Anatomy and Physiology of the Vestibular System

Charleston Vestibular Update
November 6th , 2025

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Medical University of South Carolina

Changing What's Possible | MUSC.edu

Disclosures

- Spiral Therapeutics
- Vestibular Disorder Association
- None of the above disclosures are relevant to the talk and course

Outline and Objective

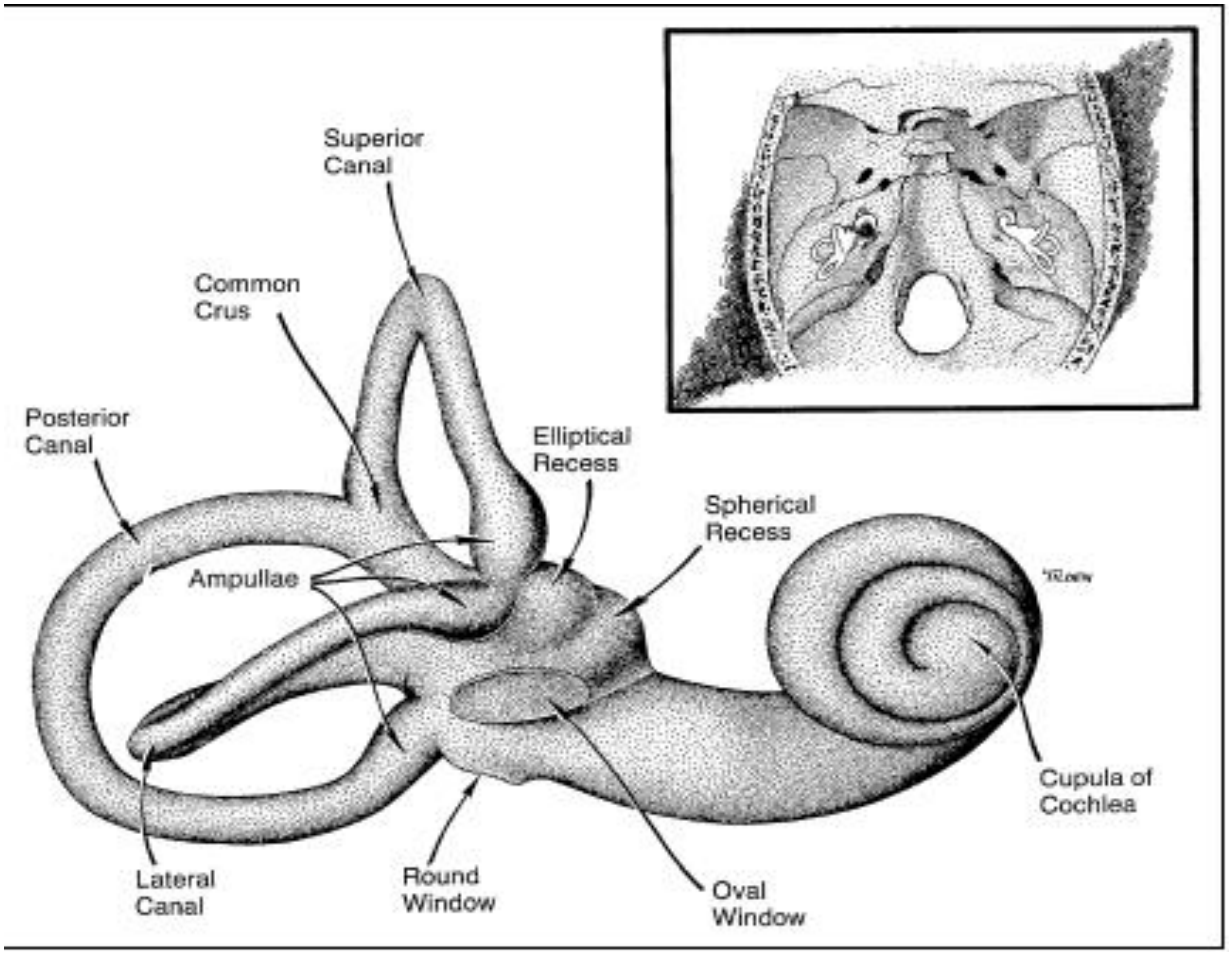
Anatomy and Physiology of the Vestibular System

- Understand site of lesion and mode of presentation
- Define Peripheral and Central Vestibular Disorders
- Describe the Vestibulo-ocular Reflex

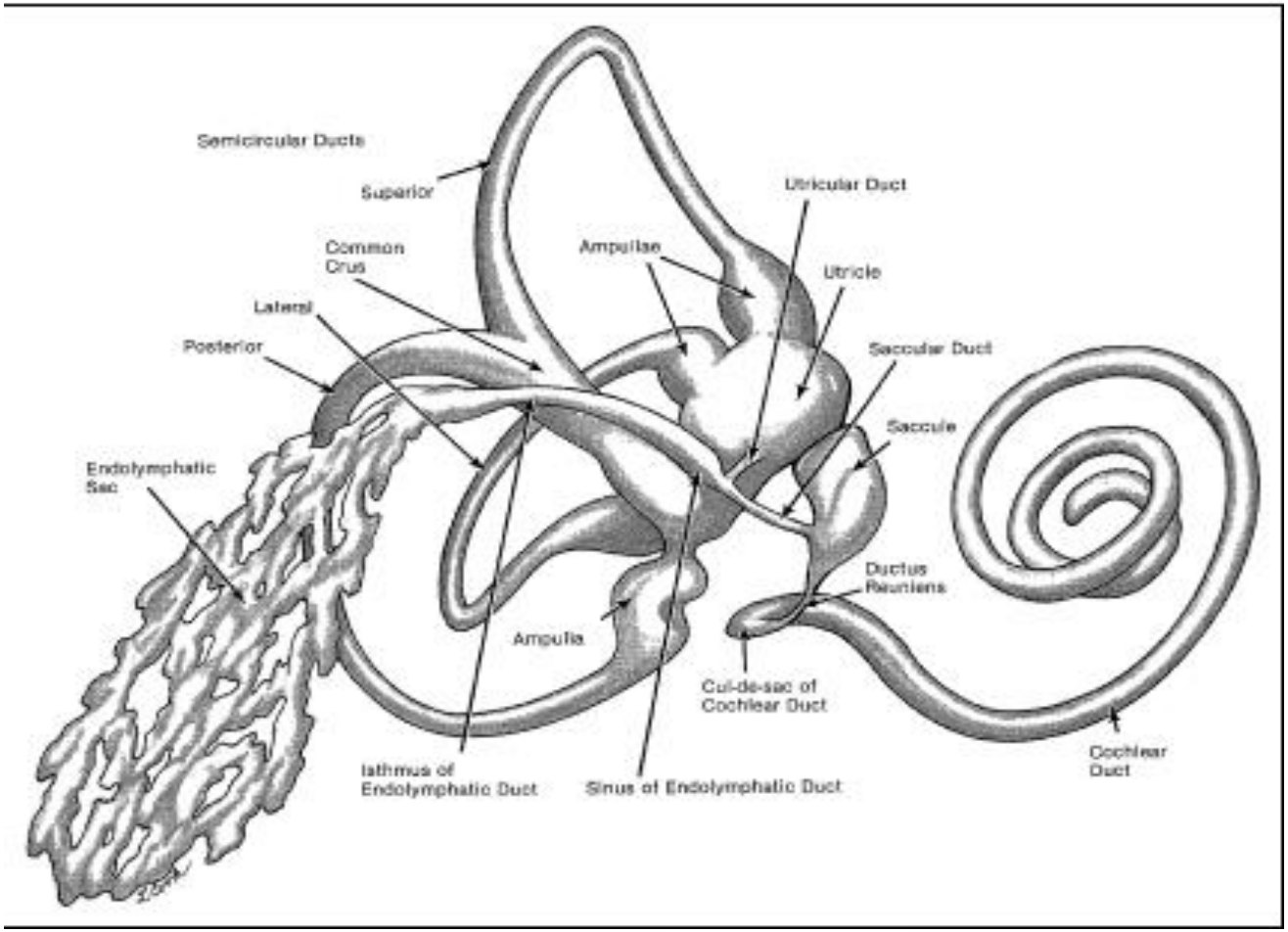
Aging of the Vestibular System

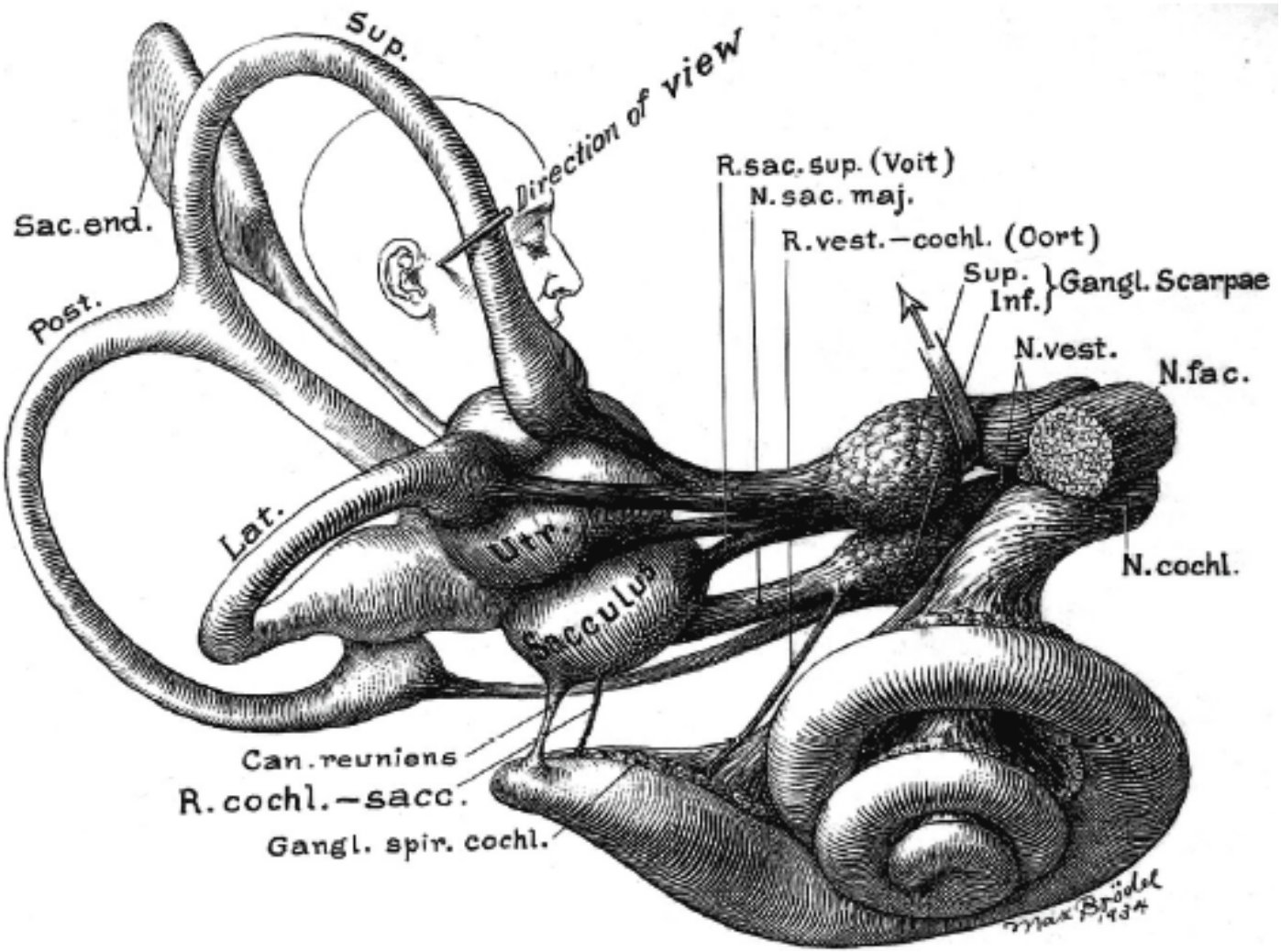
Part I Anatomy, Physiology and clinical implications

Peripheral Vestibular Anatomy and Physiology

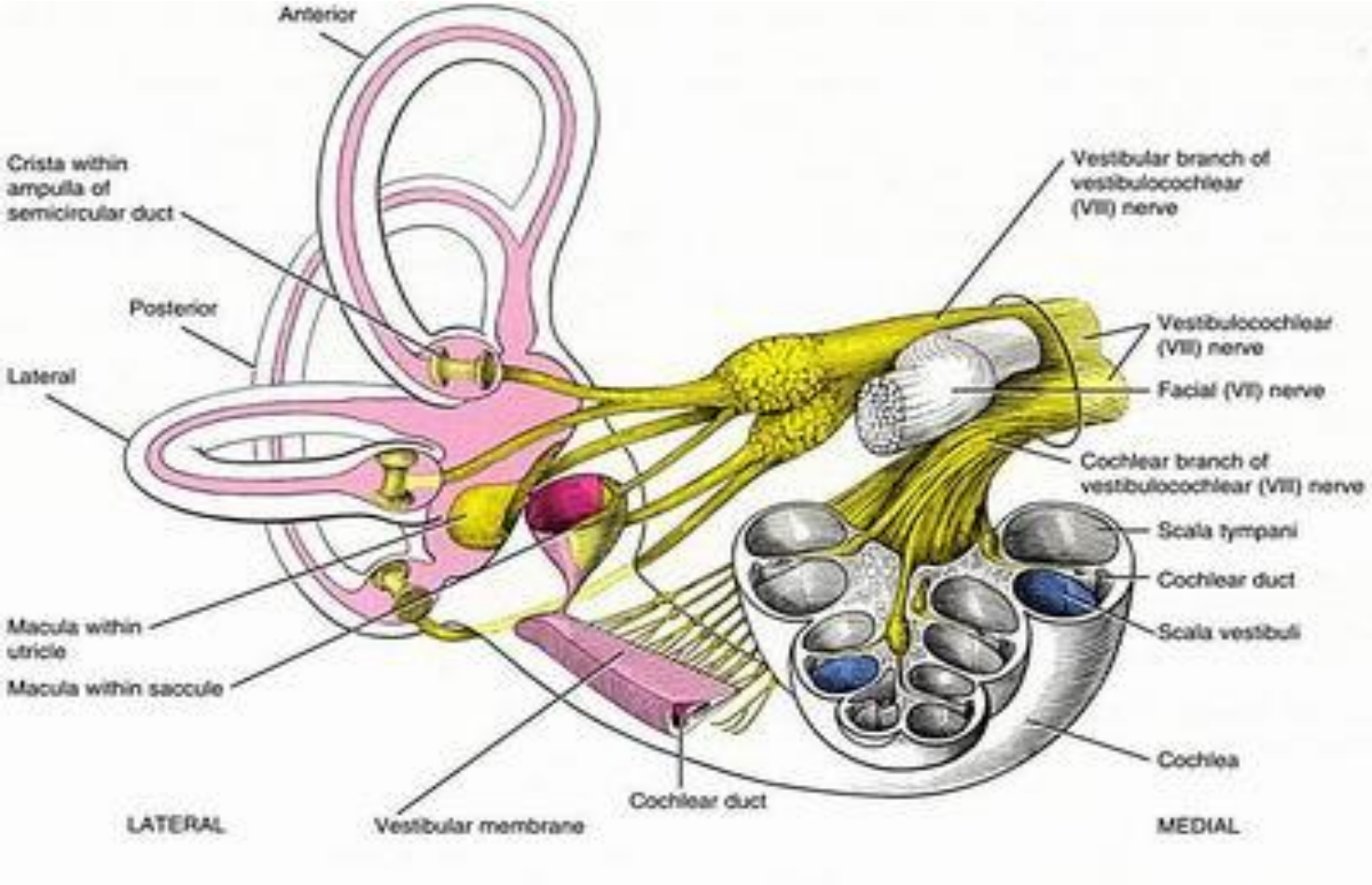


Peripheral Vestibular Anatomy and Physiology

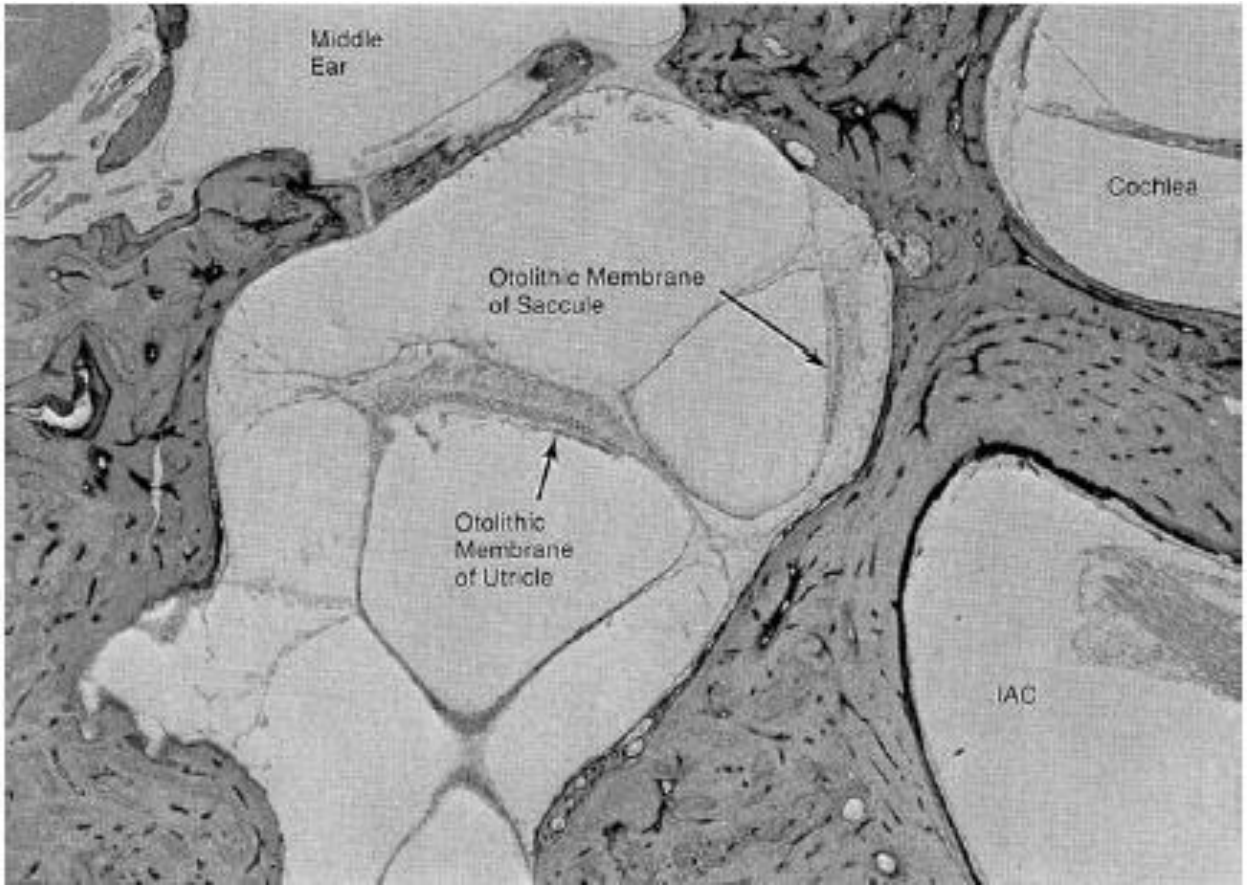


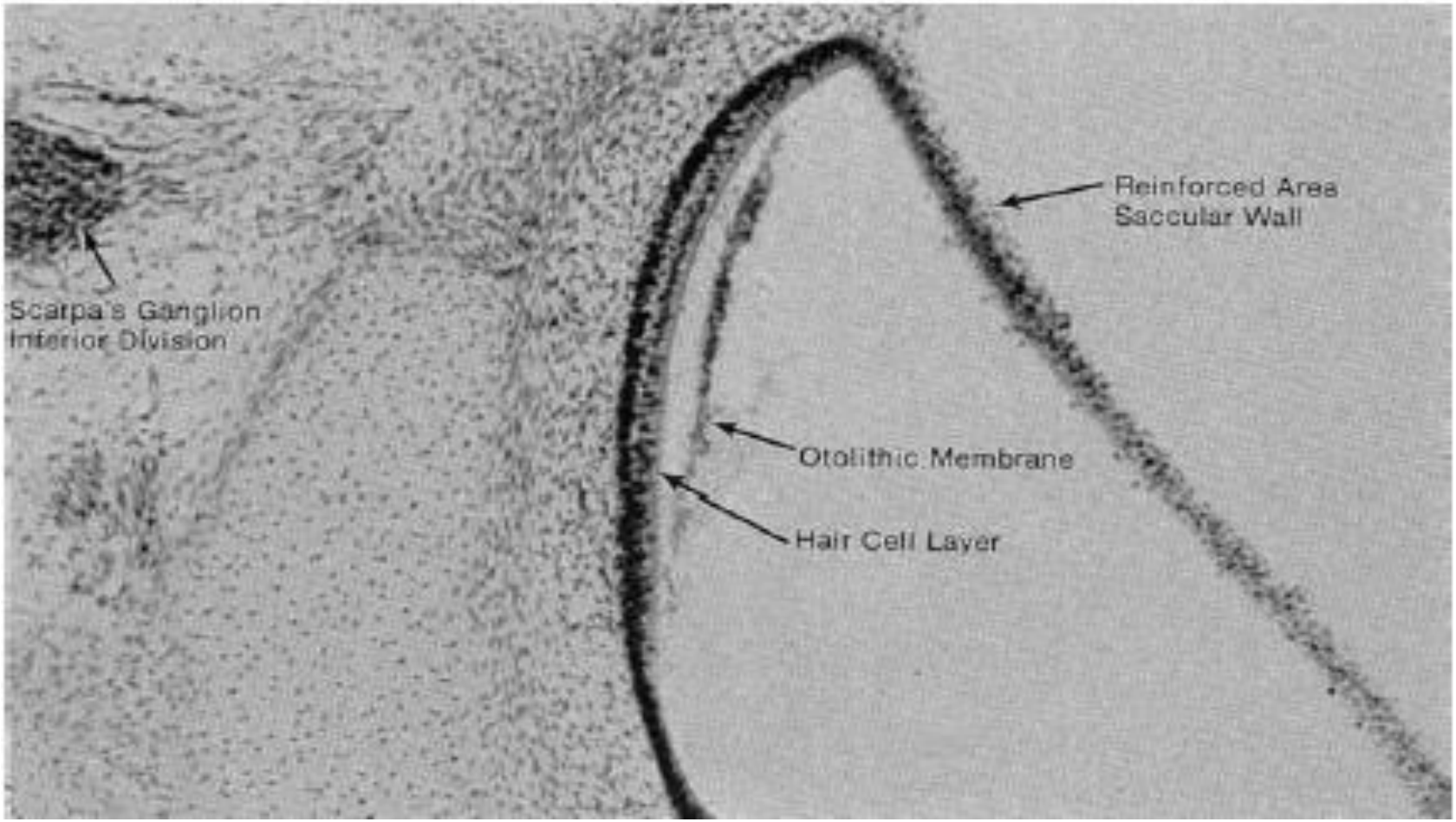

Sup.
Direction of view
Sac.end.
Post.
R.sac.sup. (Voit)
N.sac. maj.
R.vest.–cochl. (Oort)
Sup.
Inf.
Gangl. Scarpae
N.vest.
N.fac.
Lat.
Utr.
Sacculus
N.cochl.
Can. reuniens
R.cochl.–sacc.
Gangl. spir. cochl.
Max Brödel 1934

