ER Assessment :**
- Usually acute onset
- Level of consciousness and ABC
- Asso. : HA ; Vertigo /Dizziness /Nausea / Vomiting ; Gait : Veering - Unilateral /Bilateral
- Meningeal signs ; Full Neurological Examination
- Eye Exam : Vision ; Eyeball positions : Skew ; Deviations ; EOM - Ocular Palsy / Gaze Palsy ;
- **NYSTAGMUS : D/D - VESTIBULAR vs CENTRAL NYSTAGMUS**
- Fundoscopy
- Head Impulse Test (HIT) ; Peripheral vs Central Lesion
- IMAGING : Pitfalls
- **Clinic Assessment :**
- Subacute / Insidious onset : Progressive / Static / Improving
- Diagnosis often made as patient enters clinic ; Mobility ; Unsupported ; Supported ; Wheelchair ; **Enquire : Cognition ; Continence**
- Full neurological examination as above. (Proprioception ; Limb ataxia)
- Head Impulse Test ; Unilateral periipheral ; Bilateral peripheral ; Central
- Specific Balance Tests : Walking ; Limb / Truncal ataxia ; Romberg (EO /ES) ; Sharpened Romberg ; Tandem walking (EO/ES) ; Pull test / Press Release Test
- GAIT : Stance ; Start hesitency ;. Speed ; Stride ; Shuffling ; Base – Narrow / Broad : Sway – Uni / Bilateral ; U/L swing ; Abnormal Movements ; Bizzare
- **IMAGING and VESTIBULAR FUNCTION TESTING**

Assessment Of Balance Disorders

- **NOTES :**

1. In cases with significant loss of proprioception diagnosis of Bilateral Vestibulopathy becomes difficult. Establish diagnosis by Bilateral Abnormal HIT ; Caloric Testing and vHIT.

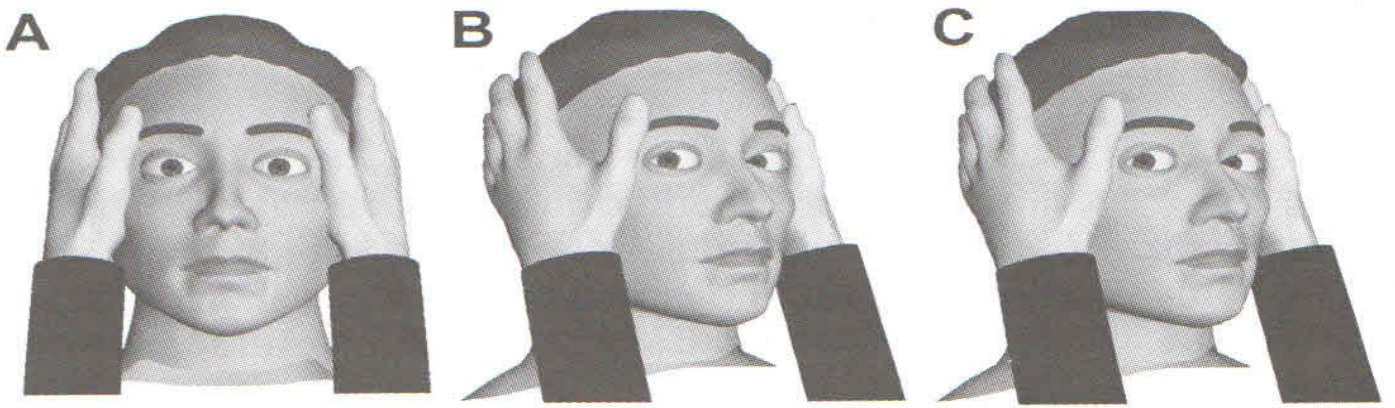
2. **Vestibular impairment does not cause a POSITIVE Romberg's Test when proprioception is intact : Ask patient to stand on a 20cm thick foam cushion which interrupts proprioception and causes inability to maintain upright stance in subjects with bilateral Vestibular Loss.**

3.UNTERBERGER TEST : March in one place 30secs with ES → ↑ turning to side of vestibular loss.

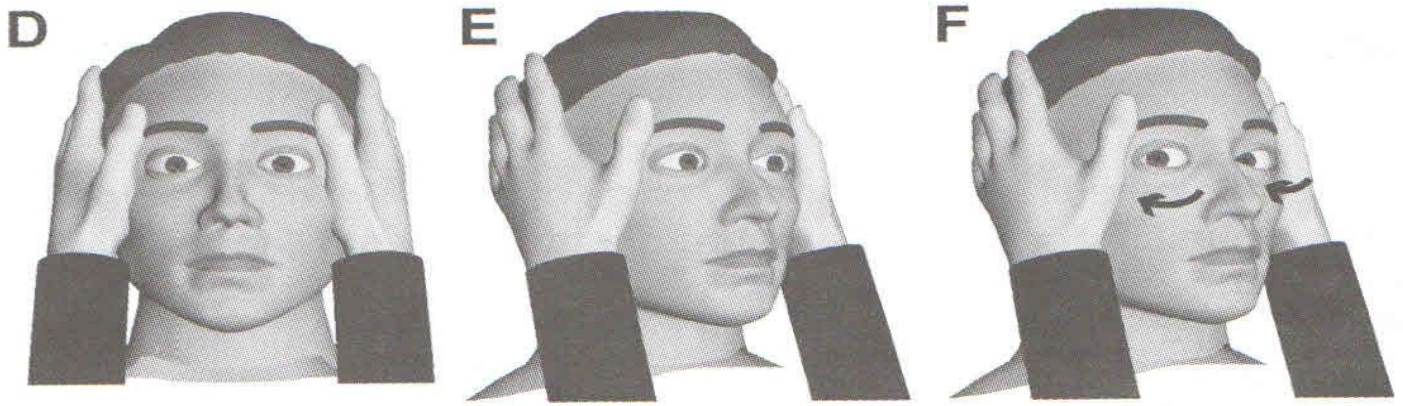
4.CANVAS Syndr, : Cerebellar ataxia ; Neuropathy : Vestibular areflexia

(Bilateral canal paresis ; downbeat nystagmus often seen)

Head Impulse Test (HIT)