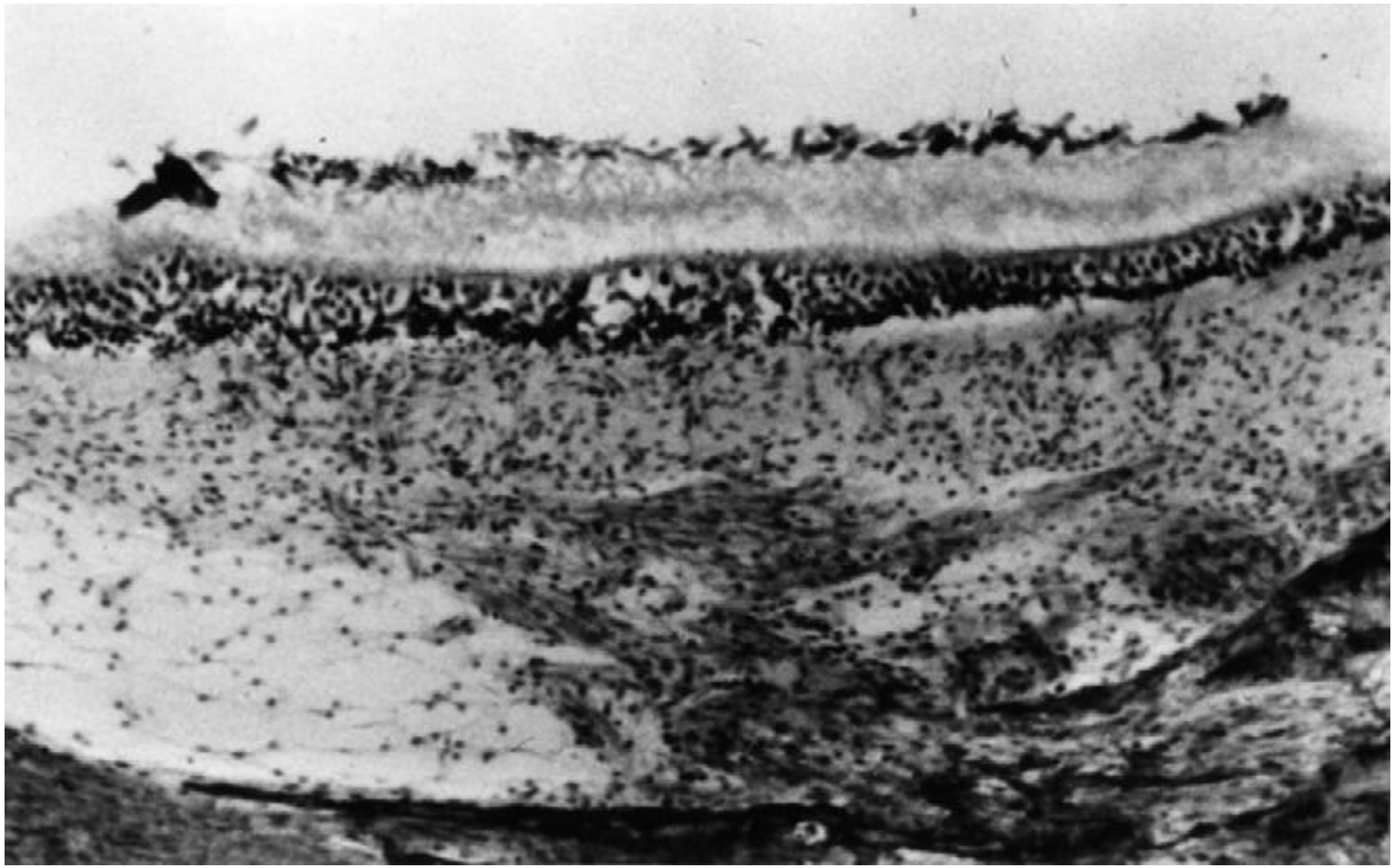
Peripheral Vestibular Anatomy and Physiology

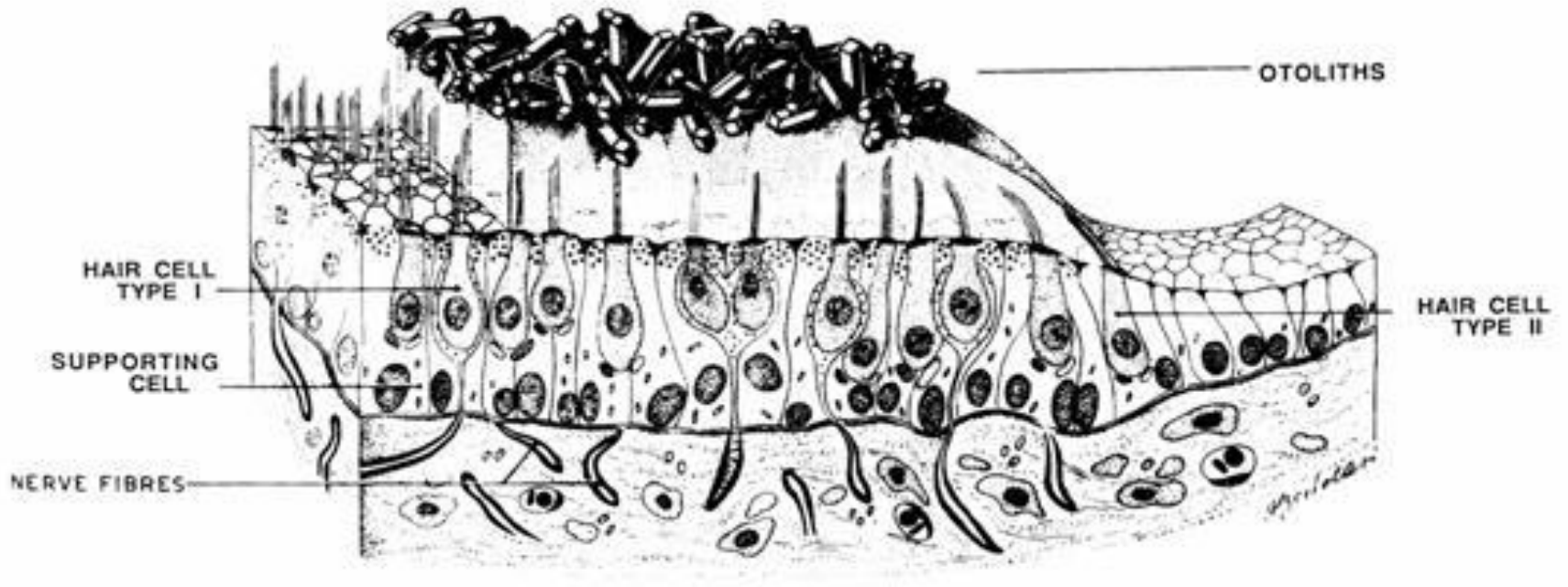


Otolithic Membranes

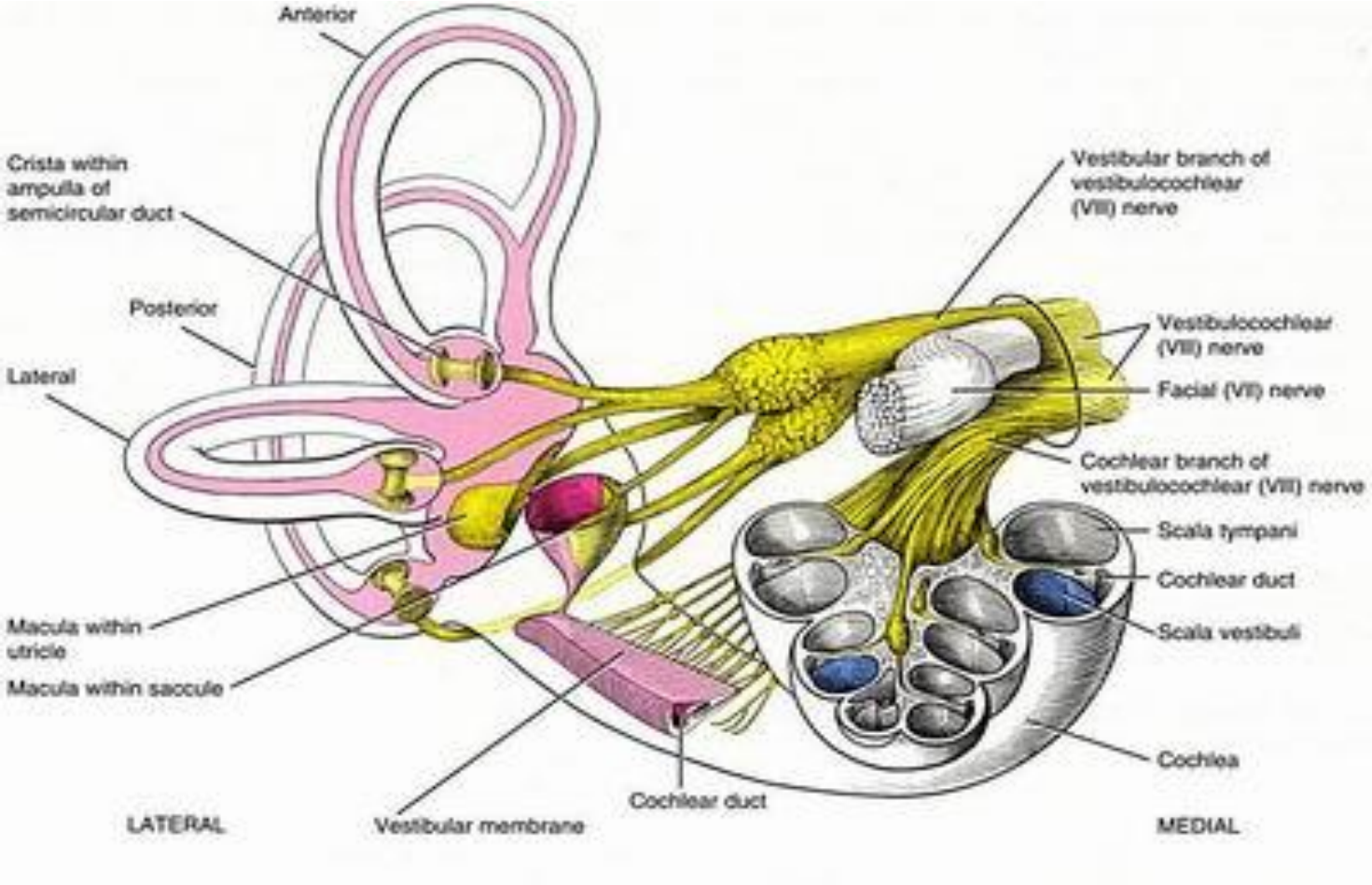



Reinforced Area
Saccular Wall
Scarpa's Ganglion
Inferior Division
Otolithic Membrane
Hair Cell Layer

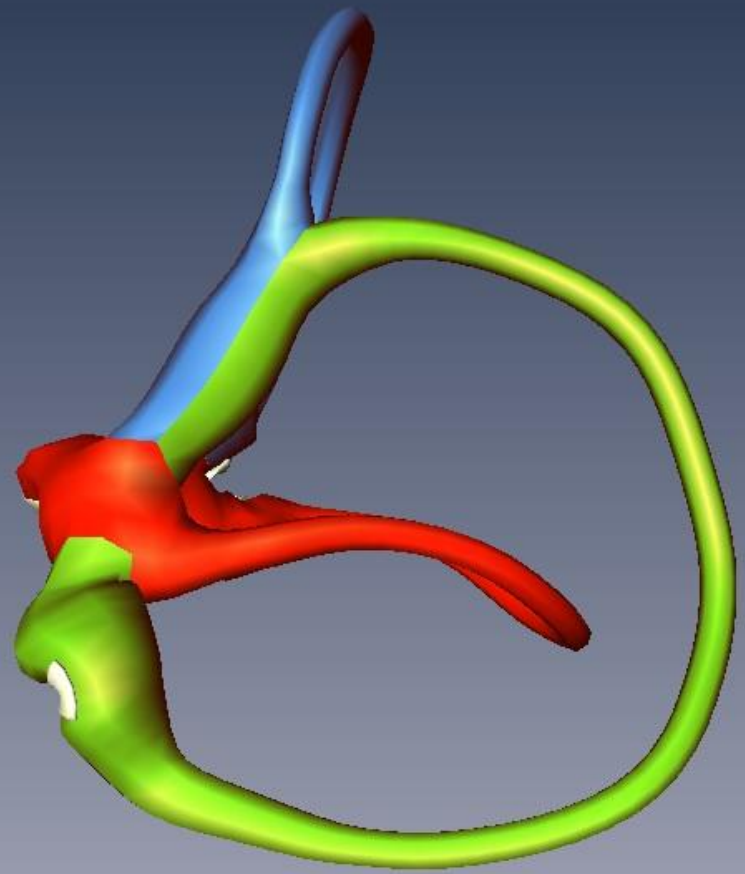



OTOLITHS
HAIR CELL TYPE I
SUPPORTING CELL
NERVE FIBRES
HAIR CELL TYPE II

Peripheral Vestibular Anatomy and Physiology



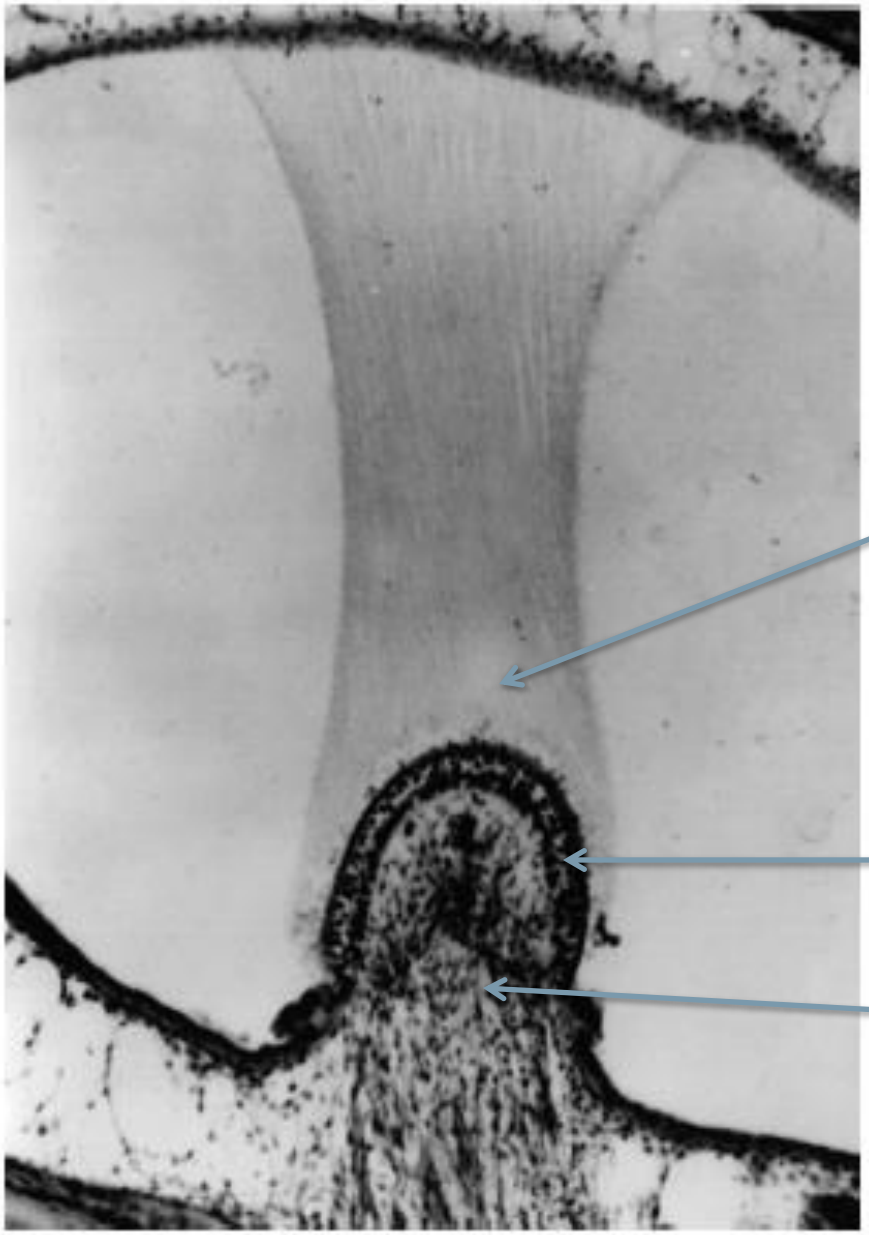
Courtesy of Michael Teixido



Labyrinth placed in 3D network with MRI based skull anatomy;
Amira 6.5 (Mercury Computer Systems, Chelmsford, MA, USA)

Labyrinth cloned / positioned relative to skull

Skin surface applied

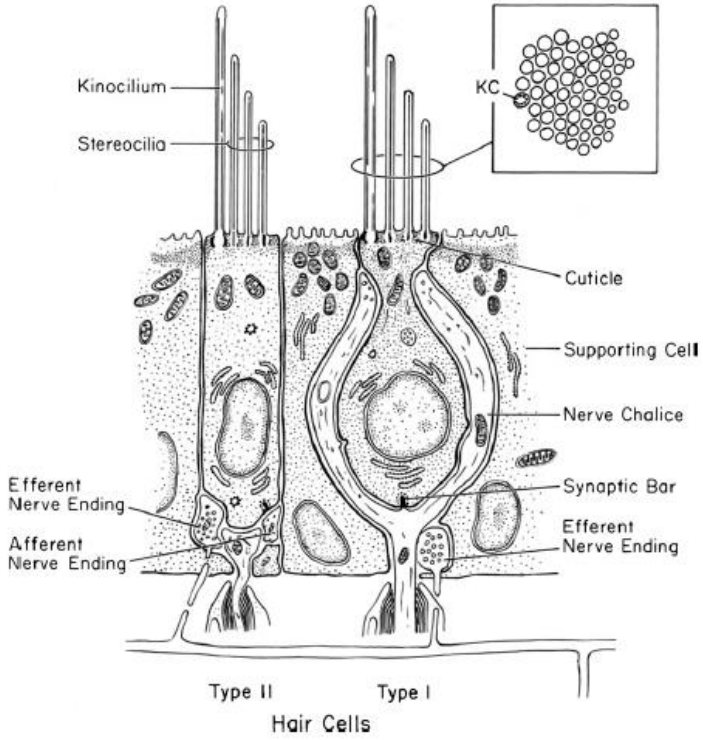


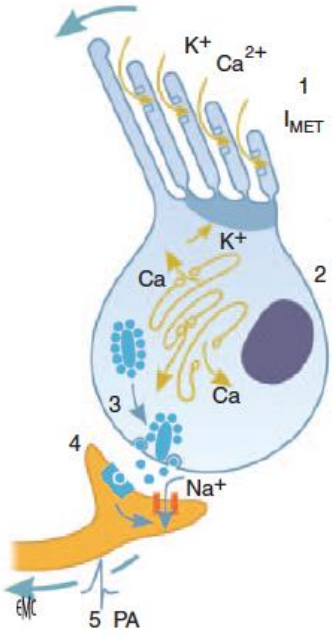
CUPULA

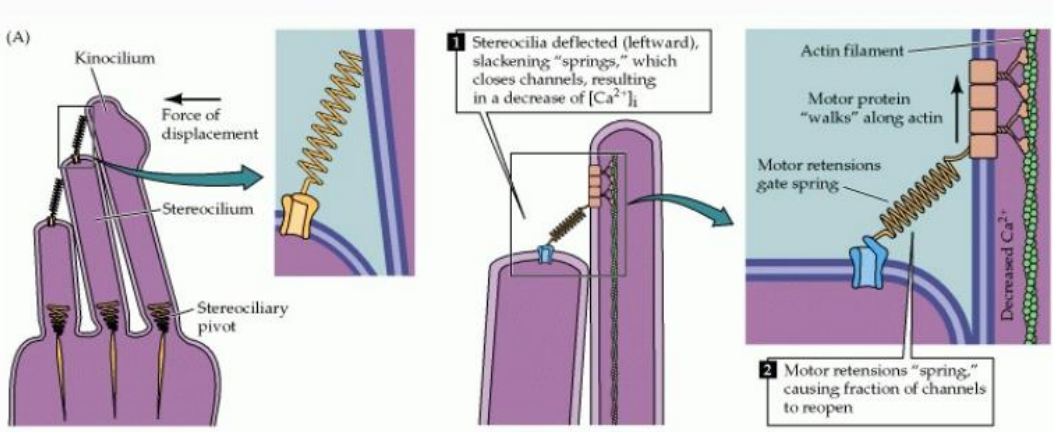
CRISTA AMPULLARIS

AMPULLARY NERVE

Peripheral Vestibular Anatomy and Physiology



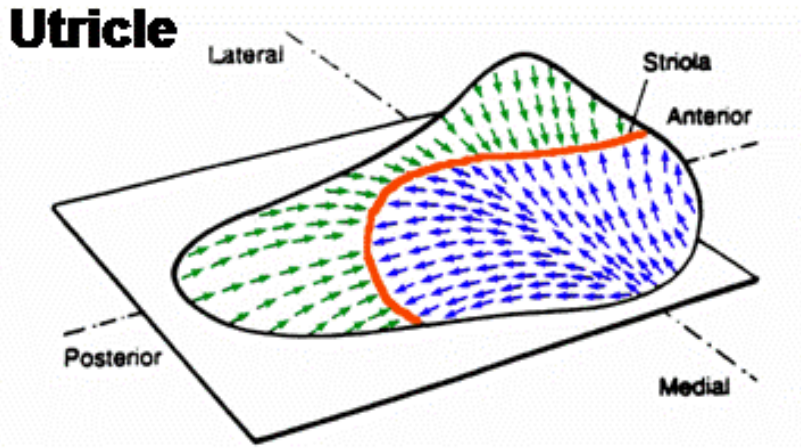




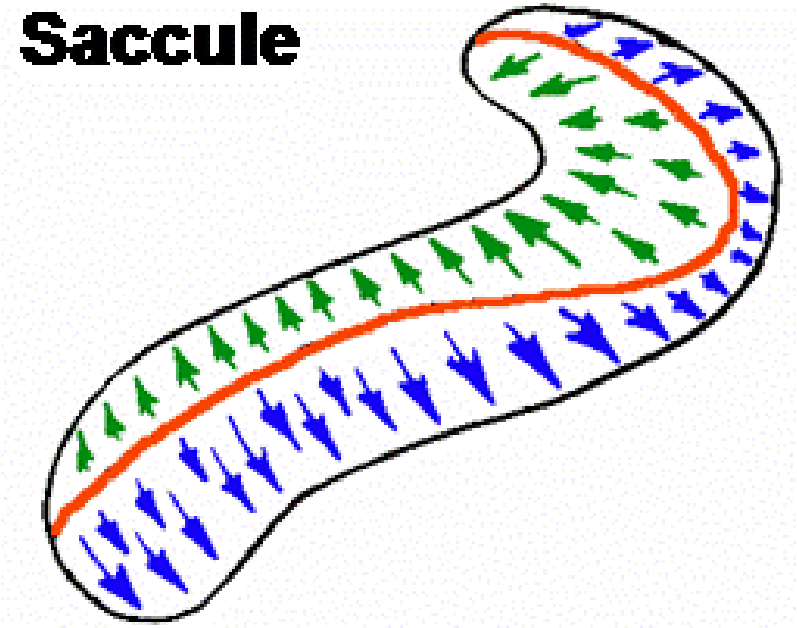
(A) Adaptation is explained in the gating spring model by adjustment of the insertion point of tips links. Movement of the insertion point up or down the shank of the stereocilium, perhaps driven by a Ca^{2+}-dependent protein motor, can continually adjust the resting tension of the tip link. (After Hudspeth and Gillespie, 1994.)

Peripheral Vestibular Anatomy and Physiology

Kinocilium oriented toward Striola



Kinocilium oriented away from striola



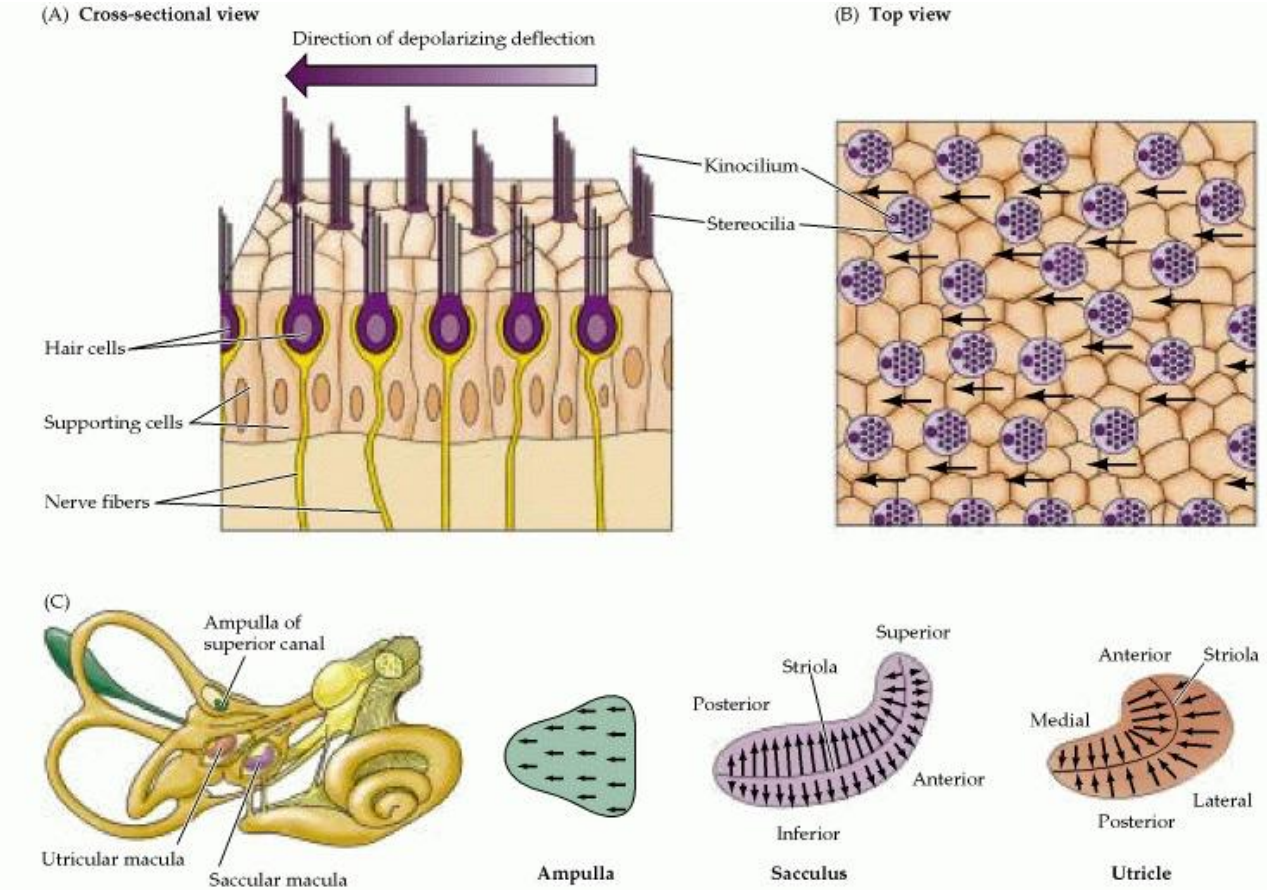


Figure 14.2

The morphological polarization of vestibular hair cells and the polarization maps of the vestibular **organs**. (A) A cross section of hair cells shows that the kinocilia of a group of hair cells are all located on the same side of the hair cell. The arrow indicates the direction of deflection that depolarizes the hair cell. (B) View looking down on the hair bundles. (C) In the ampulla located at the base of each semicircular canal, the hair bundles are oriented in the same direction. In the **sacculus** and **utricle**, the striola divides the hair cells into populations with opposing hair bundle polarities.

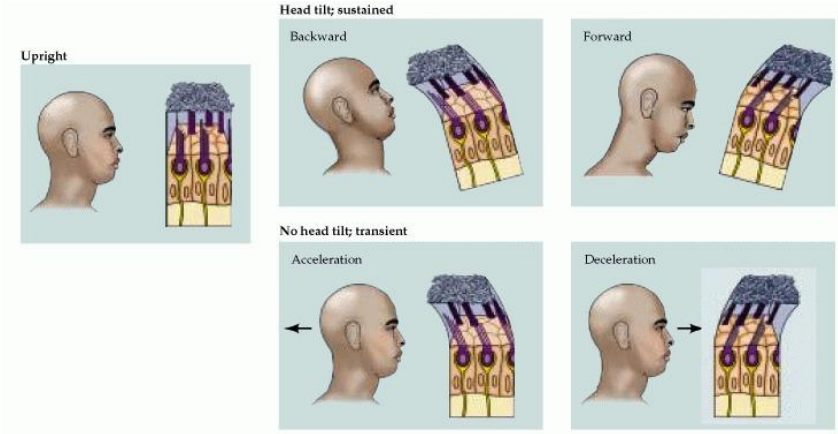


Figure 14.5

Forces acting on the head and the resulting displacement of the **otolithic membrane** of the utricular macula. For each of the positions and accelerations due to translational movements, some set of hair cells will be maximally excited, whereas another set will be maximally inhibited. Note that head tilts produce displacements similar to certain accelerations.

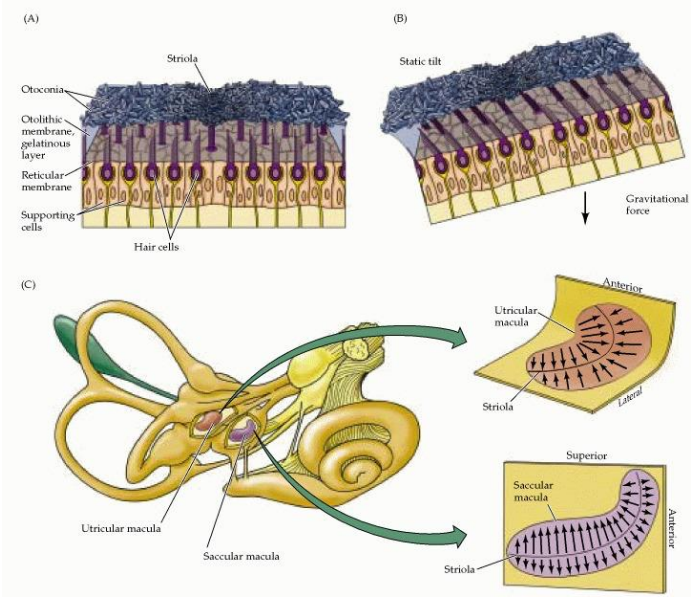
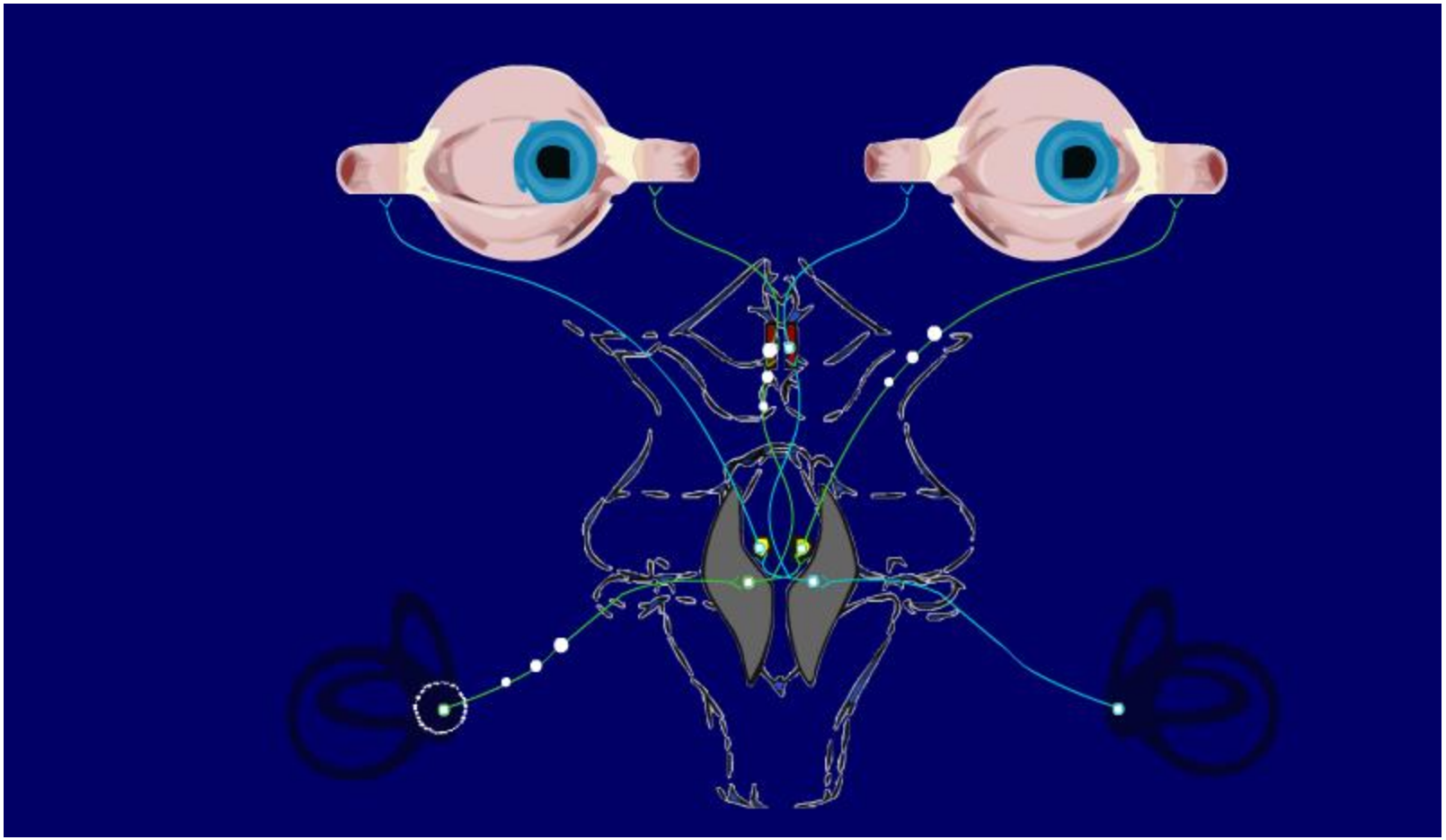


Figure 14.4

Morphological polarization of hair cells in the utricular and saccular maculae. (A) Cross section of the utricular macula showing hair bundles projecting into the gelatinous layer when the head is level. (B) Cross section of the utricular macula when the head is tilted. (C) Orientation of the utricular and saccular maculae in the head; arrows show orientation of the kinocilia, as in Figure 14.2. The *saccules* on either side are oriented more or less vertically, and the *utricles* more or less horizontally. The striola is a structural landmark consisting of small otoconia arranged in a narrow trench that divides each **otolith** organ. In the utricular macula, the kinocilia are directed toward the striola. In the saccular macula, the kinocilia point away from the striola. Note that, given the **utricle** and **sacculus** on both sides of the body, there is a continuous representation of all directions of body movement.

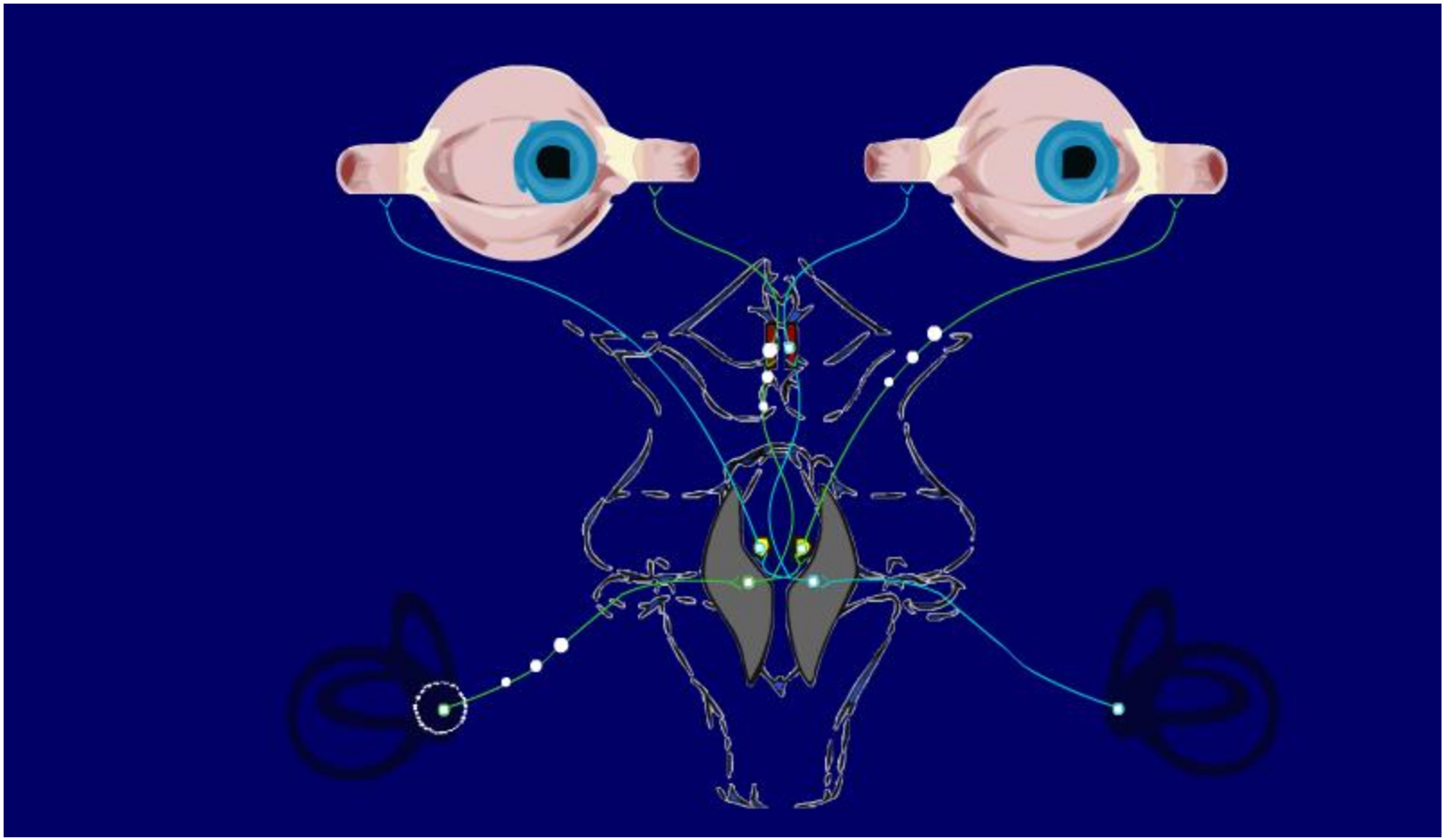
Remember: Both vestibular systems are working when we are not moving

Tonic function of vestibular organs



Courtesy of Dr Manuel Oliva-Domínguez ENT Department Hospital Costa del Sol, Marbella (Málaga)Normal VOR

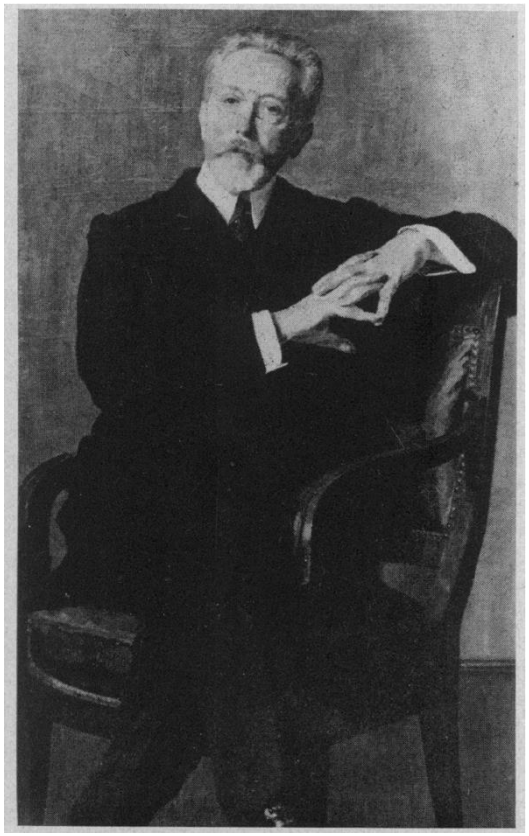
Tonic function of vestibular organs



Courtesy of Dr Manuel Oliva-Domínguez ENT Department Hospital Costa del Sol, Marbella (Málaga)Left vestibular hypofunction or Right vestibular Hyperfunction