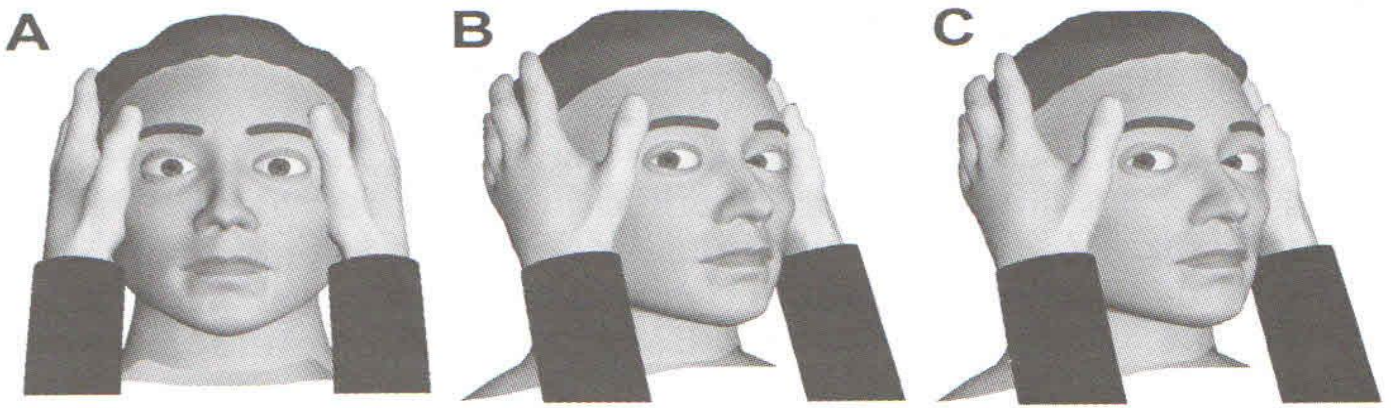


Normal leftward head impulse



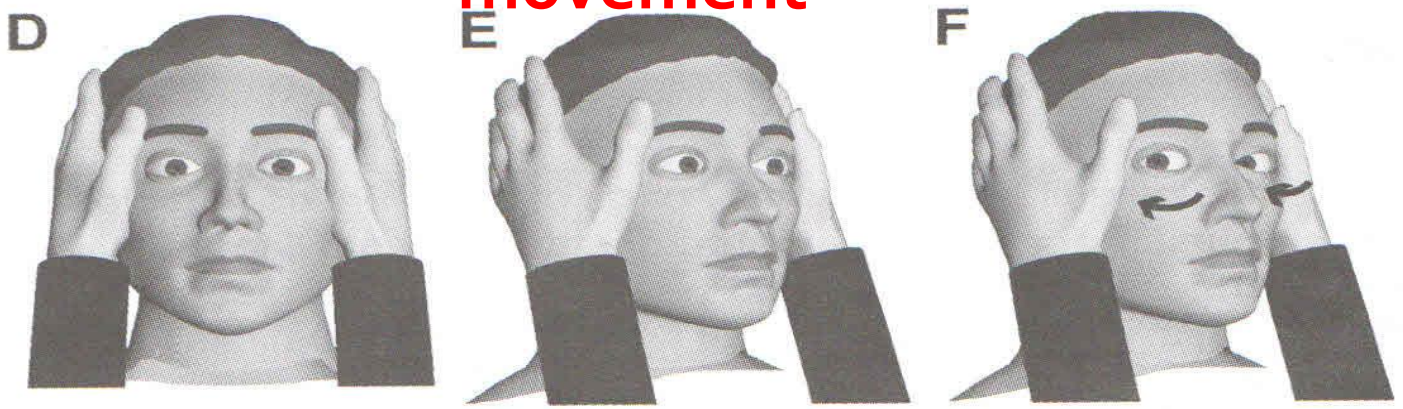
Positive leftward head impulse

Head Impulse Test (HIT)