Semicircular canals and Balance
Flourens 1842 Ewald 1892

Ewald's laws



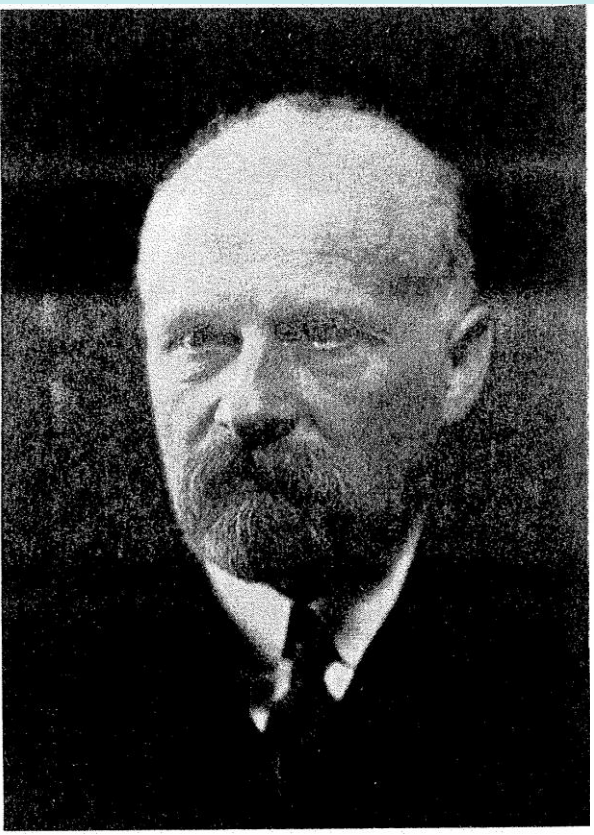
EWALD'S THREE LAWS

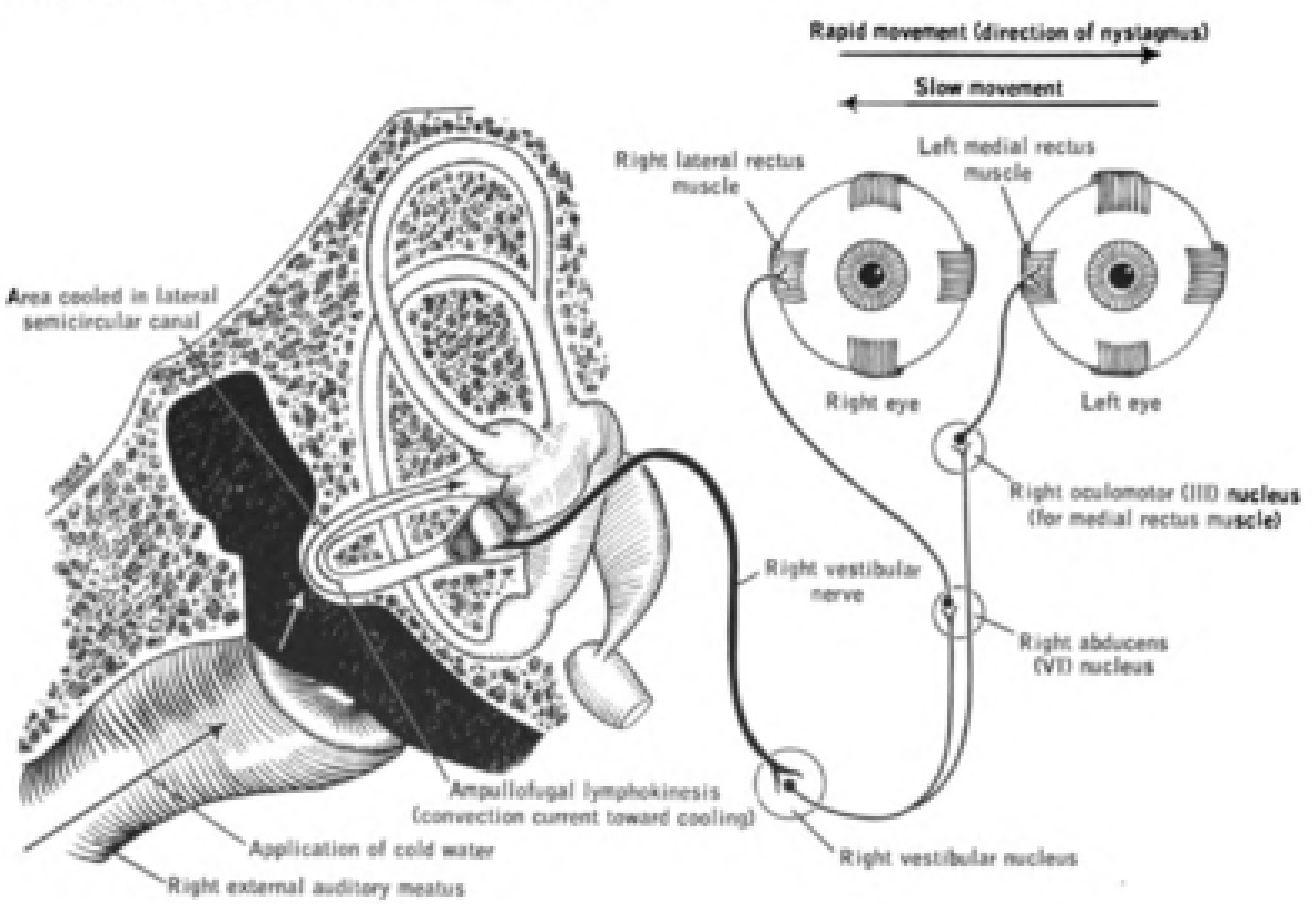
1. A stimulation of the semicircular canal causes a movement of the eyes in the plane of the stimulated canal : **tonic movements**(slow phase of nystagmus and segmental and axial tilts) are in the **direction of the endolymph movement**

2. In the *horizontal* semicircular canals, an *ampullopetal* endolymph movement causes a *greater stimulation* than an ampullofugal one. **Ampullopetal flow is utriculopetal**

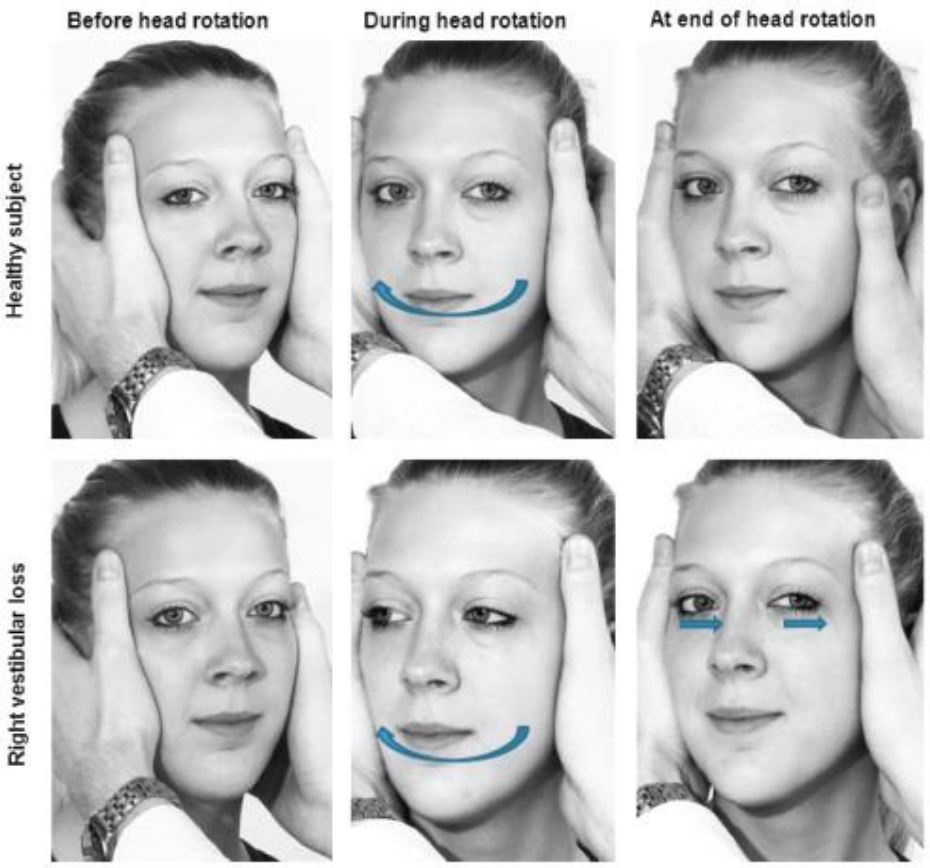
3. In the *vertical* semicircular canals, the reverse is true. **Ampullopetal flow is utriculofugal**

Robert Bárány XXth Century





Head Impulse Test (bHIT)**



**Petersen JA, Straumann D and Weber K, Clinical Diagnosis of bilateral vestibular loss: three simple bedside tests, Ther Adv Neurol Disord, 2013,6: 41-45

VOR SUPPRESSION by visual fixation

Brandt T and Strupp M, General Vestibular Testing, Clin Neurophys, 2005, 116: 406-426

Dix, M.R.; Hallpike, C.S.: The pathology, symptomatology and diagnosis of certain common disorders of the vestibular system. Proc. R. Soc. Med. *45:* 341–354 (1952).

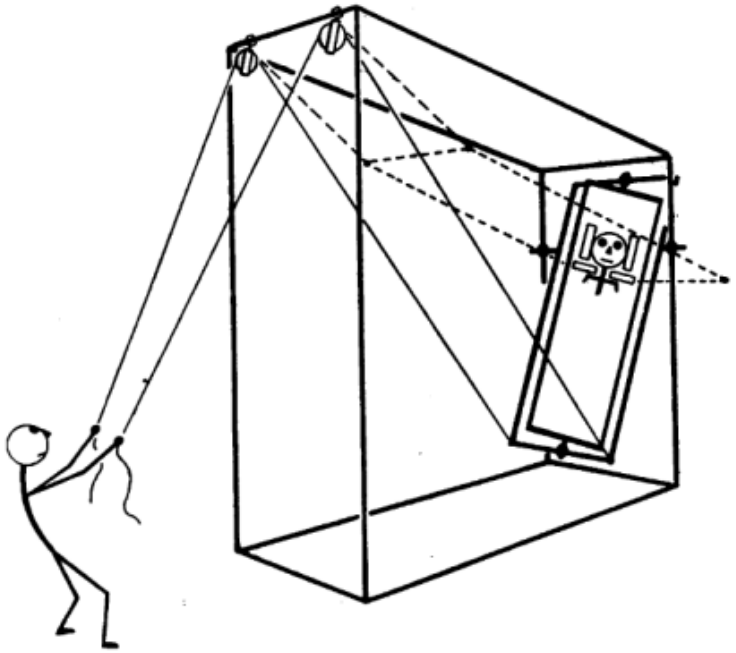
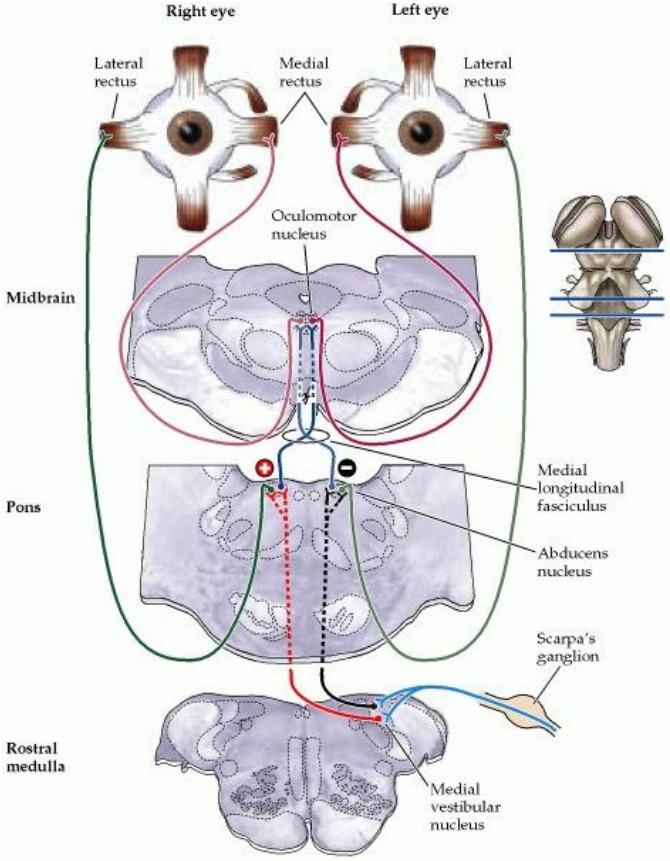


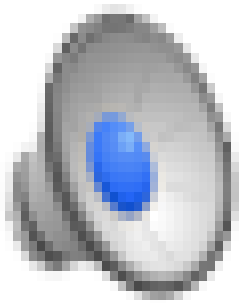
FIG. 7.

By means of apparatus shown in Fig. 7 it is possible to move the patient *en masse* into the critical position,



	Excitatory Pathway	Inhibitory Pathway
Superior SCC	• Bilateral Superior Recti • Contralateral Inferior oblique	• Bilateral Inferior Recti • Contralateral Superior Oblique
Posterior SCC	• Ipsilateral superior oblique • Bilateral Inferior Recti	• Ipsilateral inferior oblique • Bilateral Superior Recti
Lateral SCC	• Contralateral lateral Rectus • Ipsilateral Medial Rectus	• Contralateral Medial Rectus • Ipsilateral Lateral Rectus

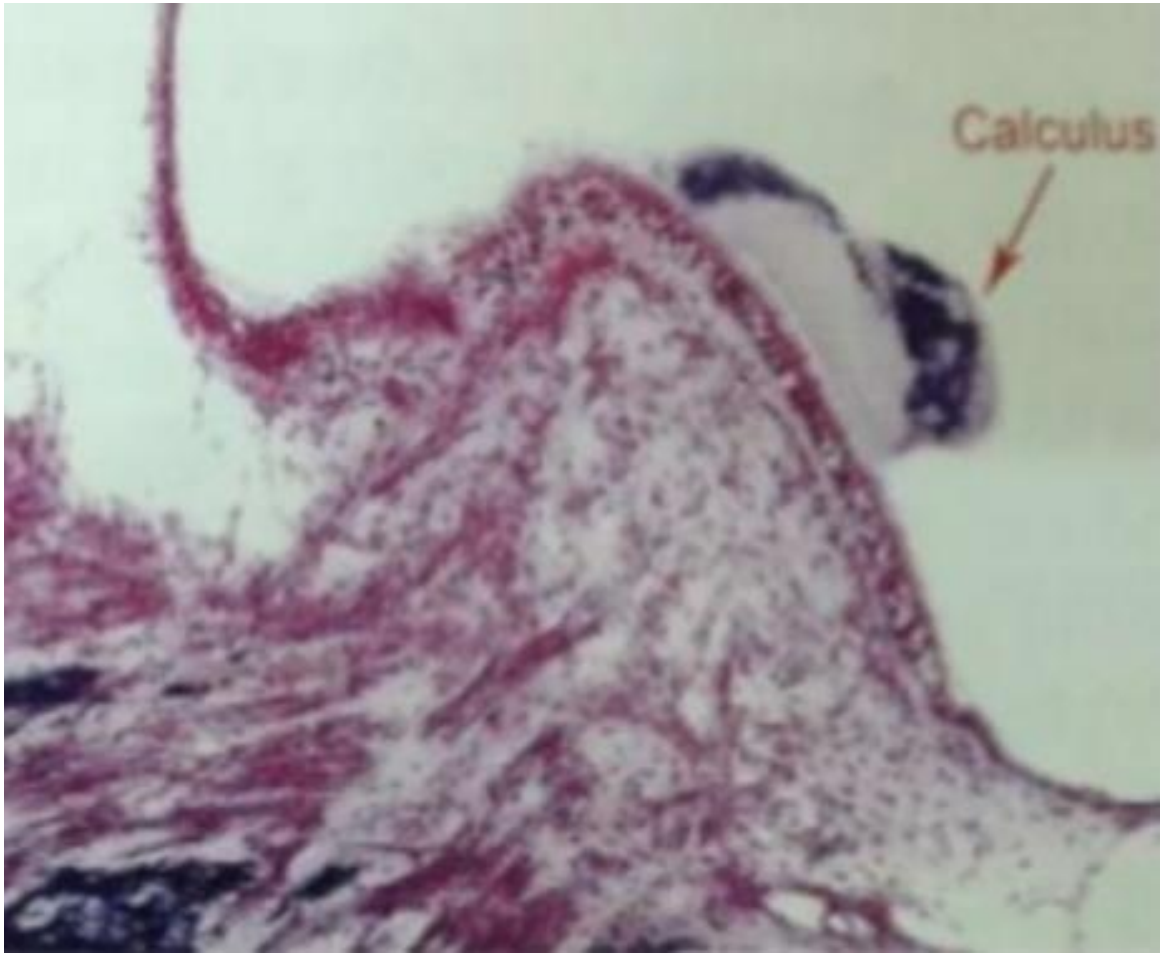
Posterior Canal BPPV



Superior oblique muscle: depressor when eye is adducted and intorter when abducted

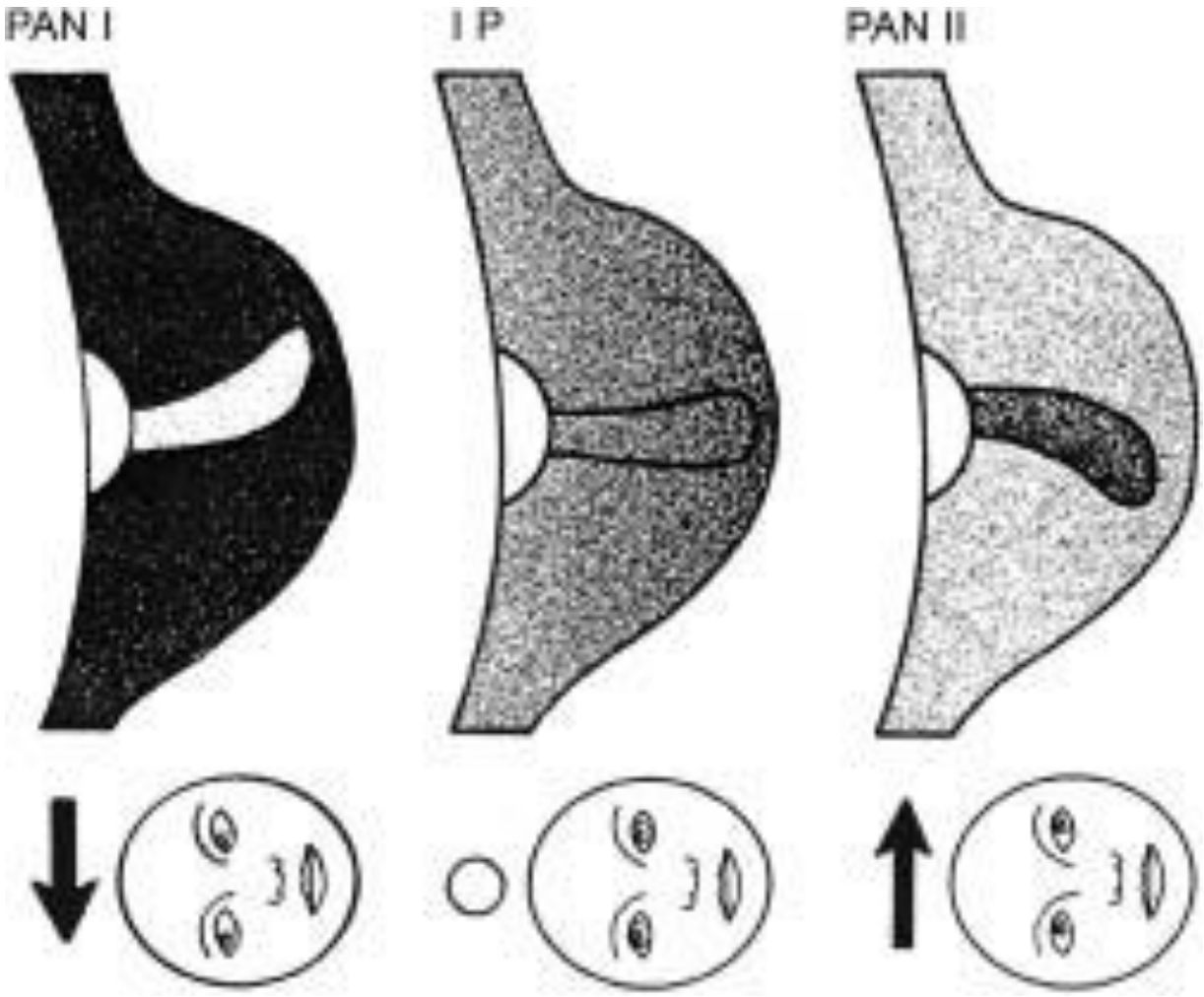
Left Posterior BPPV

- NEUTRAL GAZE: TYPICAL UPBEAT GEOTROPIC NYSTAGMUS WITH VERTICAL AND ROTARY NYSTAGMUS
- LEFT GAZE: PURE GEOTROPIC NYSTAGMUS ROTARY COMPONENT ONLY
- RIGHT GAZE: PURE UPBEAT NYSTAGMUS VERTICAL COMPONENT ONLY