Do head shake slowly few times before jerk movement

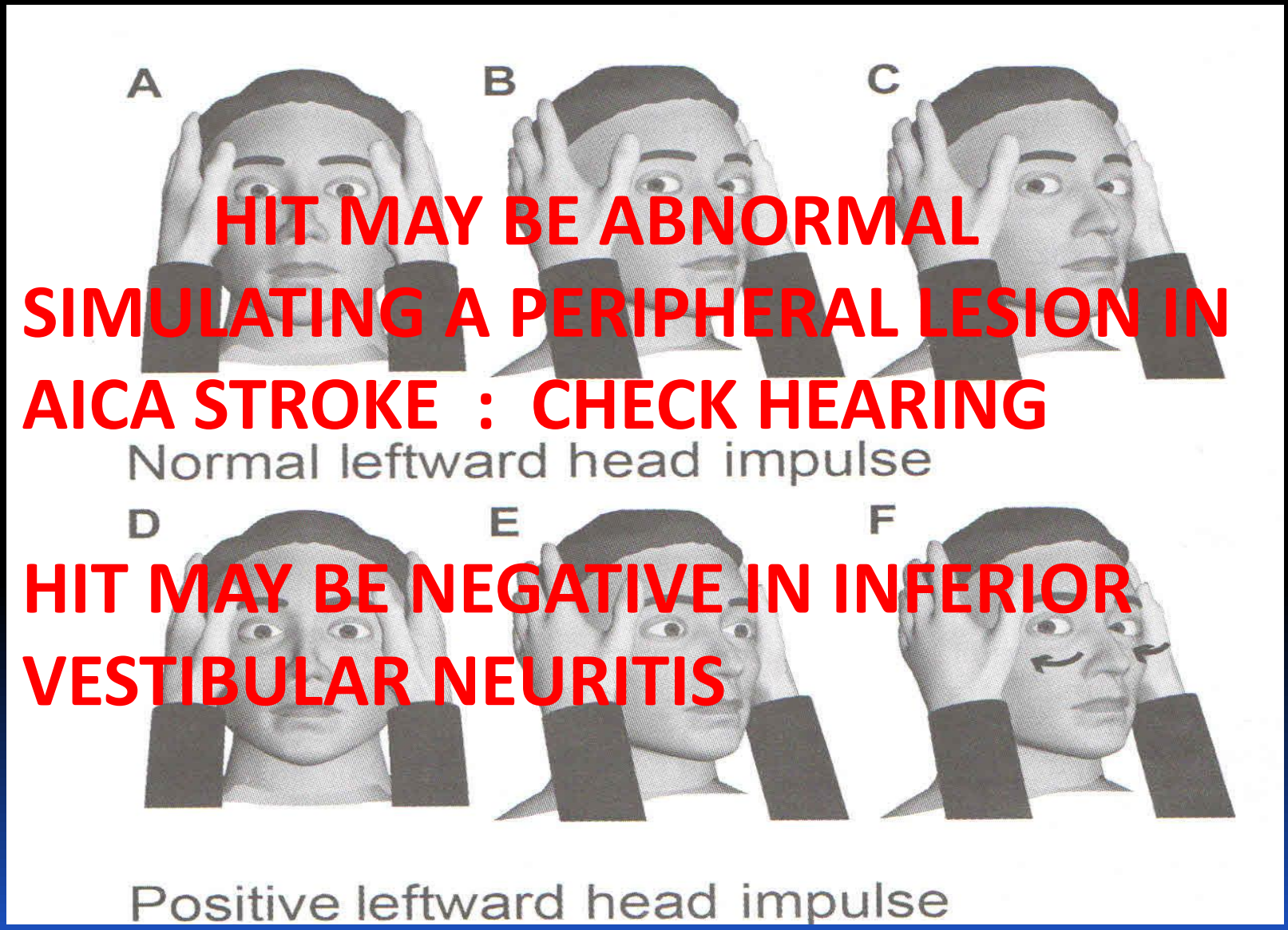
Normal leftward head impulse



Catch up saccade

Positive leftward head impulse

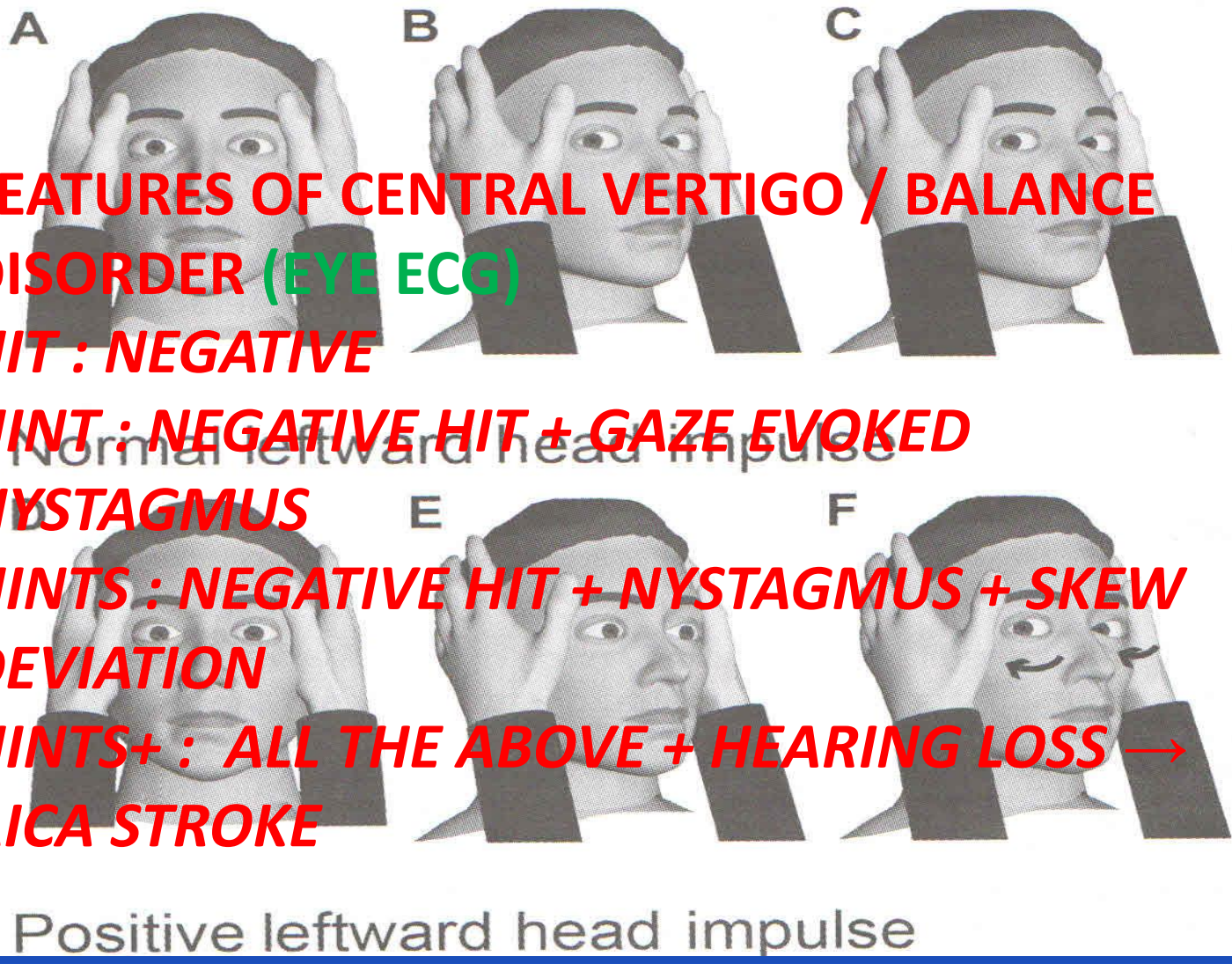
Head Impulse Test (HIT)



HIT MAY BE ABNORMAL SIMULATING A PERIPHERAL LESION IN AICA STROKE : CHECK HEARING

HIT MAY BE NEGATIVE IN INFERIOR VESTIBULAR NEURITIS

Head Impulse Test (HIT)



FEATURES OF CENTRAL VERTIGO / BALANCE DISORDER (EYE ECG)