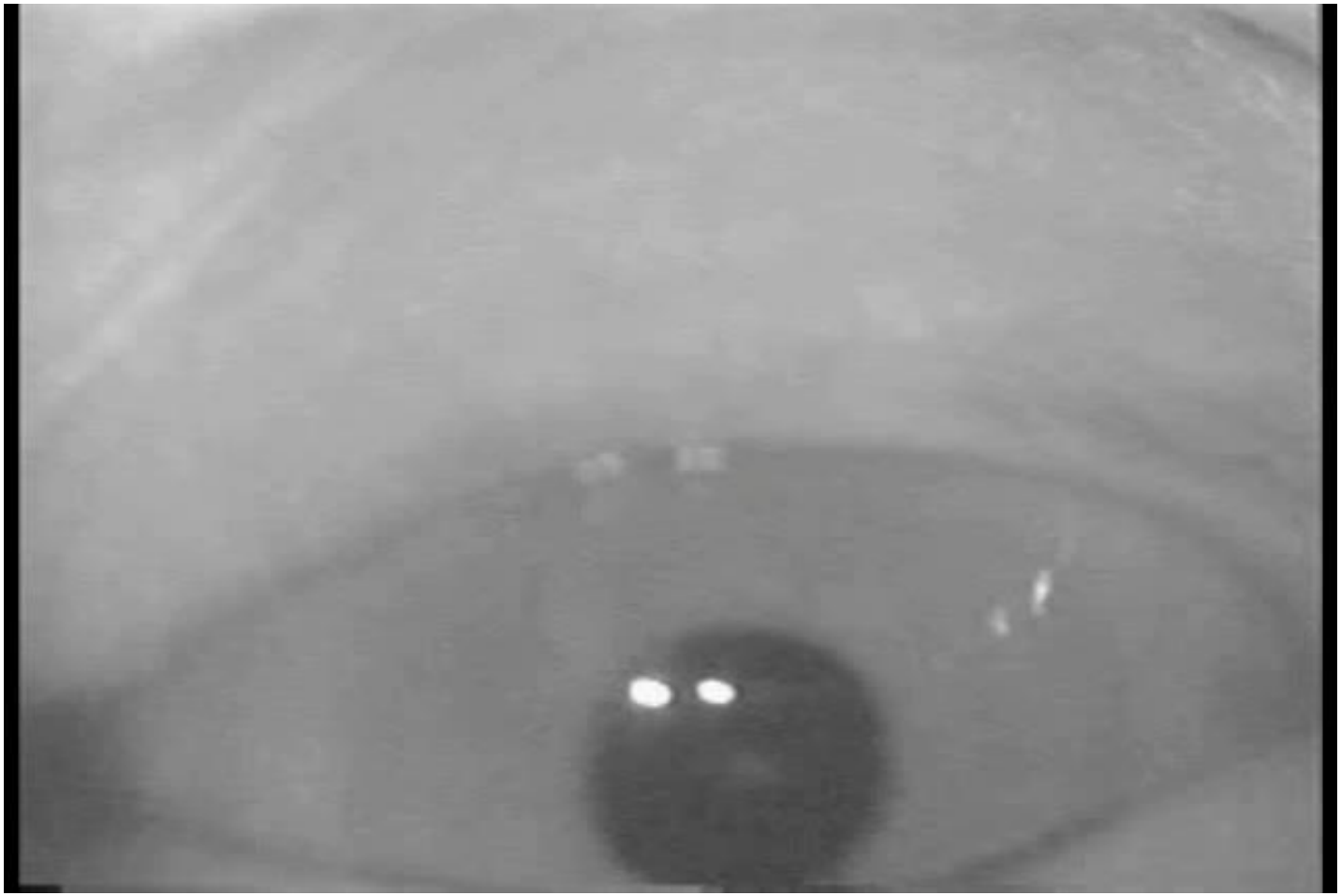


Arch Otolaryngol 1969: 90, 765

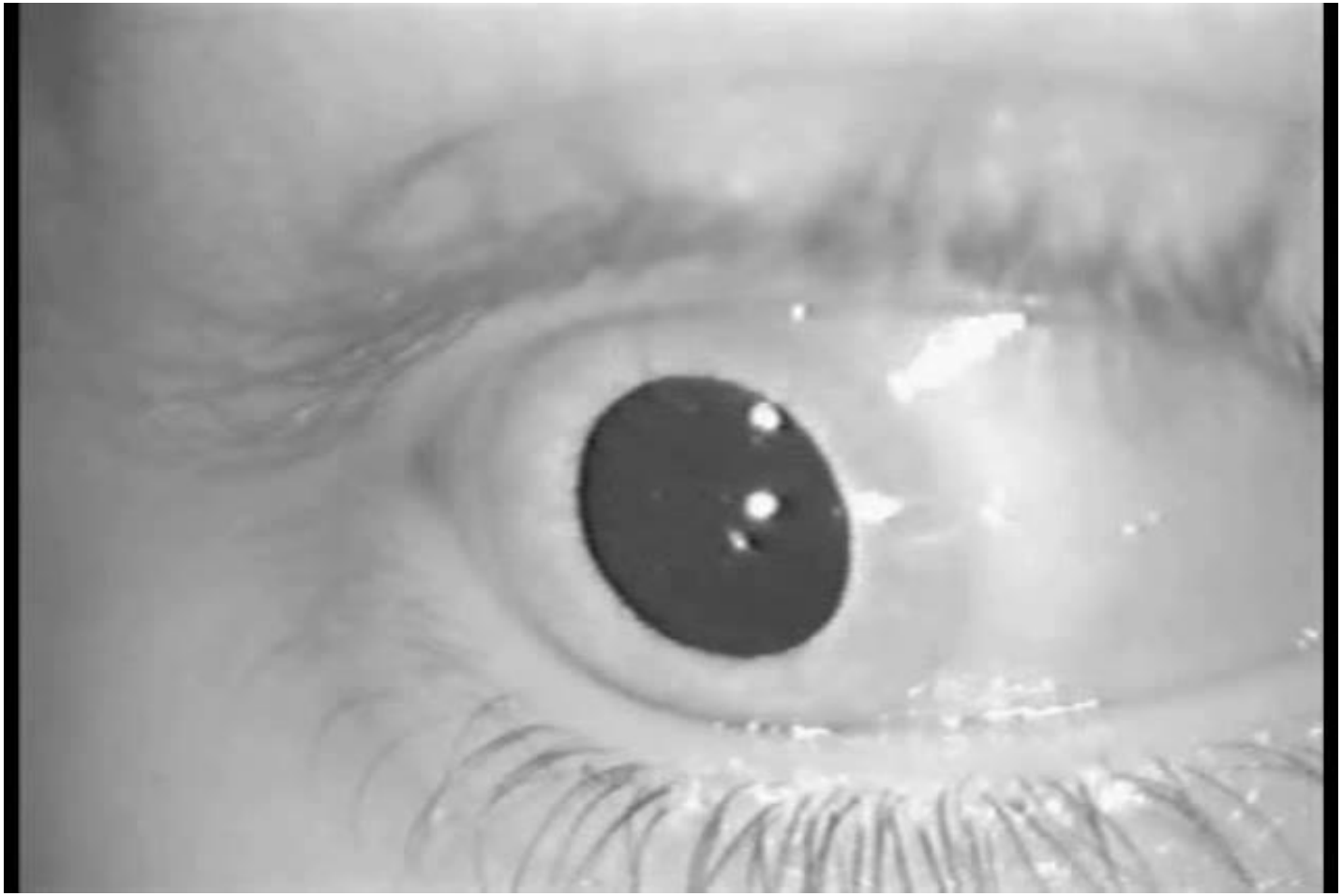
Buoyancy Cupulopathy



Horizontal BPPV



Superior BPPV



Excitation of right and left superior recti and inhibition of right and left inferior recti
Excitation of left contralateral inferior oblique and inhibition of left contralateral superior oblique:

ELEVATES THE LEFT EYE WHEN IT IS ADDUCTED

Define the symptoms

Lightheadedness/Presyncope	• **Sensation of impending loss of consciousness** • **Multiple sensory disturbances, orthostatic hypotension, cardiac arrhythmias and panic attacks**
Disequilibrium	• Imbalance or unsteadiness experience while standing or walking. • Caused by diminished vision, loss of vestibular function, defects in proprioception and motor dysfunction from the CNS and PNS. • Also related to visual vertigo, presyncopal faintness and somatoform phobic postural vertigo

Oscillopsia	• Subjective illusion of visual motion.
	• Happens ONLY when eyes are open:
	  • Acquired nystagmus can cause retinal slip.
	  • Bilateral vestibular loss

Vertigo	Illusion of movements of oneself or the environment: rotationally but not necessarily

Huh YE, Kim JS, Bedside Evaluation of dizzy patients, J Clin Neurol, 2013,9: 203-213

Journal of Vestibular Research 19 (2009) 1–13
DOI 10.3233/VES-2009-0343
IOS Press

Classification of vestibular symptoms: Towards an international classification of vestibular disorders

First consensus document of the Committee for the Classification of Vestibular Disorders of the Bárány Society

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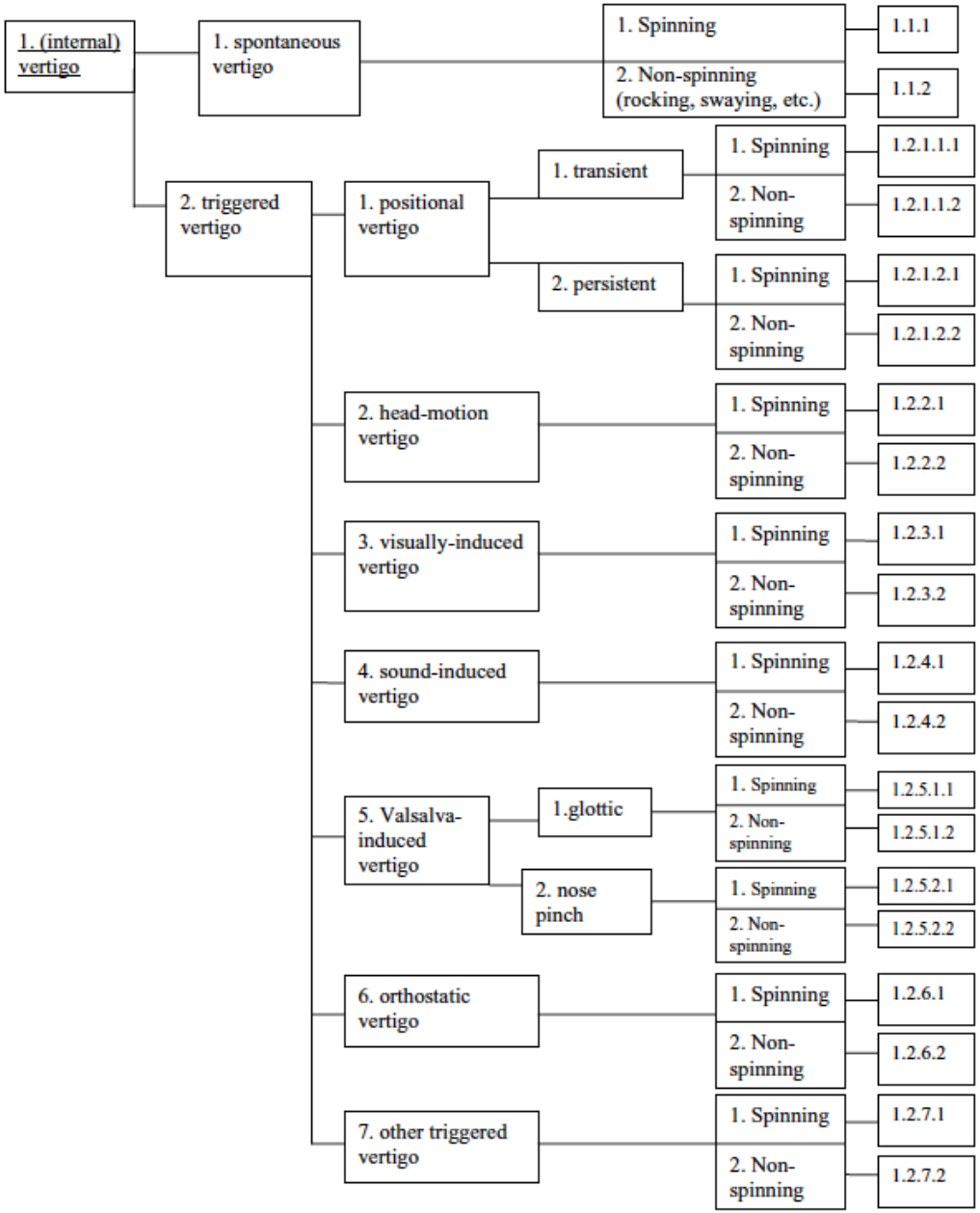
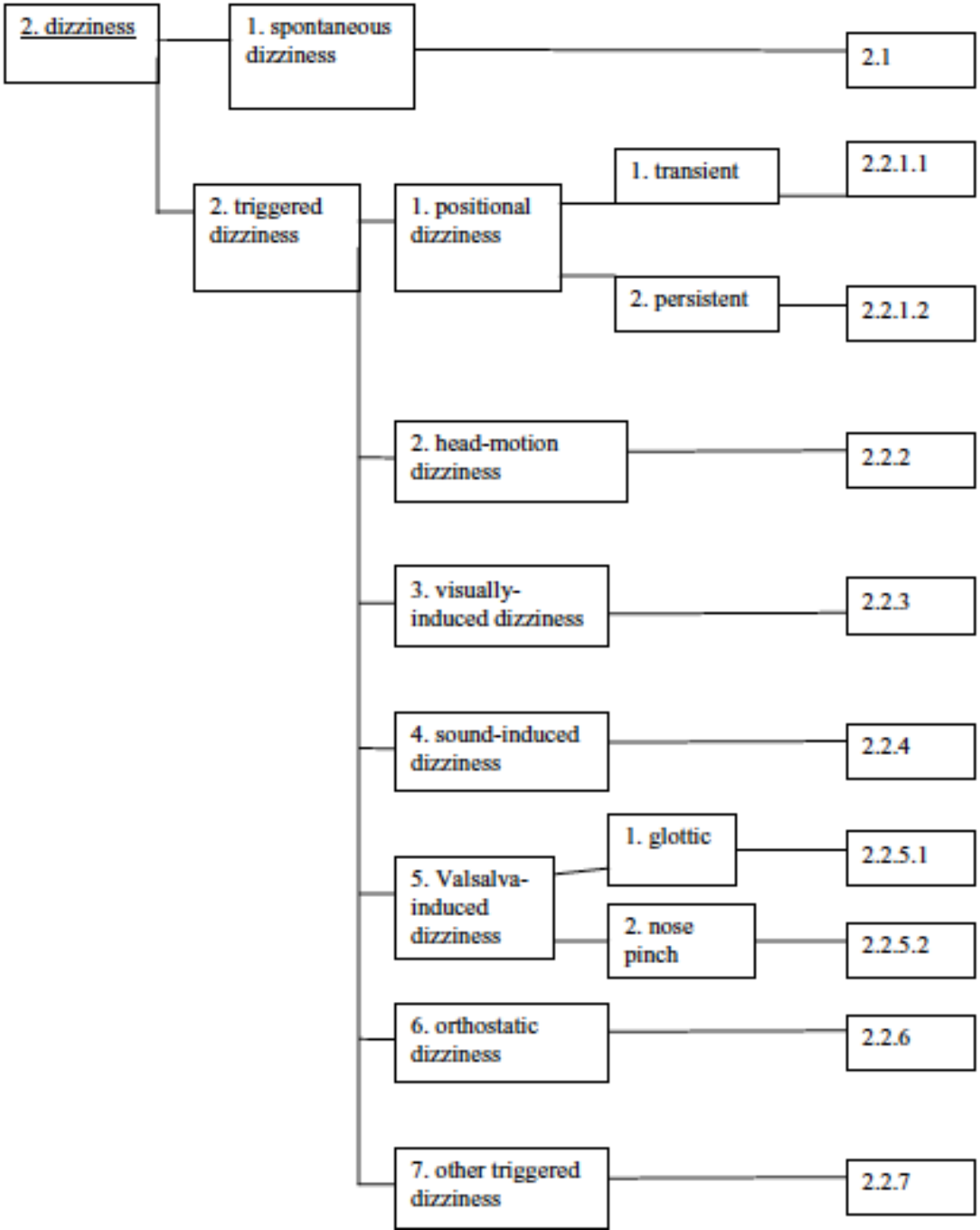
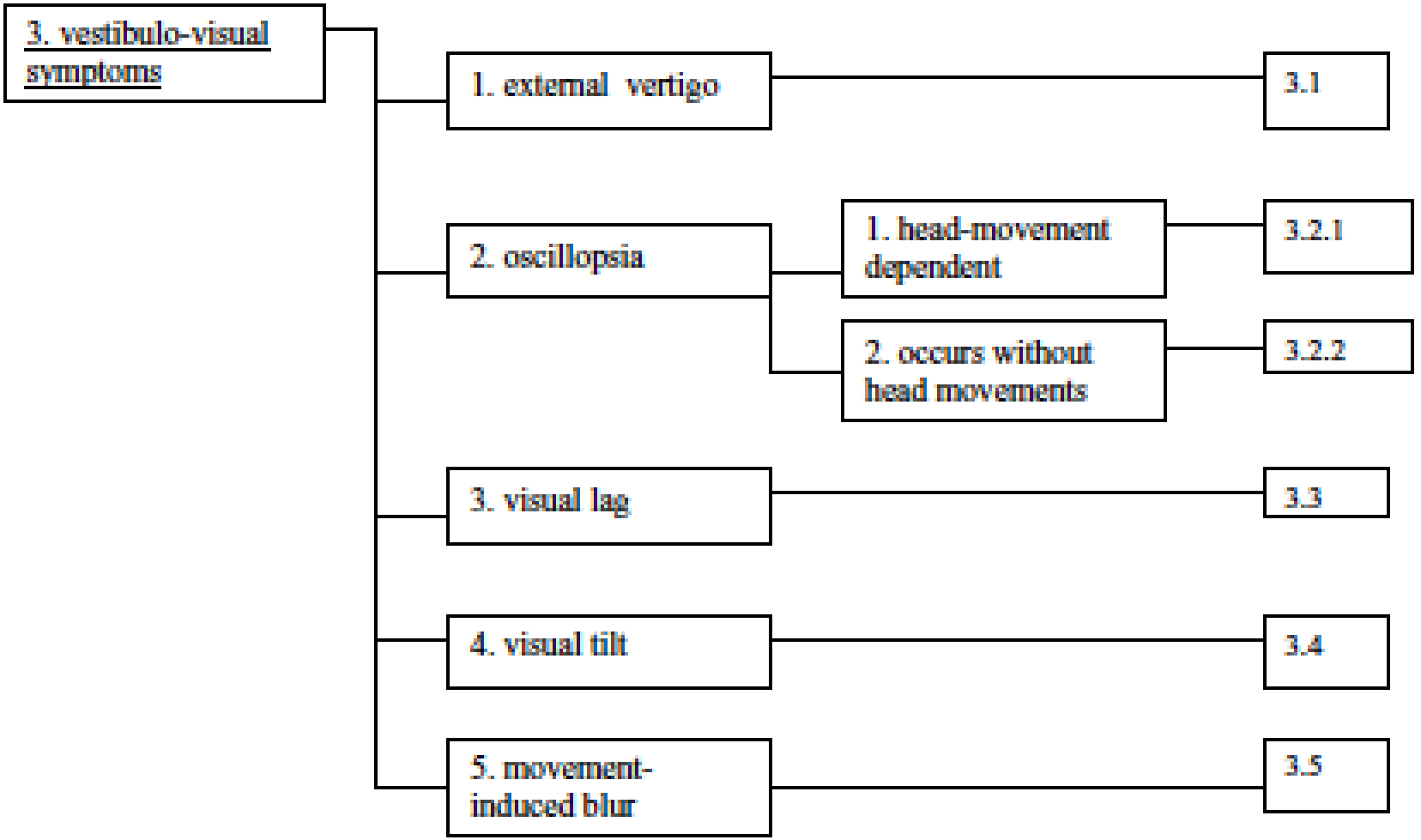
[c]*Vestibular Research Group Berlin, Department of Neurology, Schlosspark-Klinik, Berlin, Germany*
[d]*Department of Neurology, The Johns Hopkins University School of Medicine, Baltimore, MD 21287, USA*

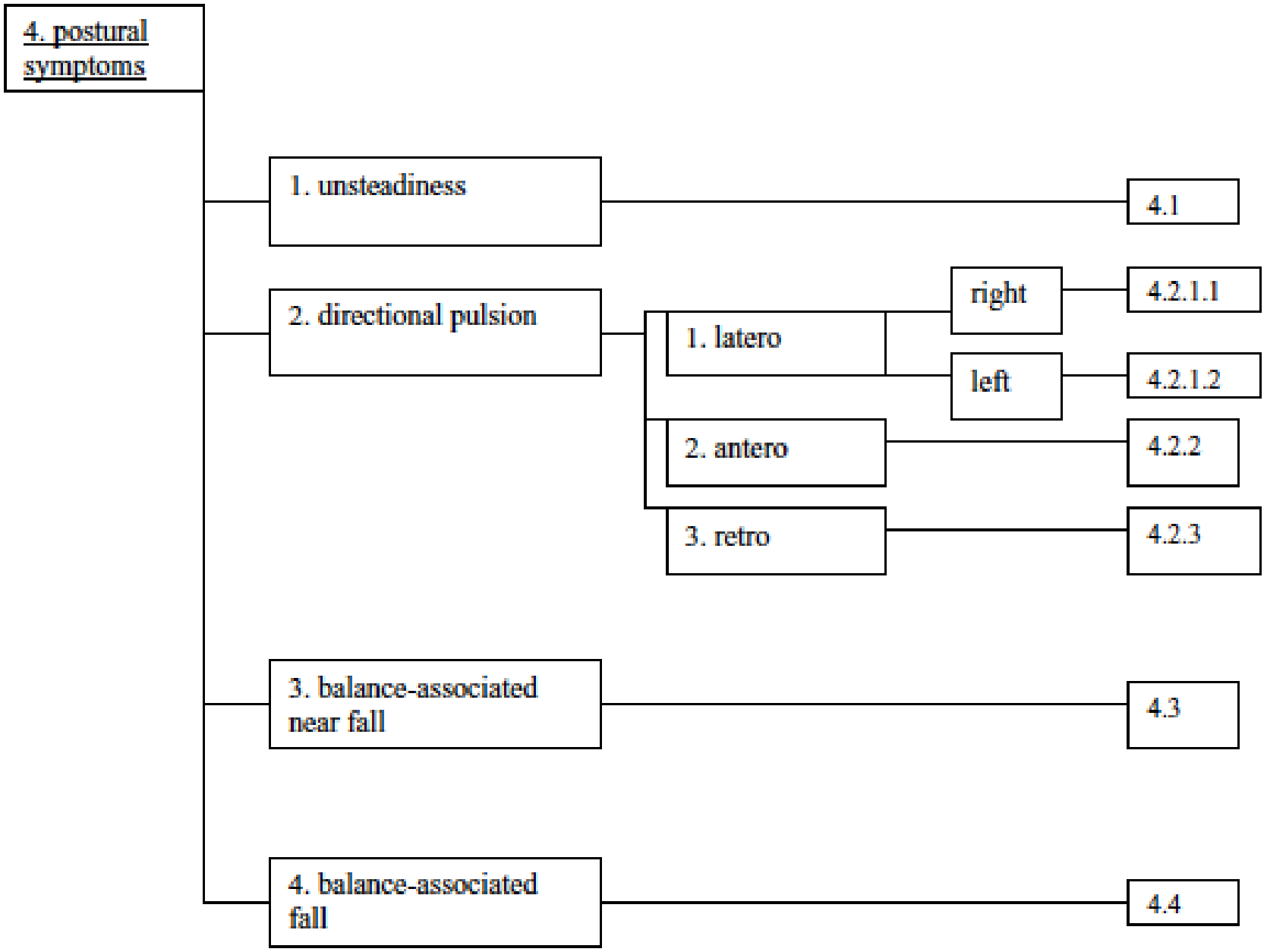
On behalf of the Committee for the Classification of Vestibular Disorders of the Bárány Society: Pierre Bertholon, Alexandre Bisdorff, Adolfo Bronstein, Herman Kingma, Thomas Lempert, Jose Antonio Lopez Escamez, Måns Magnusson, Lloyd B. Minor, David E. Newman-Toker, Nicolás Pérez, Philippe Perrin, Mamoru Suzuki, Michael von Brevern, John Waterston and Toshiaki Yagi

Appendix 2. Symptom Coding Algorithm



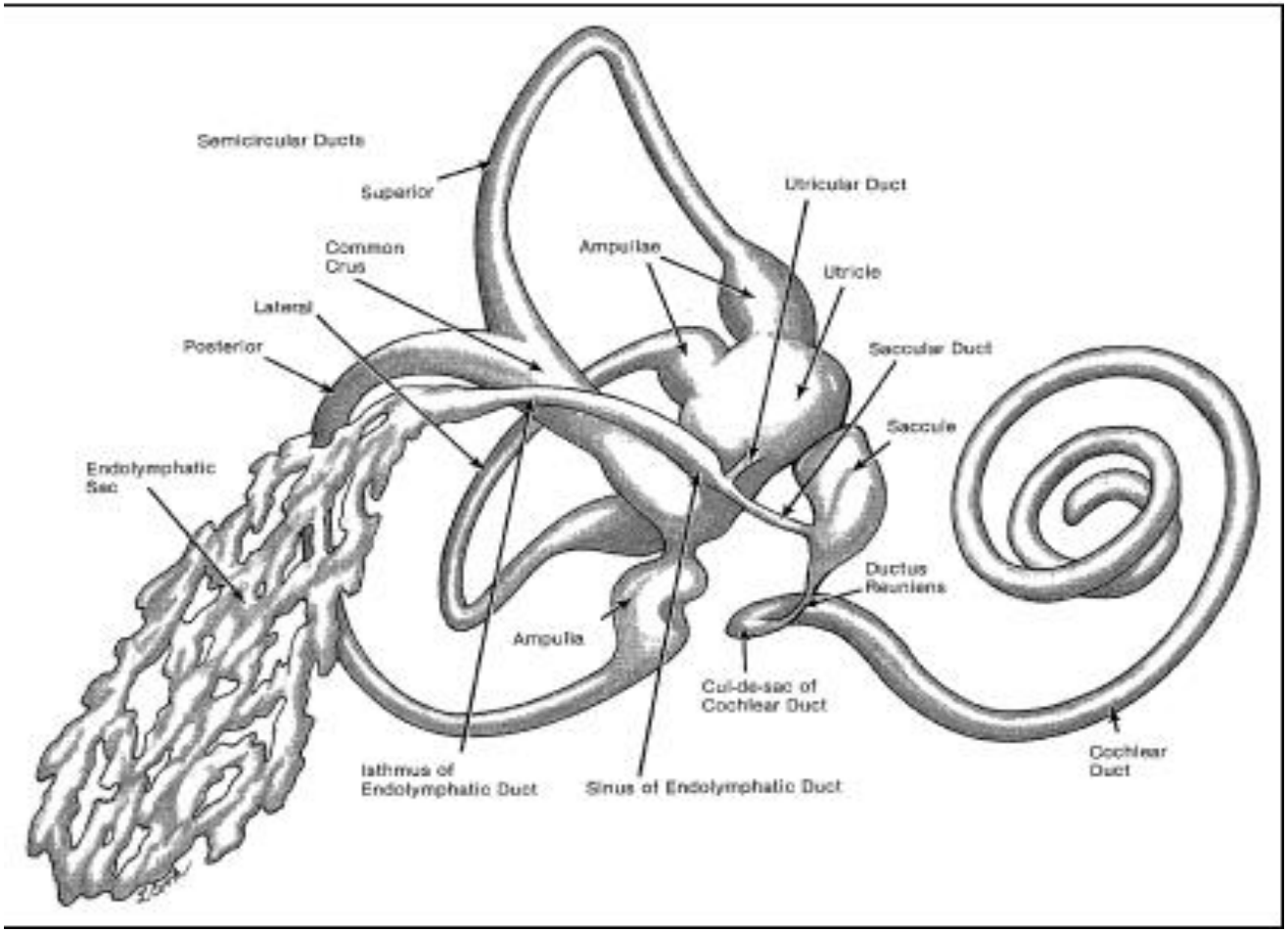

2. dizziness
1. spontaneous dizziness
2.1
2. triggered dizziness
1. positional dizziness
1. transient
2.2.1.1
2. persistent
2.2.1.2
2. head-motion dizziness
2.2.2
3. visually-induced dizziness
2.2.3
4. sound-induced dizziness
2.2.4
5. Valsalva-induced dizziness
1. glottic
2.2.5.1
2. nose pinch
2.2.5.2
6. orthostatic dizziness
2.2.6
7. other triggered dizziness
2.2.7


3. vestibulo-visual symptoms
1. external vertigo
3.1
2. oscillopsia
1. head-movement dependent
3.2.1
2. occurs without head movements
3.2.2
3. visual lag
3.3
4. visual tilt
3.4
5. movement-induced blur
3.5

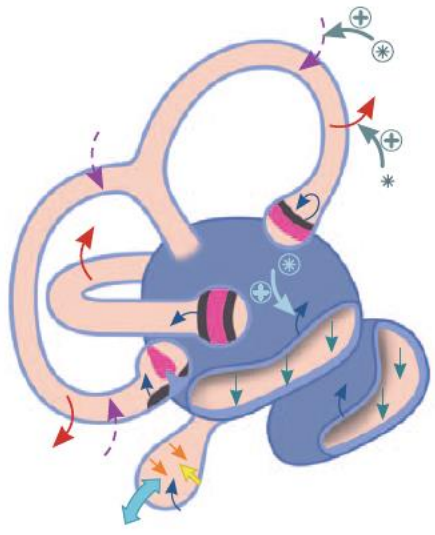

4. postural symptoms
1. unsteadiness
4.1
2. directional pulsion
1. latero
right
4.2.1.1
left
4.2.1.2
2. antero
4.2.2
3. retro
4.2.3
3. balance-associated near fall
4.3
4. balance-associated fall
4.4

Oscillopsia

Peripheral Vestibular Anatomy and Physiology



Physiology of Endolymph



K+ SECRETION

Na+ REABSORPTION

Cl- SECRETION

H+ SECRETION

Ca2+ SECRETION

GLUCOCORTICOID ACTION

βADRENERGIC ACTION

ENDOLYMPH

DARK EPITHELIUM

NEUROEPITHELIUM

Water flow via aquaporins

K+ absorption

Leveque M, Seidermann L, Ulmer E and Chays A, [Vestibular Physiology: anatomic, cellular, immunohistochemical and electrophysiological substrates], EMC, 2015;18(98)-A-10

Tumarkin crisis

Tumarkin A. The otolithic catastrophe. A new syndrome. Br Med J 1936;1:175–7

- Utricle affectation: accesses of rigidity or flexor spasm
  - "Mr X 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. It The thing came like a bolt from the blue but he was immediately able to assure onlookers he was all right and almost immediately got up and carried on."
- Saccular affectation: Fall to the side
  - "Mr Y was sent to me on account of giddiness.[…] On one occasion he was standing in his van receiving packages when suddenly he felt as if the wheel had collapsed under him.

	Canal	Vestibule
Onset	Ingravescent	Instantaneous
Duration	May be minutes up to days	A minute or less
Nausea, pallor, sweating, etc.	Yes	No
Giddiness...	Yes	No
Deafness	Variable in type; may be little or none	Definite reduction of bone conduction

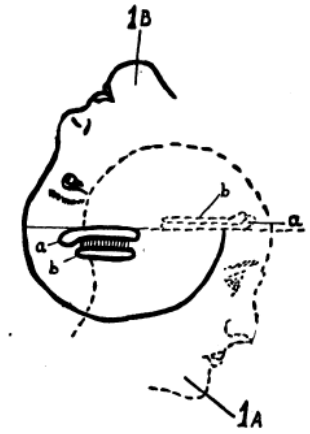


FIG. 1A AND 1B.

FIG. 2.

FIG. 3.

In the upright position (Fig. 1A) *b* is pressing on *a*. In the upside-down position (Fig. 1B) it will drag, and intermediate positions will produce intermediate effects. The right and left lapilli are synergic.

Sudden shift of utricle macula, sudden changes in EL pressure, sudden electrolye changes from rupture of membrane

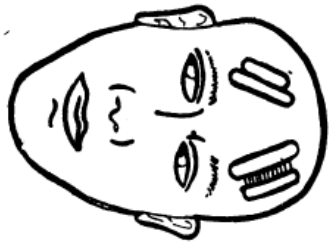
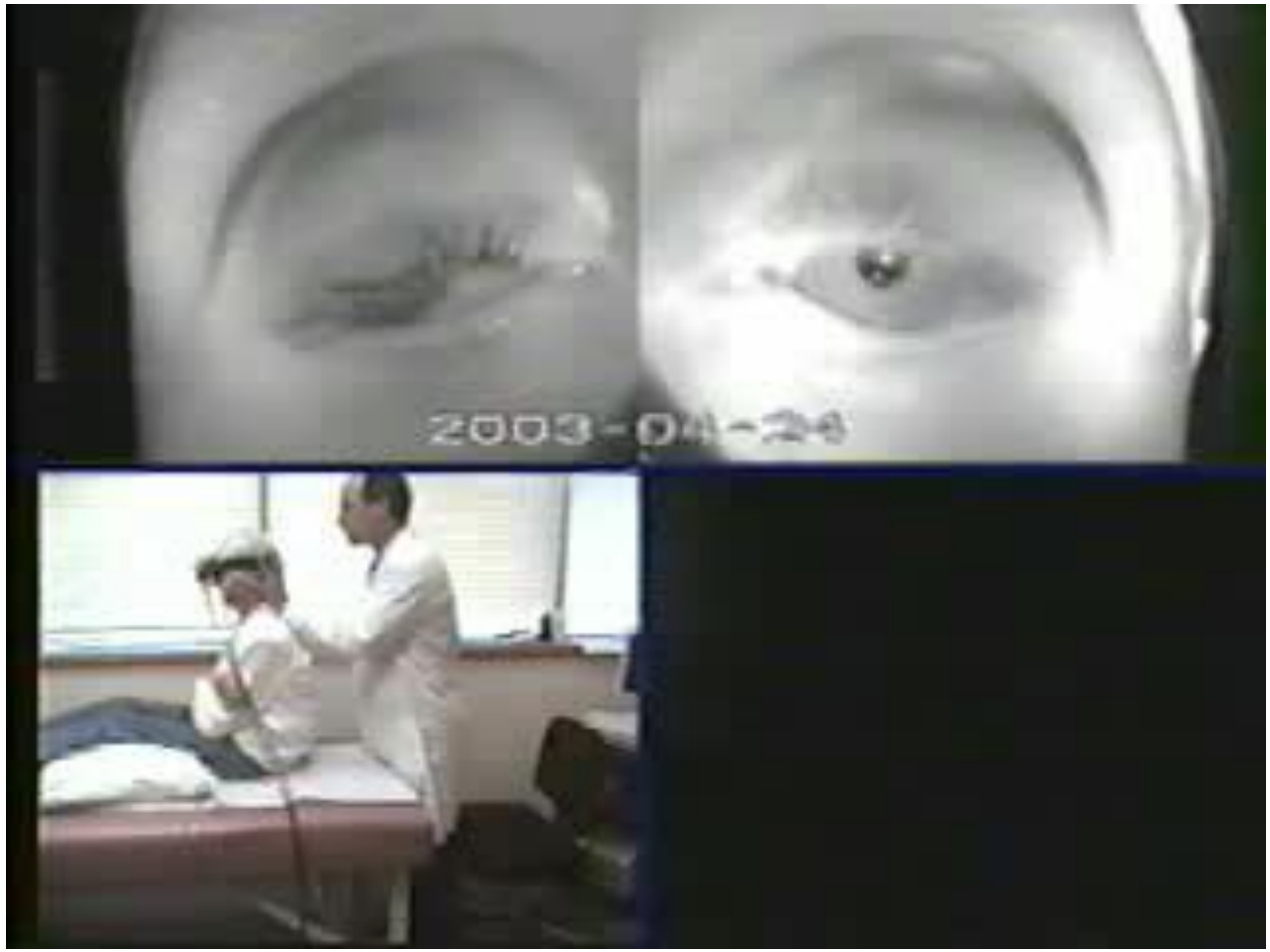


FIG. 4.

FIG. 5.

The sensitive area (sagitta) lies on the side instead of the floor of its retaining sac; furthermore the right and left sagittae are roughly at right angles to each other, so that unlike the lapilli they are opponents and not synergists.

Tumarkin Crisis Post-Epley



Are we gravity bound?

Caloric experiments in microgravity

- Convection currents not present
- Thermal effect

Antigravity vector “built-in” in otolith/graviceptive pathways.

Pierrot-Deseilligny C, Effect of Gravity on Vertical Eye Position, Ann NY Acad Sci, 2009, 1164: 155-165

Parabolic Flight “The Vomit Comet”

Are we gravity bound?

Are we gravity bound?

Pierrot-Deseilligny C, Effect of Gravity on Vertical Eye Position, Ann NY Acad Sci, 2009, 1164: 155-165

Young, L.R. (1988). Gravitational Effects on Brain and Behavior. In: Sensory Systems: II. Readings from the Encyclopedia of Neuroscience . Birkhäuser Boston. https://doi.org/10.1007/978-1-4684-6760-4_12

Caloric experiments in microgravity
- Convection currents not present
- Thermal effect

- VOR is disturbed
  - Reduction in postrotatory nystagmus time decay
    - mechanical return of displaced cupula 5 seconds
    - Velocity storage in vestibulocerebellum
  - Loss of nystagmus dumping when head pitched forward
- Oscillopsia upon reentry

Young, L.R. (1988). Gravitational Effects on Brain and Behavior. In: Sensory Systems: II. Readings from the Encyclopedia of Neuroscience . Birkhäuser Boston. https://doi.org/10.1007/978-1-4684-6760-4_12

- In weightlessness the otolith organs no longer provide meaningful information regarding static orientation of the head. The only act like accelerometers and only detect linear acceleration and not the relationship of the head tilt to the earth's vertical
- When astronauts return home they experience some after-effects from this otolith organ disruption:
  - Difficulty walking around corner
    - Sensation of linear movement or freefall when tilting one's head
  - Vestibular neuronitis can cause similar symptoms
  - Vestibular Migraine can cause similar symptoms
  - Sensation of head being detached from their body

- Space motion sickness and Visual reorientation illusions
  - Conflict between unexpected otolith signals and other senses

Young, L.R. (1988). Gravitational Effects on Brain and Behavior. In: Sensory Systems: II. Readings from the Encyclopedia of Neuroscience . Birkhäuser Boston. https://doi.org/10.1007/978-1-4684-6760-4_12

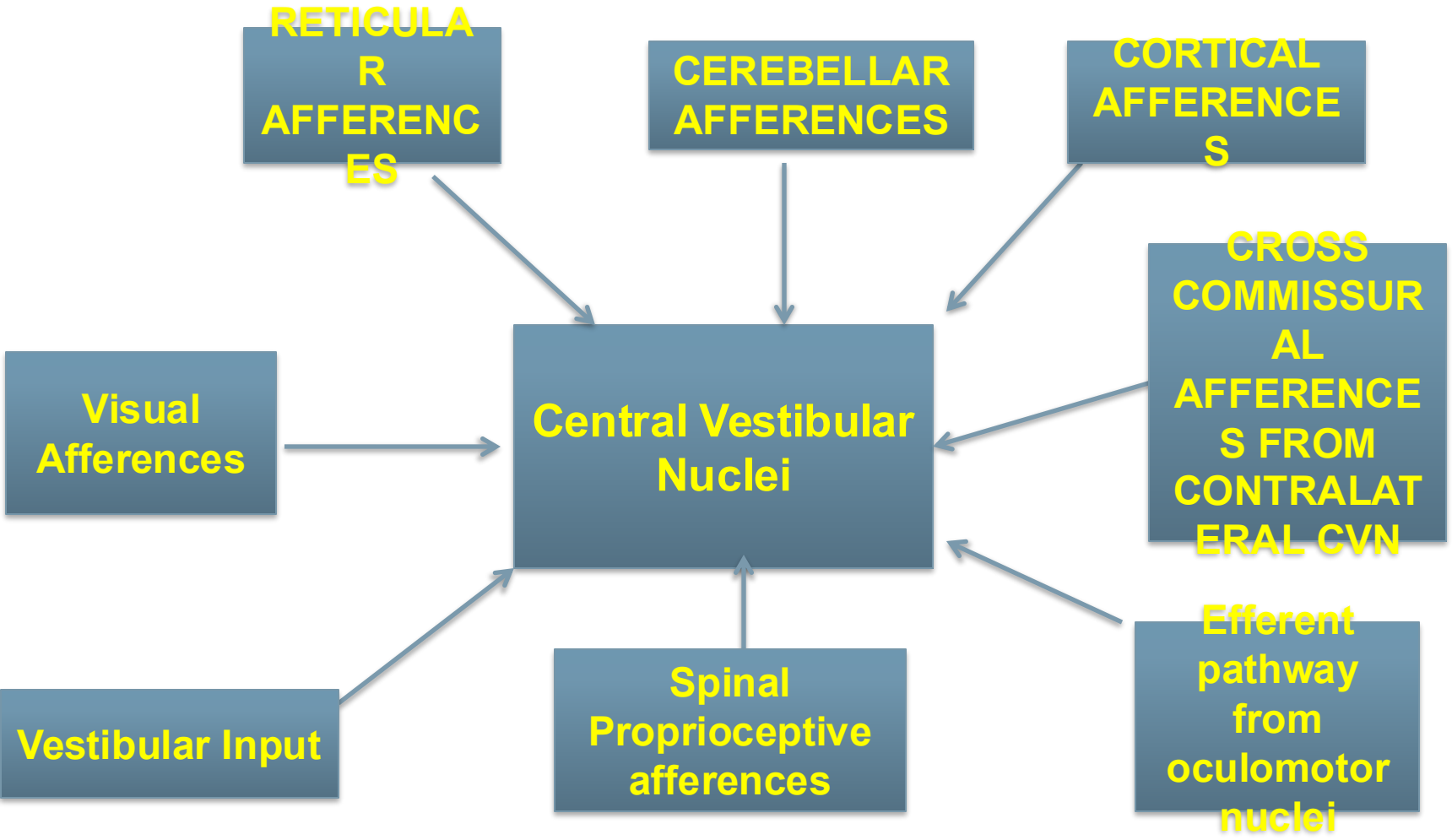
- Proprioception fails in space because of absent static muscle tension
- Otolith spinal reflex is reduced