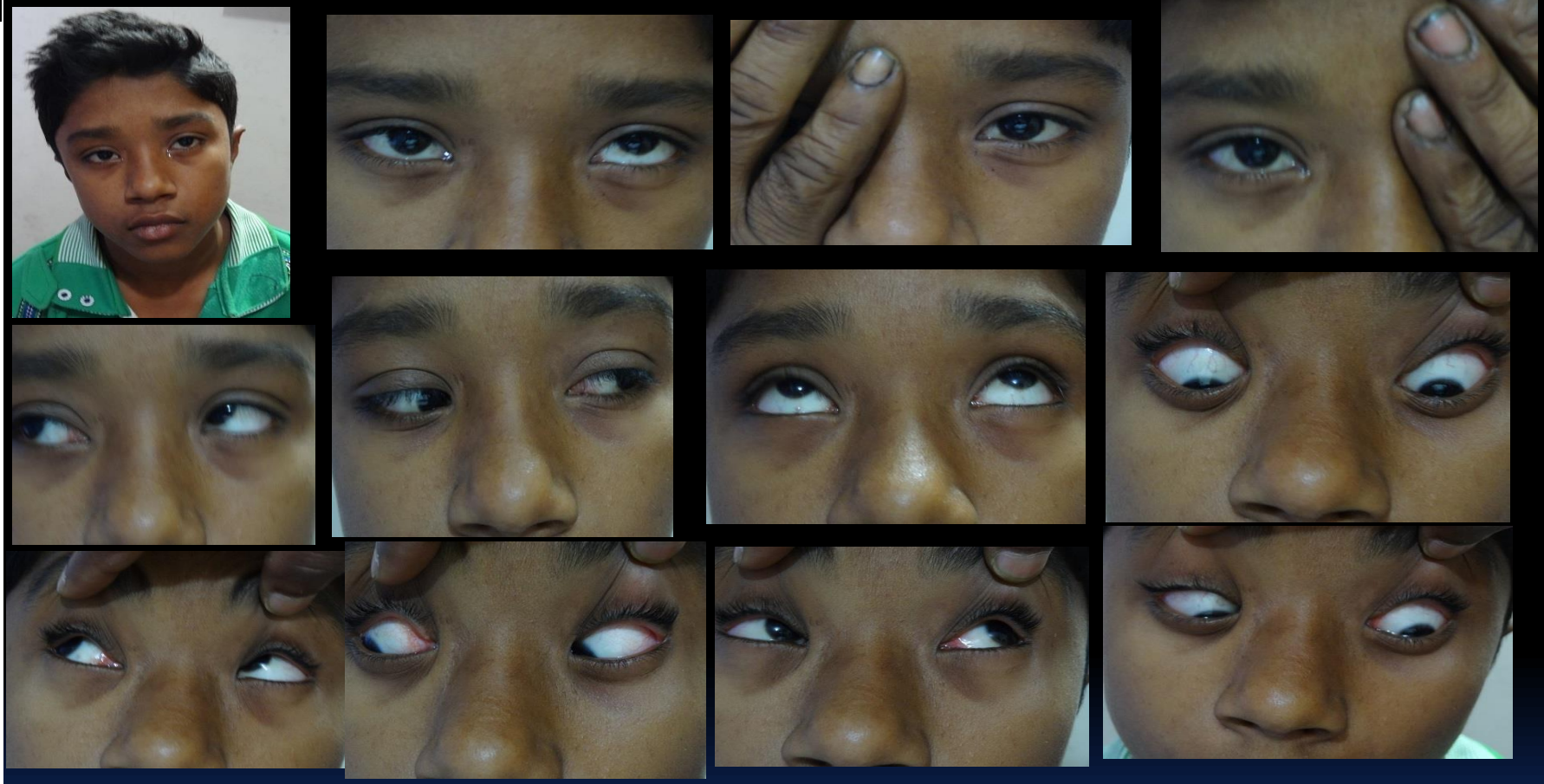
HIT : NEGATIVE

HINT : NEGATIVE HIT + GAZE EVOKED NYSTAGMUS

HINTS : NEGATIVE HIT + NYSTAGMUS + SKEW DEVIATION

HINTS+ : ALL THE ABOVE + HEARING LOSS → AICA STROKE

Head Tilt; Skew Deviation; Ocular Counter Roll = Ocular Tilt Reaction (OTR)



Skew eviation may also occur in Trochlear Nerve palsy: Ask patient to lie down : OTR skew disappears but not Trochlear palsy skew.

Higher Level Gait Disorders

Types of Gait Disorders :

- Antalgic gait
- Paretic / Hypotonic gait
- Spastic gait
- Vestibular gait **(Don't forget : Bilateral Vestibulopathy)**
- Cerebellar gait
- Sensory ataxic gait
- Dyskinetic gait
- Hypokinetic rigid gait **(L-DOPA Responsive PD)**
- Cautious gait
- **Higher Level Gait Disorders**

Higher Level/Order Gait Disorders

Higher level gait disorders should not be used as a dumping ground for all unclassifiable gait disorders. It is stressed that it indeed is a distinct clinical entity characterized by the presence of one or more core features which would include :

- Gait disorders occurring in close temporal relation to cognitive dysfunction.
- Gait disorders with falls (not with impaired consciousness), not explained by any sensory modality dysfunction or hemodynamic disturbance.
- Severe balance impairment (no rescue reactions with the pull test; falling like a log).
- Inadequate synergies
- Inappropriate or bizarre foot placement
- Crossing of the legs
- leaning into wrong direction when turning
- Variable performance (influenced by environment and emotion).
- Hesitation and freezing (ignition failure).

Higher Level/Order Gait Disorders

The major predominantly locomotory disorders include :

- *Freezing*
- *a) narrow based*
- *b) broad based*
- *Marche a petit pas*
- *Cautions gait*
- *Careless gait*

Extrapyramidal Disorders :

Parkinsons disease

PSP ; Comm Hydrocephalus

Lower Body Parks.

Following Falls

Dementias

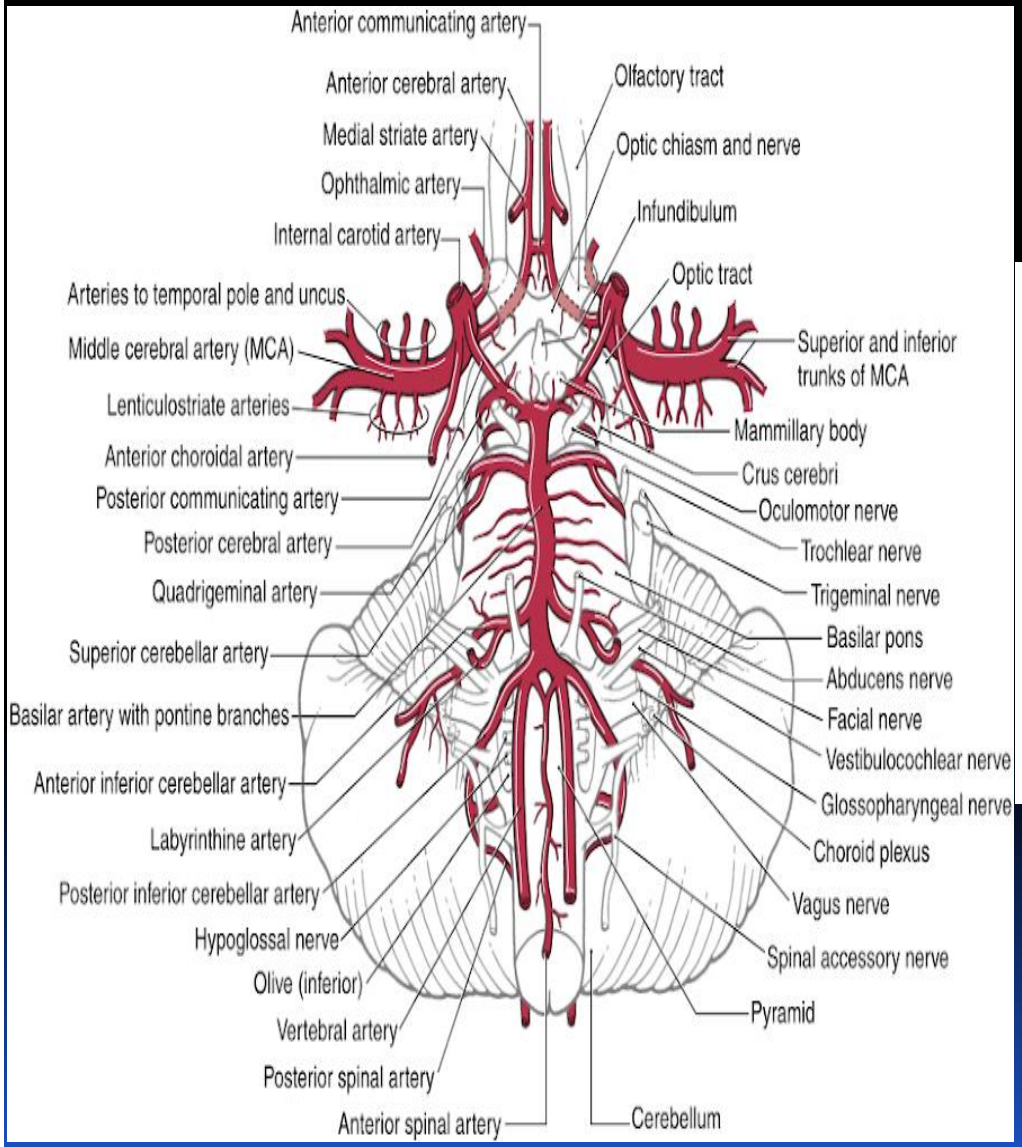
The principal types of higher level balance disorders include :