End organs may be dramatically altered in weightlessness

- Peripheral nerve connections to hair cells increase in density
- Sensory adaptation seems to occur at the periphery, changing the sensitivity and function of otolith organs

All the otolith disruptions cause a sensation of weightlessness and floating that we also observe in our Vestibular Migraine patients

Vestibular System Pleotropic Mediator



Central Vestibular Anatomy and Physiology

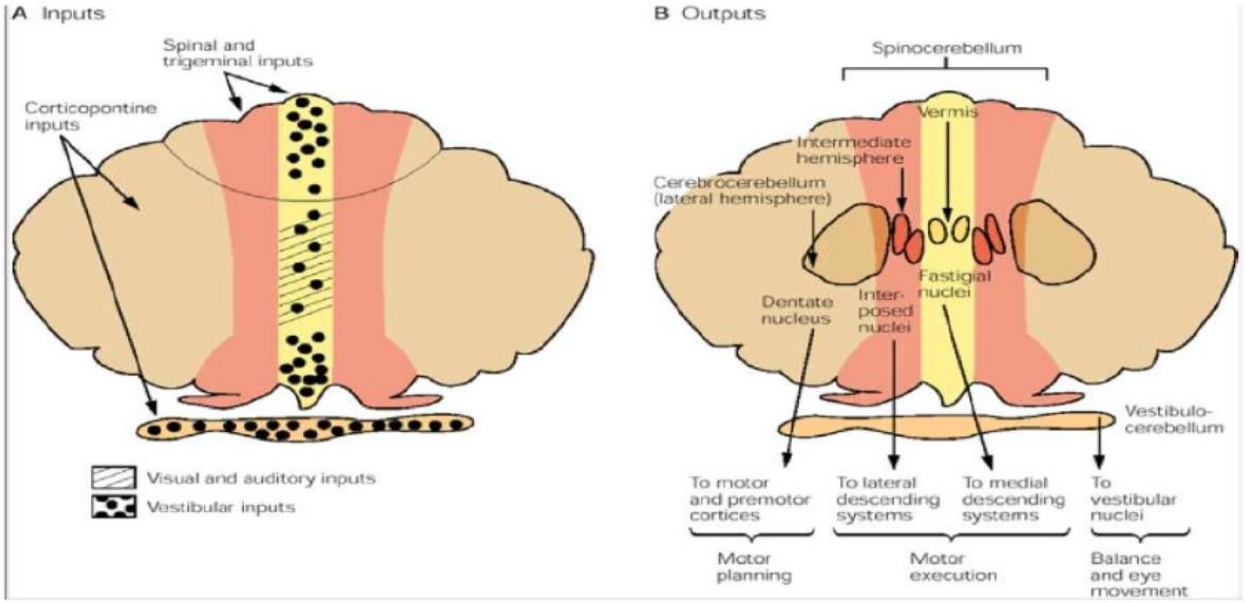
Cerebellum: major role in motor coordination

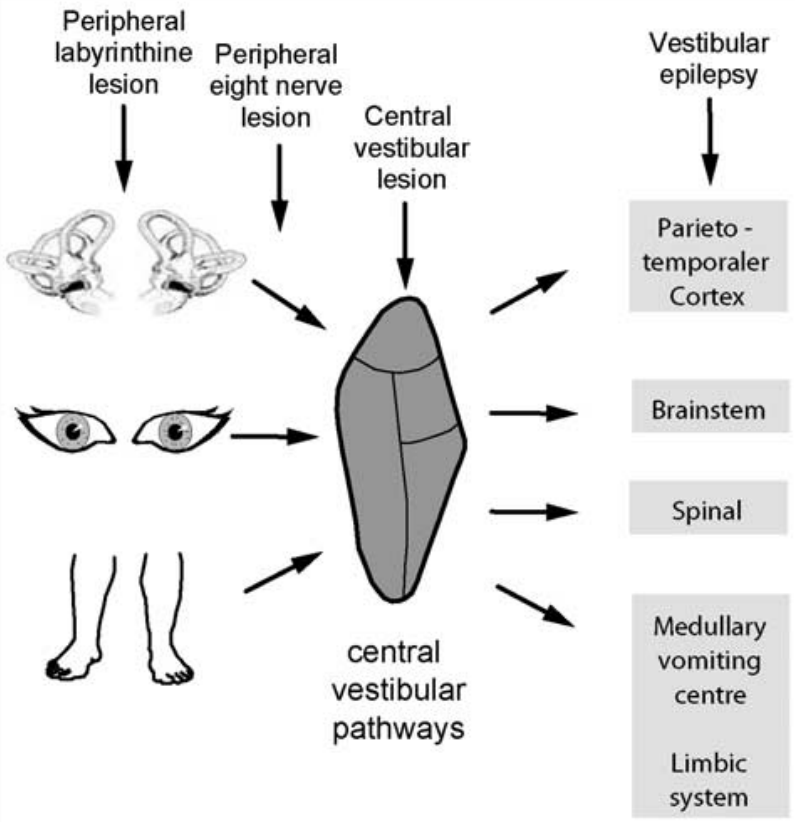
Archeocerebellum or Vestibulocerebellum: flocculus, nodulus, vermis, fastigial nucleus

- Coordinate movements of head and eyes
- Motor learning, adaptation and compensation

Paleocerebellum or Spinocerebellum

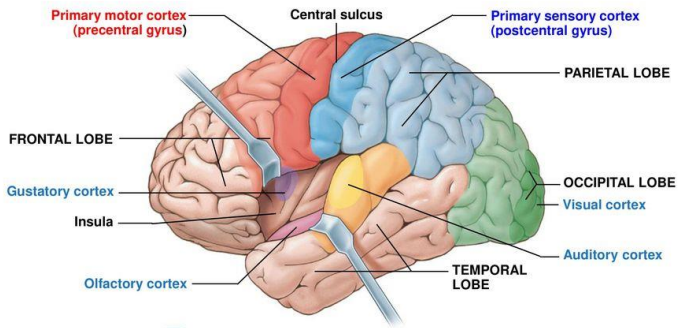
FUNCTIONAL ORGANIZATION OF CEREBELLUM



Physiological vertigo	Pathological Vertigo	Vestibular Function	Vertigo Syndrome
Vestibular		Spatial orientation Motion perception	**Vertigo**
Optokinetic		Vestibulo-Ocular Reflex	**Nystagmus**
		Posture	**Ataxia**
Somatokinetic		Vegetative effects	**Nausea**

Brandt T, Daroff RB. The multisensory physiological and pathological vertigo syndromes. Ann Neurol 1980;7:194–203.

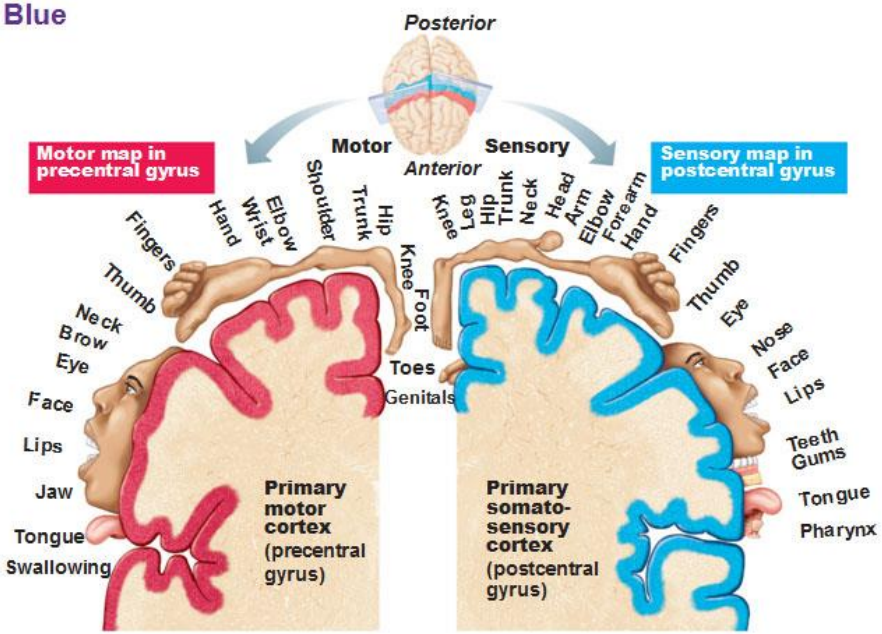
Figure 14-15a Motor and Sensory Regions of the Cerebral Cortex



a Major anatomical landmarks on the surface of the left cerebral hemisphere. The lateral sulcus has been pulled apart to expose the insula.

Homunculus of Primary Somatosensory Cortex in Blue

Note that each hemisphere receives info from the opposite side of the body



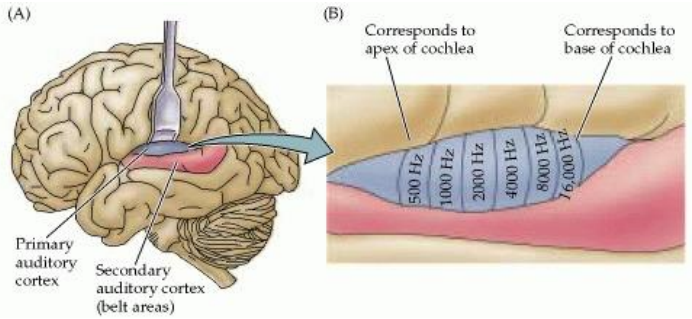
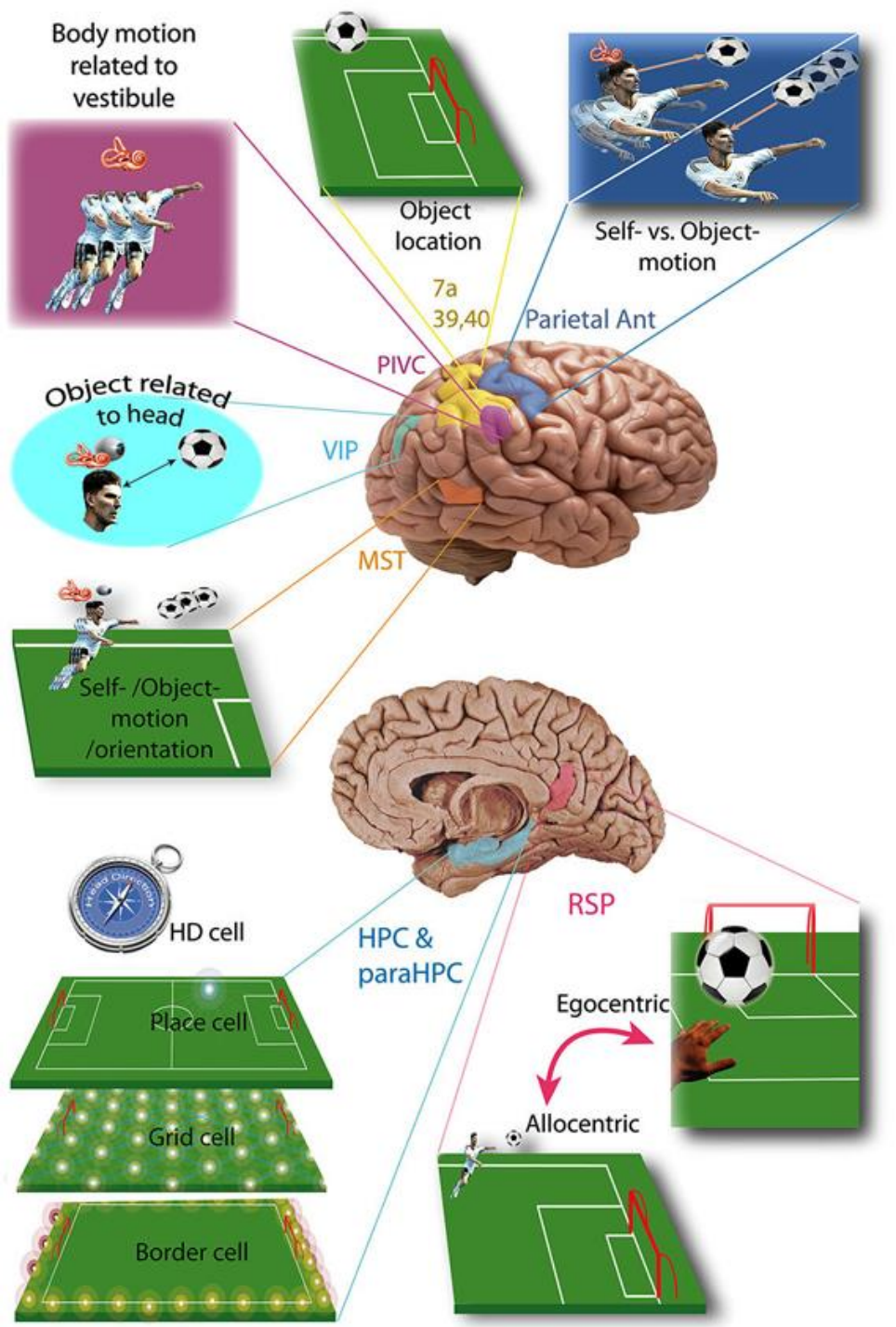


Figure 13.14

The human **auditory cortex**. (A) Diagram showing the brain in left lateral view, including the depths of the lateral sulcus, where part of the **auditory cortex** occupying the superior temporal gyrus normally lies hidden. The primary **auditory cortex** (A1) is shown in blue; the surrounding belt areas of the **auditory cortex** are in red. (B) The primary **auditory cortex** has a tonotopic organization, as shown in this blowup diagram of a segment of A1.



Area 7 – Visuo-Motor Coordination
Point of convergence between vision and proprioception to determine where moving objects are in relation to the body

Area 39 – Angular gyrus, considered by some to be part of Wernicke's area
It is also involved in a number of processes related to language, number processing and spatial cognition, memory retrieval, attention, and theory of mind.
Area 40 – Supramarginal gyrus, involved with language processing. Lesions in it can result in receptive aphasia

Vestibular cortices and spatial cognition. Vestibular cortices involved in spatial cognition, illustrated in a soccer player: HD cell, head direction cell; HPC, hippocampus; MST, medial superior temporal area; ParaHPC, parahippocampal cortex; Parietal Ant, parietal anterior cortex; PIVC, parieto-insular vestibular cortex; RSP, retrosplenial cortex; VIP, ventral intraparietal area; 7a, 39, 40 Brodmann area.

Central Vestibular Anatomy and Physiology

Vestibulothalamic and vestibulocortical pathways:

- ***sensation and perception of head movement***

Vestibulospinal pathways:

- ***motor commands to the muscles of the neck, upper torso and lower limbs to maintain balance***
- ***Cortical efferent and cerebellar efferents modulate vestibulospinal output***
- ***Related to drop attacks***

Vestibulocular pathways

- ***responsible for the VOR***
- Responsible for gaze, saccades, smooth pursuit and optokinetic nystagmus

Two questions come to mind

Q1 Can someone recover from damage to the end-organ?

Vestibular Neuronitis: Unilateral Vestibular Deafferentation

34 year old patient with no prior medical problems

6/6/2017 felt spinning when he would lay on his left side. This gets better when he lays flat.

6/8/2017 Acute right stye

6/10/2017 vertigo constant even when looking straight ahead

Alexander's Law: 1912

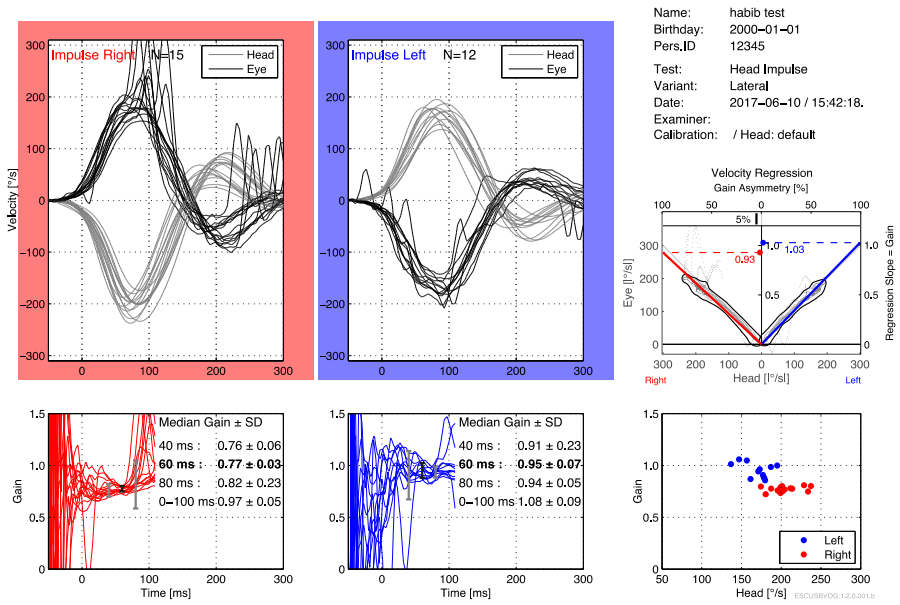
In Unilateral vestibular lesion

- Spontaneous Nystagmus with fast phase toward healthy ear

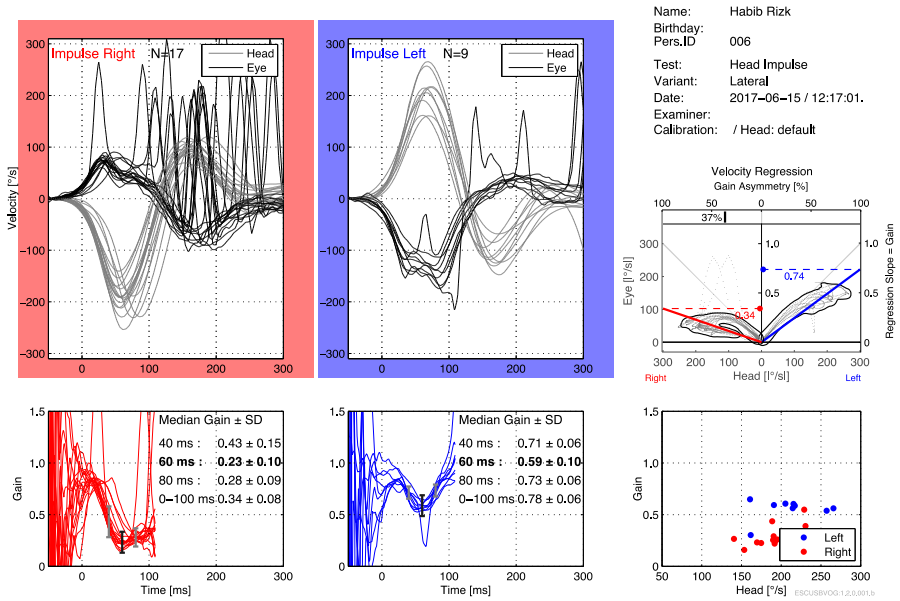
Slow phase is greatest when gaze is directed toward healthy ear or nystagmus fast phase, attenuates at central gaze and weakest when gaze is toward ipsilesioned ear

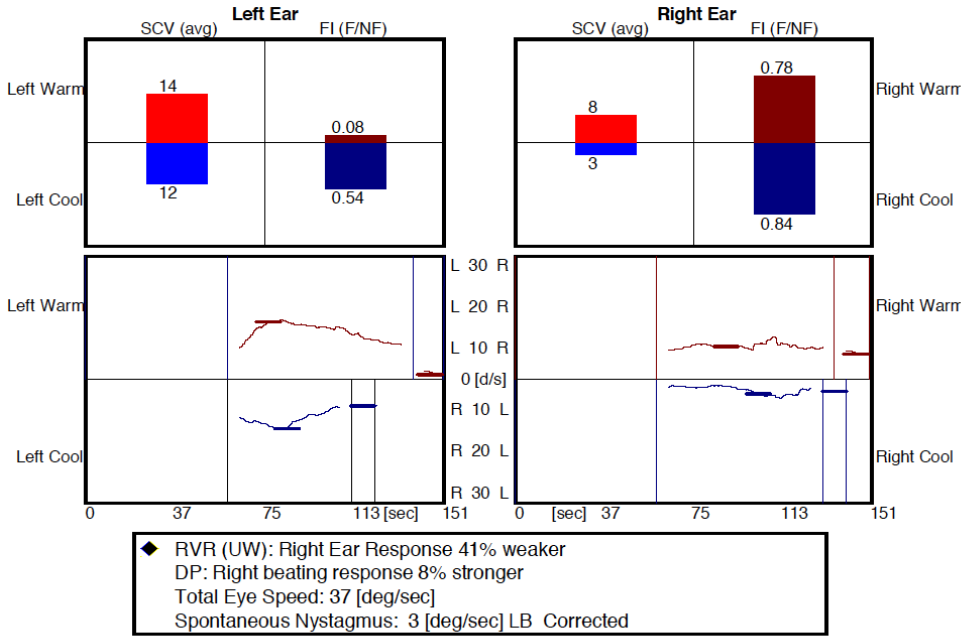
It actually is a compensation strategy from CNS in the context of vestibular imbalance