- *Lateral pusher syndrome*
- *Posterior pusher syndrome*
- *Astasia*
- *Apraxia of balance*

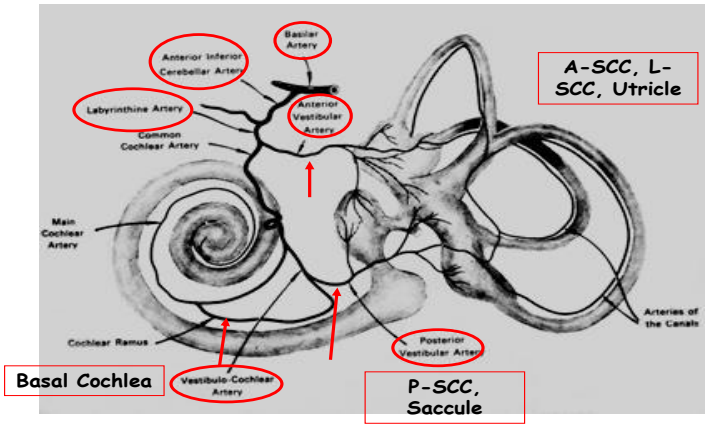
PSP

Communicating Hydrocephalus (NPH)

Cerebral and Labrynthine Circulation

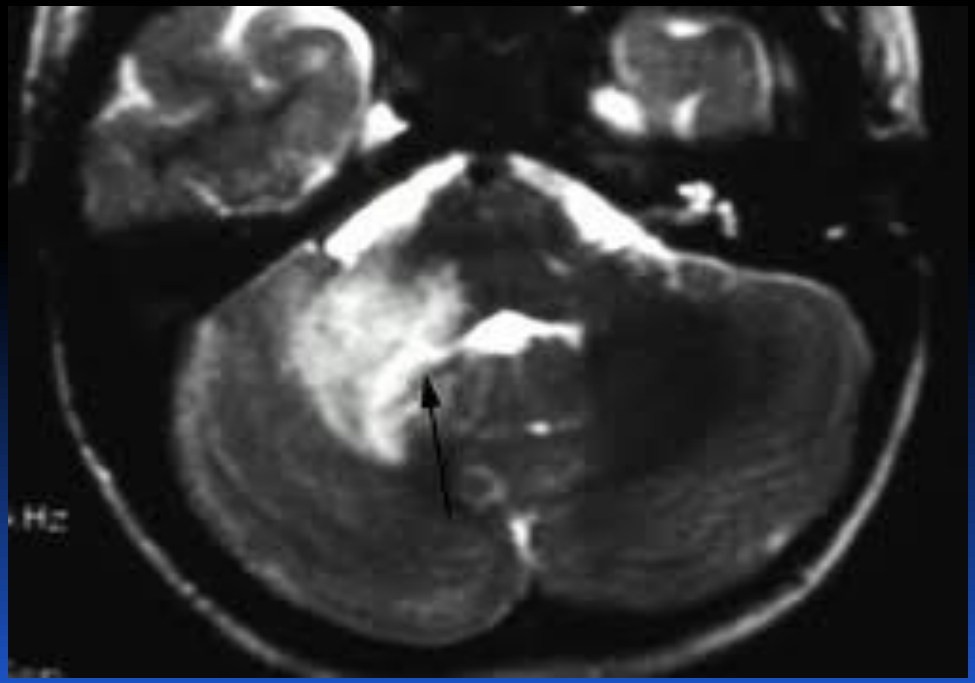


Vascular Supply of the labyrinth



Case : Acute Onset Vertigo With Hearing Loss

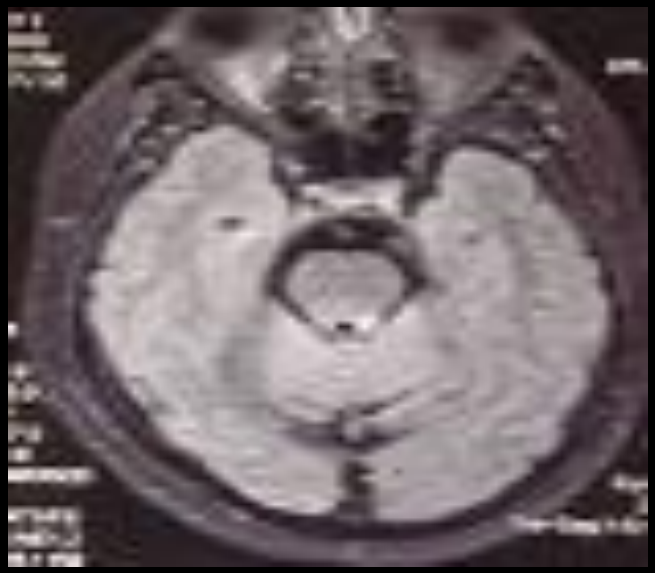
- A 65 years old hypertensive lady had acute onset of severe rotatory vertigo with nausea and recurrent vomiting. She also complained of loss of hearing in the right ear along with the onset of vertigo. On admission she had a gaze evoked horizontal nystagmus, an abnormal HIT and sensorineural hearing loss in the right ear. The vertigo took several days to settle and then on attempting to walk she was veering to the RIGHT. A MRI of brain had been done.

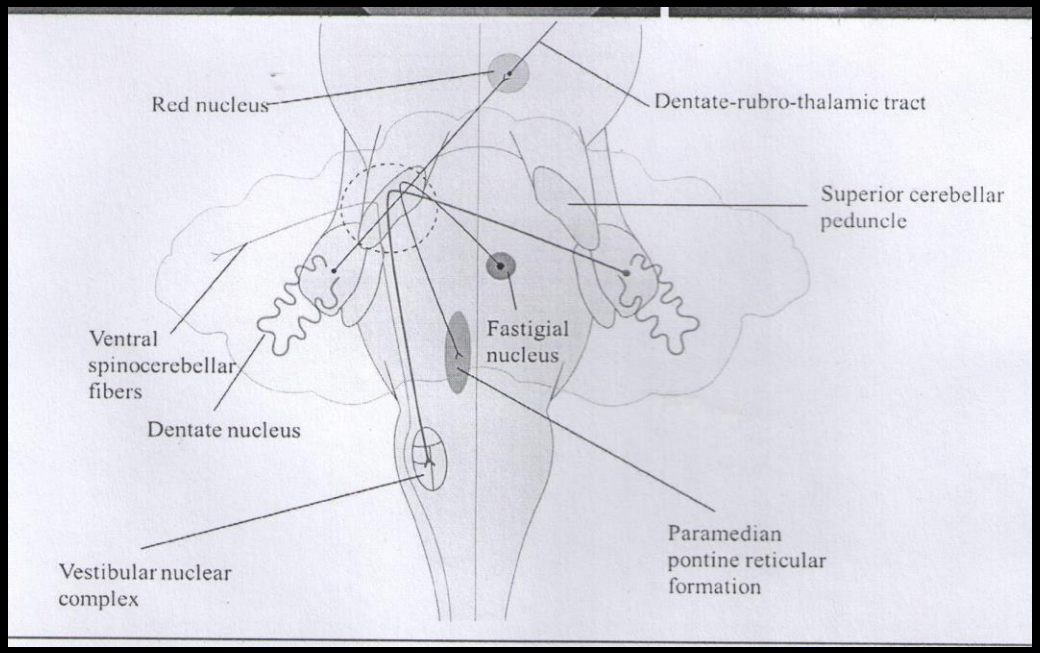


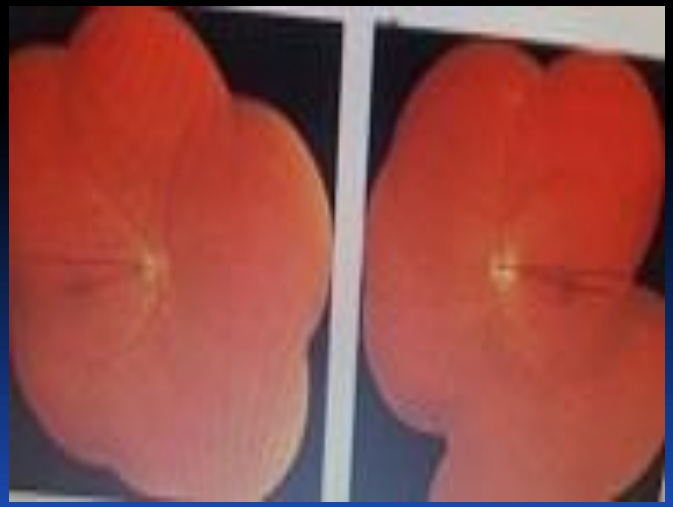
Case : It All Happened During A Train Journey!

- A 49 years old hypertensive man was returning to Calcutta after a visit to North Bengal along with some friends in April 2018.He was also a smoker. In the night he went to the washroom where he felt a severe rotatory vertigo of unknown duration. This is the last thing he remembered. It is not known whether he fell or lost consciousness for a while. A fellow passenger noted him staggering in the corridor to reach his seat. He helped him to his seat and to his friends. Apparently he started talking to his friends but later had no recollection of this. He mentioned to his friends that he was seeing double. His friends noticed that his speech was slurred, his left palpebral fissure was narrow and he was unsteady on his feet veering to the left side. Thinking that he probably had a stoke of some kind, on reaching Calcutta next morning , they admitted him in a hospital near his home. The patient has no recollection of these. A plain CT brain done in that hospital showed a small hypodense area over the left posterolateral aspect of the midbrain. Later in the day he was transferred to the neurological services of a super speciality hospital. A MRI brain was performed which showed a small hyperintense area over the left posterolateral aspect of the midbrain in the T2 and FLAIR sequences . His condition improved in the hospital and his memory returned after about 5days from the day of the incident. The discharge note from the hospital mentioned only his symptoms like dysarthria, diplopia and unsteadiness. No examination findings were mentioned.
- The present author examined the patient about 2 weeks after the onset. By this time his speech was clear but he still had a narrow left palpebral fissure with normal pupils. He was still complaing of an oblique vertical diplopia on looking to the right and was unsteady on his feet with tendency to fall on the left side. He did not have any head tilt nor any skew deviation of eyes and his horizontal HIT on both sides was normal. Ocular movements were full in all directions but he mentioned of seeing double on looking to the right. The diplopia was vertical and a little oblique with the lower image hazy (false image). Covering the right eye , he felt that the lower hazy image disappeared. This was further confirmed by putting a red glass in front of his right eye when the lower hazy image looked red; suggesting that this false image was coming from the right eye . Saccadic and pursuit movements were normal. In addition he had numbness over the right side of face restricted mostly in the distribution of the maxillary division of the trigeminal nerve. He had mild residual ataxia in his left upper and lower limbs and was falling to left on attempting to walk heel to toe. His memory, barring the first 5days since onset , was normal. His BP was controlled. A Holter monitoring showed no arrhythmias. Trans-esophageal Echocardiography was also normal. A MR angiography showed no vascular occlusion specially in the posterior circulation.

It All Happened During A Train Journey! (contd)



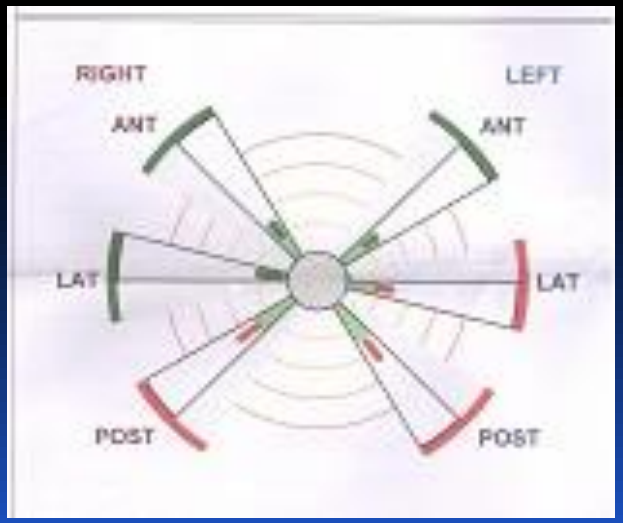


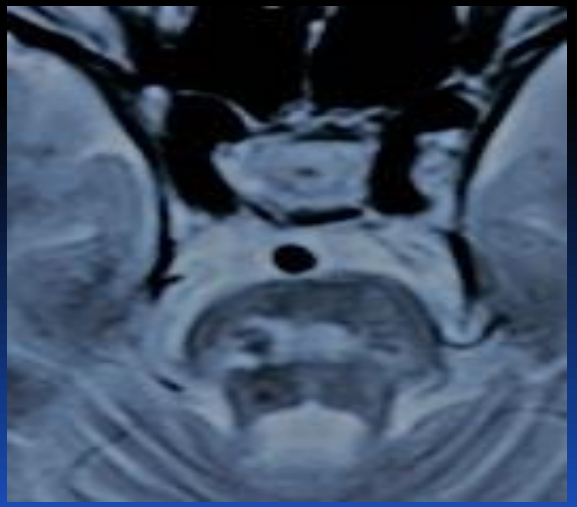


Some studies have demonstrated a role of the SCP in controlling ocular movements in the Roll (coronal) plane. This is likely controlled by the Uncinate Fasciculus (UF) which hooks round the SCP and then makes connections with the paramedian pontine nuclei, a vital structure in controlling conjugate eye movements. Fibres controlling ocular torsions ,arising from the DN also pass through the SCP and finally make connections with the vestibular nuclei. These various pathways passing through or in close proximity to the SCP may explain the ocular torsion observed in the present case.

Case With Clinical & Investigation Discordance

- On 23 January 2019, a 68 yrs old man had an acute onset of severe rotatory vertigo with nausea and vomiting and was unsteady on his feet. Though he was treated with anti-emetics by his local physician , it took about seven days for his vertigo to settle. A CT scan of brain was done in the mean time and this was normal. When examined three days after the onset of vertigo, his Bedside HIT was negative, external ocular movements were normal, no nystagmus (examined without blocking visual fixation), no facial or limb weakness , no appendicular ataxia but he had significant truncal ataxia. He had bilateral high frequency hearing loss in both ears and had a normal calorigram. The Video HIT revealed gross abnormality in the LEFT lateral SCC as well as in the RIGHT and LEFT posterior SCCs but not in the anterior canals. Perforence of MRI of brain was delayed due to financial reasons. It was done 10 days from onset.





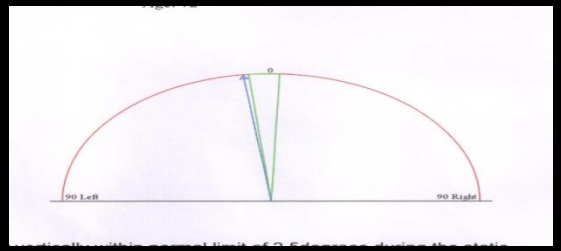
A Complex Case of BPPV

- A 72 years old doctor, diabetic ,hypertensive and a little overweight. He had a habit of getting out of bed first thing in the morning by leaning on his left side and then pushing himself up by pressing on his left elbow. This had been a long time habit.
- One morning while he just raise his head up to get up from bed with his torso obliquely turned to the left side, he experienced a severe rotatory vertigo and was thrown back on the bed. The vertigo persisted for the next several seconds so long he was lying on his left and was relieved to some extent when he laid supine on his back. Settling down a bit, he made two further attempts to get up in the same way leaning to his left and exactly the same thing happened with severe spinning and being thrown back on the bed. It took about more than 15 minutes for this process to settle down when he could get out of bed and stand on the floor. However his wife noticed that he was leaning to his left as he stood upright and had been veering to the left as he was walking to the washroom.
- Brief spells of vertigo continued to occur with any turning to the left for the next 7 days or so and althrough this period he had been veering to the LEFT. Examined a week later, he had no nystagmus , an abnormal Romberg's test with eye open and shut and on tandem walking ,was veering to the left.
- A Dix Hallpike test was done and there had been a very brief spell of vertigo with the left ear down but no nystagmus could be seen using a M-Glass.
- A left Epley's maneuver was carried out rather gently and he was taught to practice home Epley everyday at home.
- DETAILED INVESTIGATIONS FOLLOWED

A Complex Case of BPPV (contd)

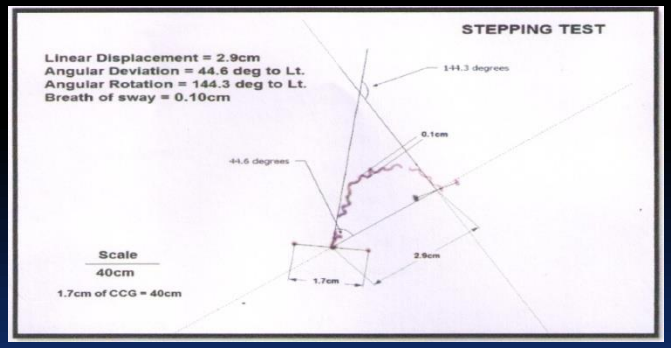
NORMAL IMAGING

- **SVV :** Patient tilted the visual vertical abnormally to the left side during the counter clockwise rotation of the background suggesting a Left Otolithic Dysfunction that had been compensated . During the clockwise rotation of the background , the patient did not tilt the vertical light bar abnormally to one side. It seemed the abnormality was limited to the Left Utricle.



- **CRANIOCORPOGRAPHY (STEPPING TEST) :**

Abnormal Angular deviation of 45 degrees to the left and Angular rotation of 144 degrees to the left as well .



- ***Both the tests thus pointed to a Left sided vestibulopathy likely affecting the Left Utricle in the light of the clinical context.***

BUT WHY ?

Elderly Doctor With Gross Unsteadiness

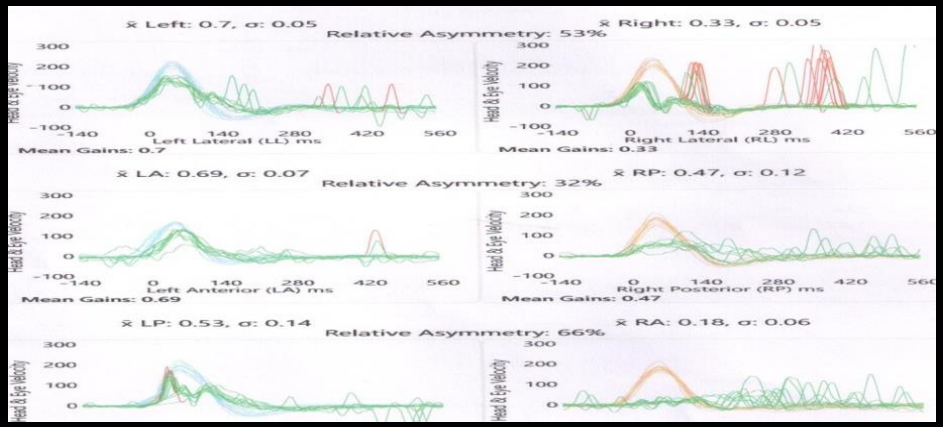
- A 90 years old gentleman, a gynaecologist by profession, with well controlled hypertension and diabetes mellitus, presented with six years history of progressive unsteadiness in walking for which he had been using a stick of late. He had had a few falls over the years with soft tissue injuries only. Falls occurred as he stumbled on uneven areas when he lost balance and fell without ever losing consciousness. He never complained of any vertigo. For the past several years ,he had developed progressive hearing loss in his right ear and currently he is totally deaf without any form of sound perception. Progressive hearing loss also occurred in the left ear as well but with help of a hearing aid , in the left ear , he has fairly good hearing and manages conversations well. He never complained of any form of fullness in either ear nor any tinnitus.
- Without his hearing aid, he could not perceive any sound from the tuning forks in either ear (256Hz and 512Hz). He walked with a wide base, swaying from side to side and could not stand at all with feet together (Romberg) either with eyes shut or open. He could not put one foot in front of the other to walk tandem due to severe gait ataxia. There was no appendicular ataxia. His bedside HIT was bilaterally abnormal with catch up saccades to the contralateral side. Tendon reflexes were absent and plantars were flexor. He was continent. His CT scan of brain only showed age related atrophic changes. He had no very significant cognitive changes and still manages office consultations for small number of patients but no surgery.

Doctor With Gross Unsteadiness (contd)

- **Vestibular function testing** :

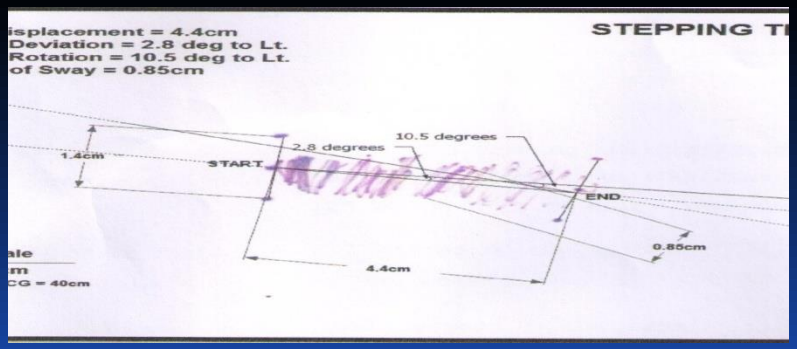
1.Caloric Test : On estimation of nystagmus by culmination frequency there were gross hypoactive responses in both ears with both cold and warm water.In fact , no nystagmus were elicitable.

2 vHIT :



Abnormal in all 6 canals.

3.Stepping test :

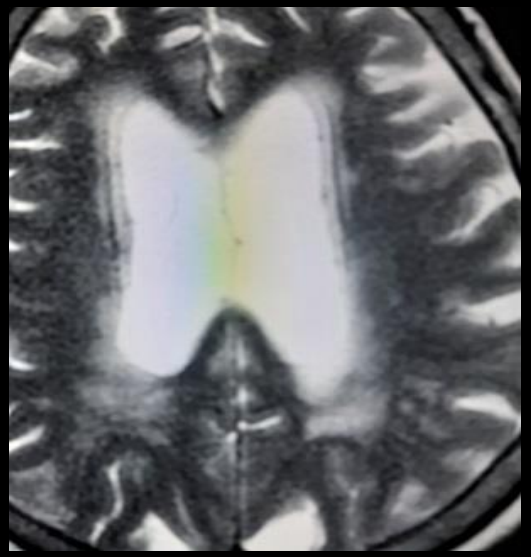


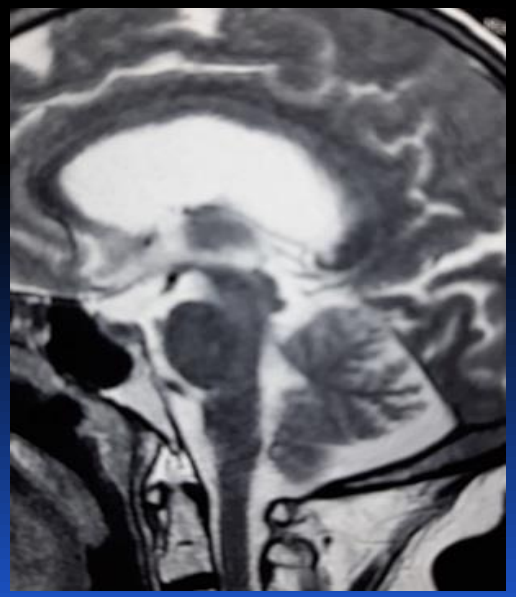
Sway +++

∆ **Bilateral Vestibulopathy (Presbatic)**

Elderly Lady With Unsteady Walking

- **76 yrs** hypertensive and diabetic lady presented with progressive unsteadiness of gait for 2 months. No vertigo. Higher mental functions normal and she was continent. She had gross truncal ataxia, broad based gait and no limb ataxia. Tendon areflexia and flexor plantars**.. HIT was normal.**
- **MRI Brain** : Age related atrophy ; white matter signal corroborating to chronic cerebral ischemia : Significant supratentorial ventricular dilation with well visualisation of aqueduct and fourth ventricle **(Communicating Hydrocephalus**)
- Within a few hours of clinic visit , became unable to walk at all and incontinent. No LOC / HA / Vertigo /Vomiting /Speech or memory disturbance.
- **Urgent LP and 15ml CSF drain done. Opening pressure 36cm water**. Next morning found sitting up in bed. Following further 15 ml CSF drain : started walking with a little support and continent.





Elderly Man Thrown Off Bike

- A 60 years old man was driving his motor bike on a road in Jamshedpur, Jharkhand, India, when for no apparent reason, he was thrown off his bike at some distance. He did not lose consciousness and remembered the fall and the sound of his helmet striking the ground. He experienced no vertigo.
- Later he mentioned of some degree of hearing loss in both ears associated with intermittent blocked sensation in either ear and also hearing noises in his ears at times. There was no past history of dizziness and vertigo.
- DISCUSSION : Falls without any warning in this case depict the classic story of the Otolithic Crisis of Tumerkin. The condition was first described by Tumerkin in 1936 in three patients and was initially described as Otolithic Catastrophe. Often such attacks were not preceded by vertigo and often labeled simply as "drop attacks". Tumerkin described what happened to one of his patients as :" *One day he was standing at his desk talking to a client when suddenly he slumped to the floor. He had no vertigo, no loss of consciousness and no malaise. The thing came like a bolt from the blue, but he could immediately assure onlookers that he was alright and got up immediately and carried on."* . It is now thought that there occurred a sudden rupture of the utricular wall causing either a sudden rise of endolymphatic pressure or an electrolyte imbalance. It is possible that somehow, probably by ballooning of the utricular membrane, the semicircular canals are shut off and they escape the effects of sudden rise of pressure and other effects and such patients do not complain of any preceding vertigo.

Human Balance Disorders : Challenges and Tips/Tricks

- Balance disorders are the commonest cause of FALLS with preserved senses in the elderly carrying significant morbidity and may be fatal as well.
- Patients and care givers need to be appraised of the seriousness but need not be overcautious or panicky imposing undue movement restrictions.
- Gait training and Balancing exercises must be stressed and adequate supporting devices and systems to be provided.
- Any visual disturbance must be corrected as far as practicable.
- Some TIPS /TRICKS need to be taught :

Tricks to overcome Freezing : Visual fixation on a point on floor and crossing over it ; Crossing over parallel lines; Marching at point of freezing to initiate movement ; Use of Laser beam guided lines fitted to sticks ;

Interestingly such patients find easier to climb stairs than walk on plain.

Patients with balance disorders should NEVER :

1. Stand on one foot to wear trousers → must sit on chair/bed
2. Walk backwards or sit without any back support → to prevent retropulsive falls.