VideoHIT



Vertigo starts subsiding and patient starts noting falling to the right side when he turns his head to the right or his ear to touch his shoulder

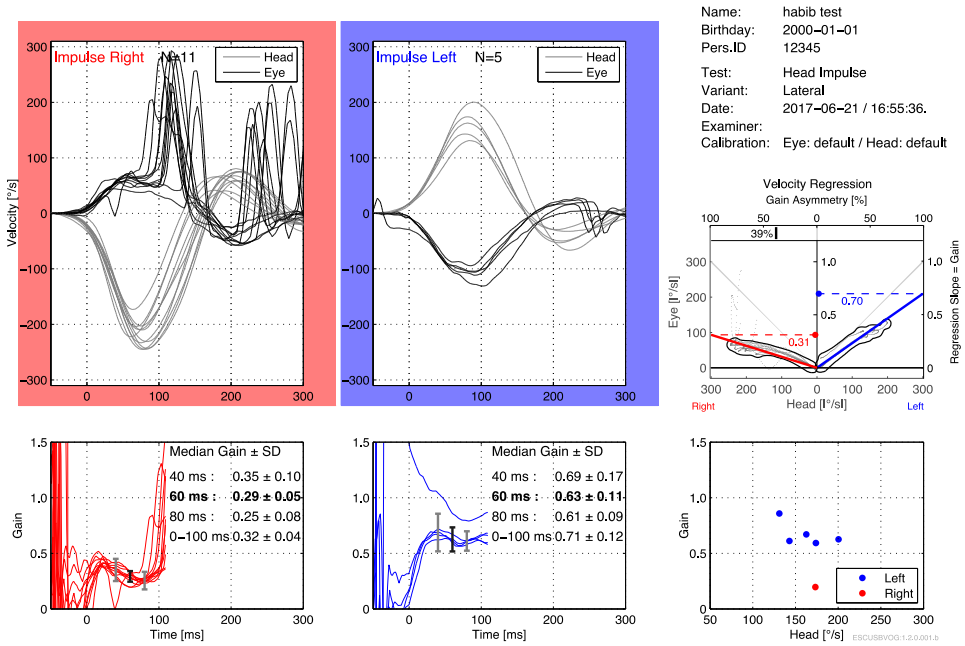




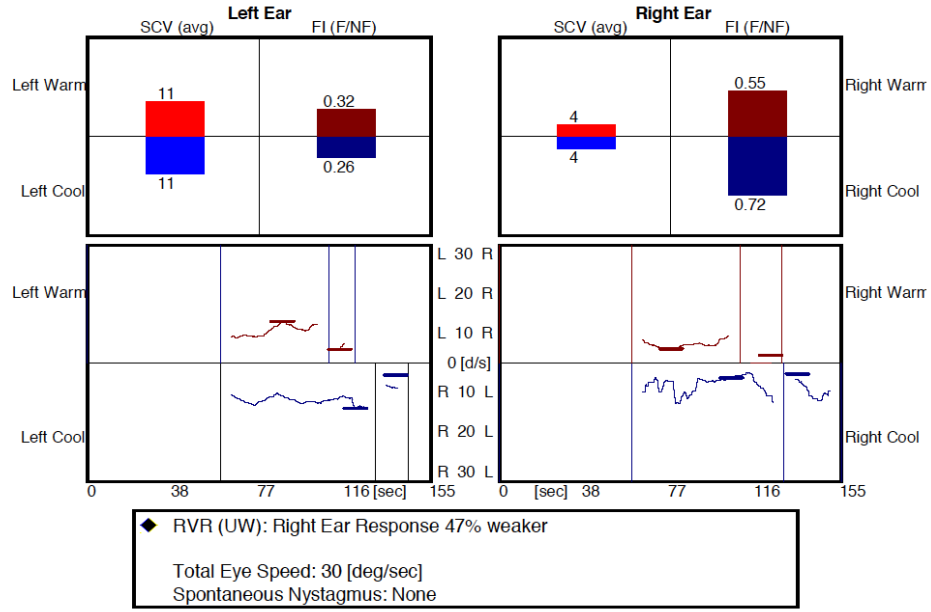
◆ RVR (UW): Right Ear Response 41% weaker
DP: Right beating response 8% stronger
Total Eye Speed: 37 [deg/sec]
Spontaneous Nystagmus: 3 [deg/sec] LB Corrected

Patient's balance is off for the next ten days

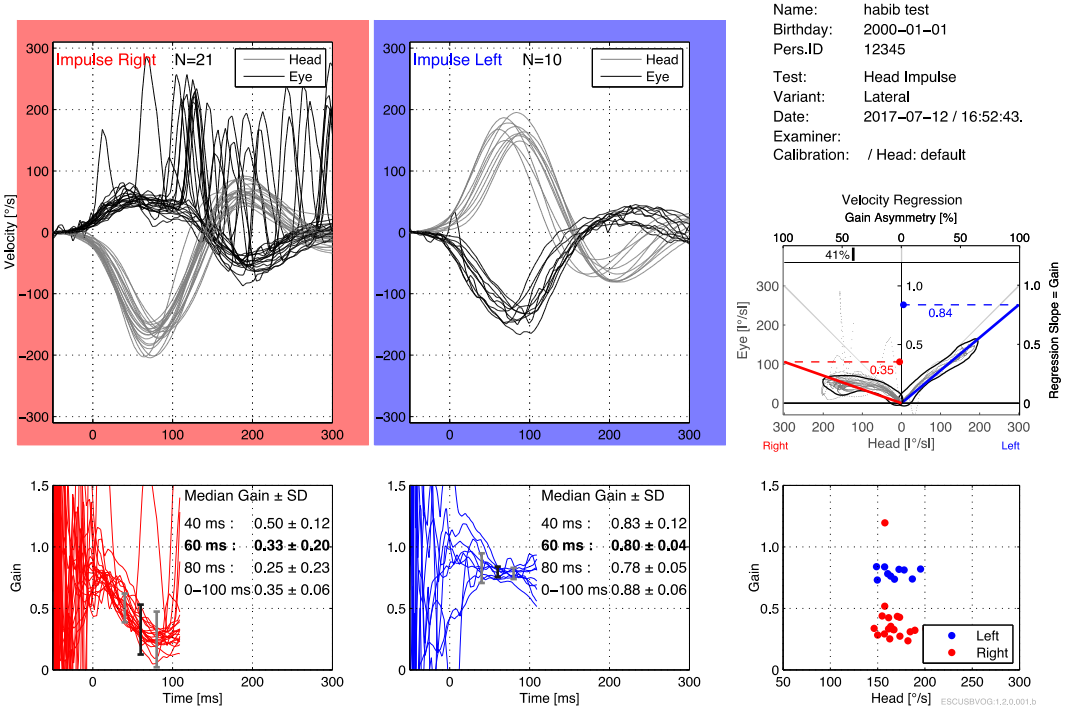
No more spinning vertigo



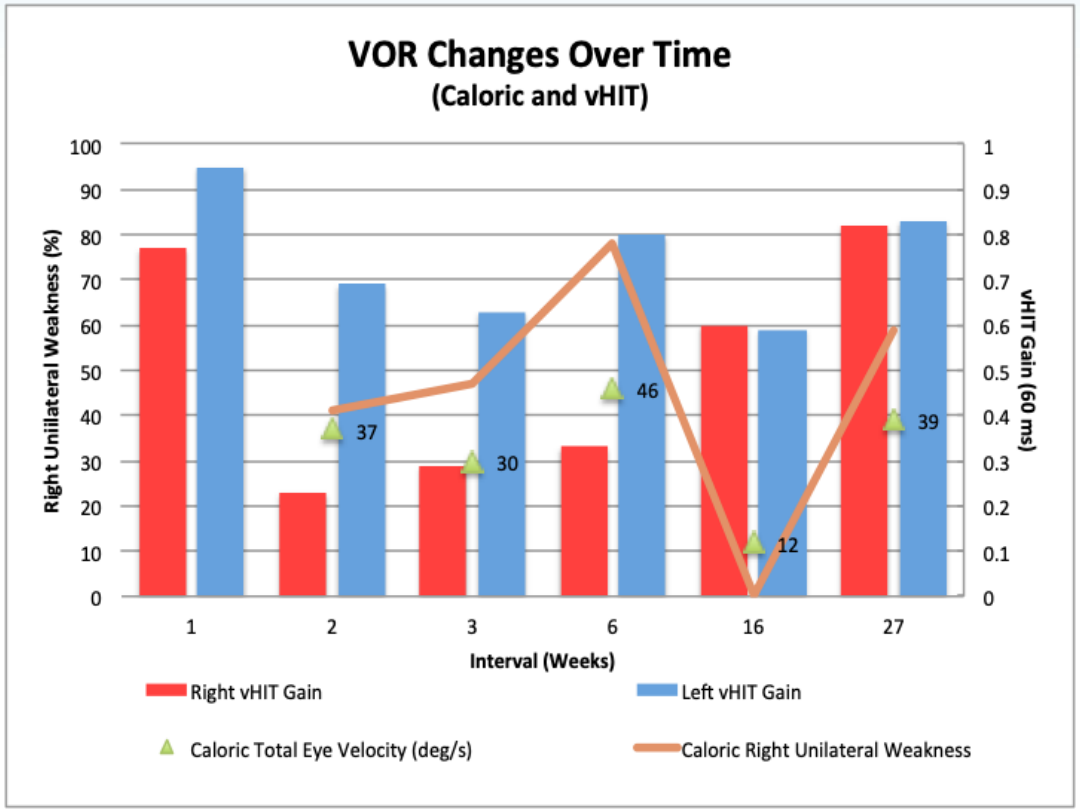
Caloric Summary



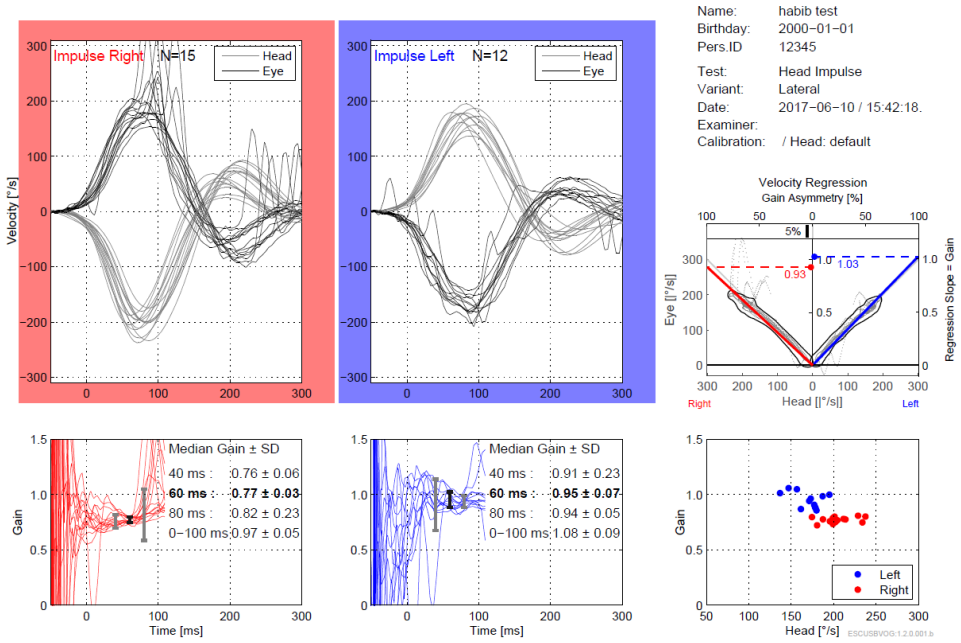
36 days s/p onset



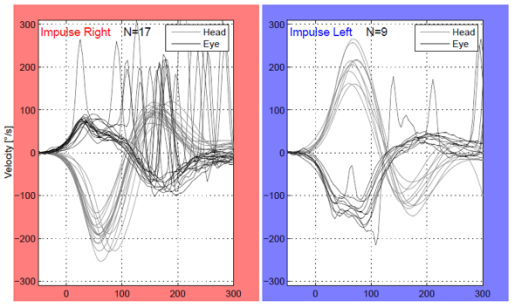
Vestibular Test Battery		Evaluation Timepoints					
		Week 1	Week 2	Week 3	Week 6	Week16	Month 8
Calorics			41% RUW	47% RUW	78% RUW	0% BW	59% RUW
Spontaneous Nystagmus			3 d/s LBN	None	None	None	None
SHA gain			Normal	Normal			Normal
SHA symmetry			CW weaker	Normal			Normal
SHA phase			Normal	Normal			Normal
vHIT gain	Right	0.77	0.23	0.29	0.33	0.60	0.82
	Left	0.95	0.59	0.63	0.8	0.59	0.83
vHIT overt saccades	Right	Present	Present	Present	Present	Present	Present
	Left	Absent	Absent	Absent	Absent	Absent	Present
vHIT covert saccades	Right	Present	Present	Present	Present	Absent	Absent
	Left	Absent	Absent	Absent	Absent	Absent	Absent

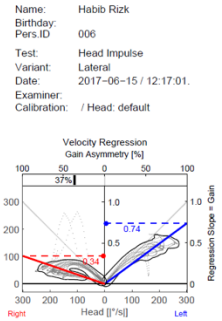

VOR Changes Over Time
(Caloric and vHIT)
Right Unilateral Weakness (%)
vHIT Gain (60 ms)
100
90
80
70
60
50
40
30
20
10
0
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
37
30
46
12
39
1
2
3
6
16
27
Interval (Weeks)
Right vHIT Gain
Left vHIT Gain
Caloric Total Eye Velocity (deg/s)
Caloric Right Unilateral Weakness

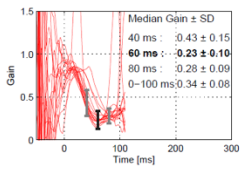
Acute Peripheral Vestibular Lesion
Day 4

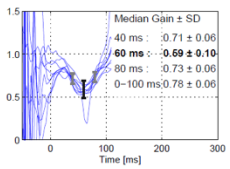


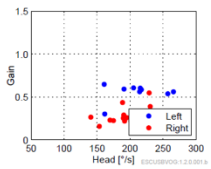
6/15/2017
Day 9

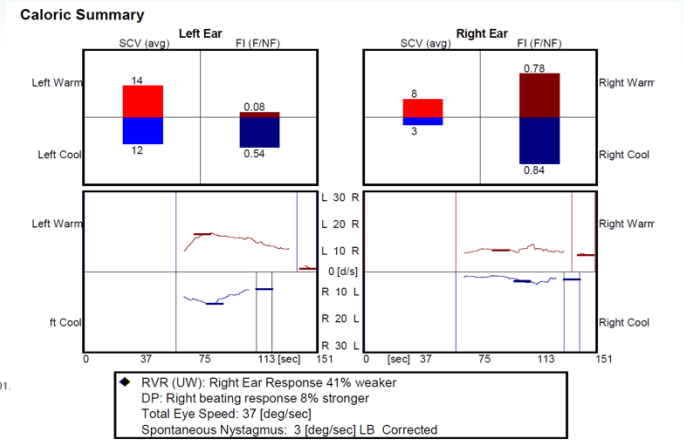


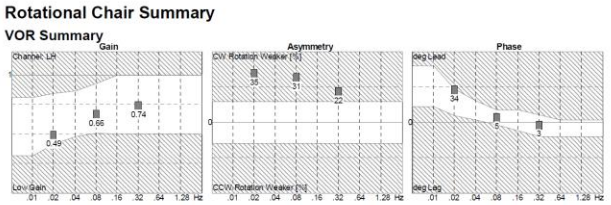




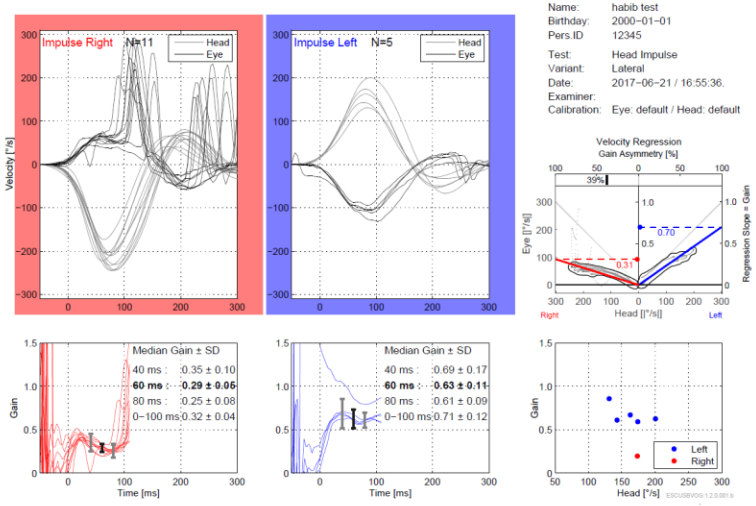


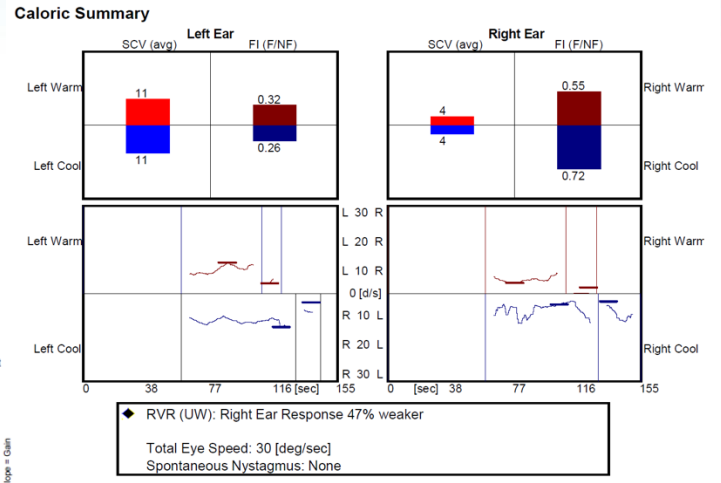


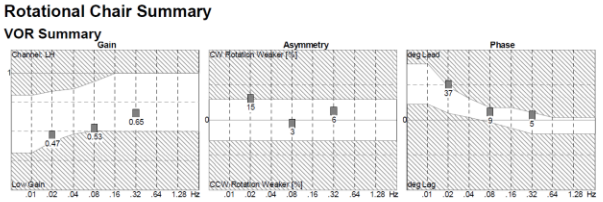




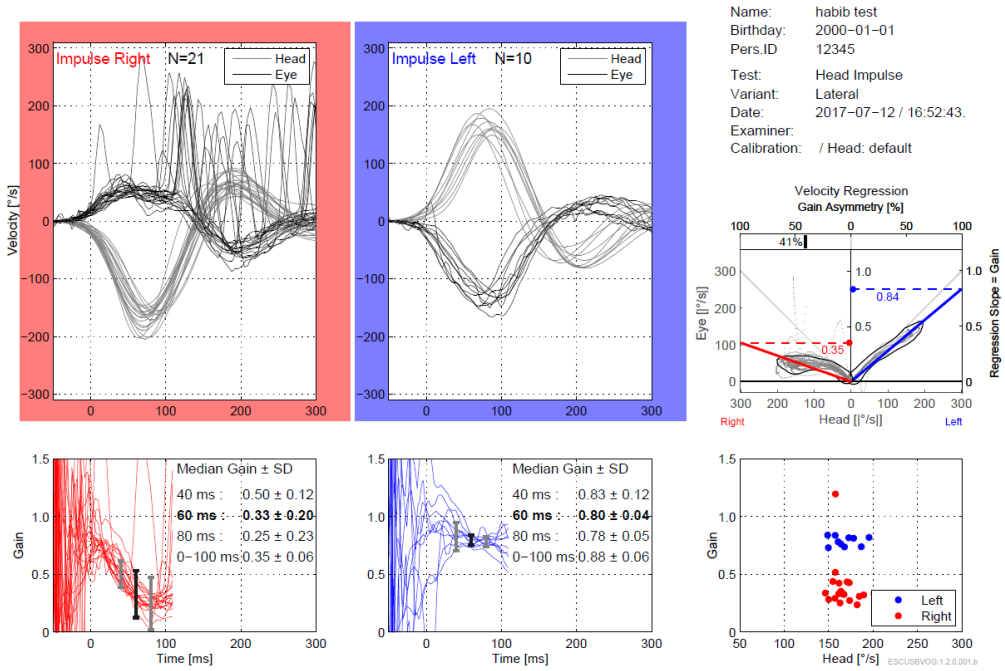
6/23/2017
Day 17



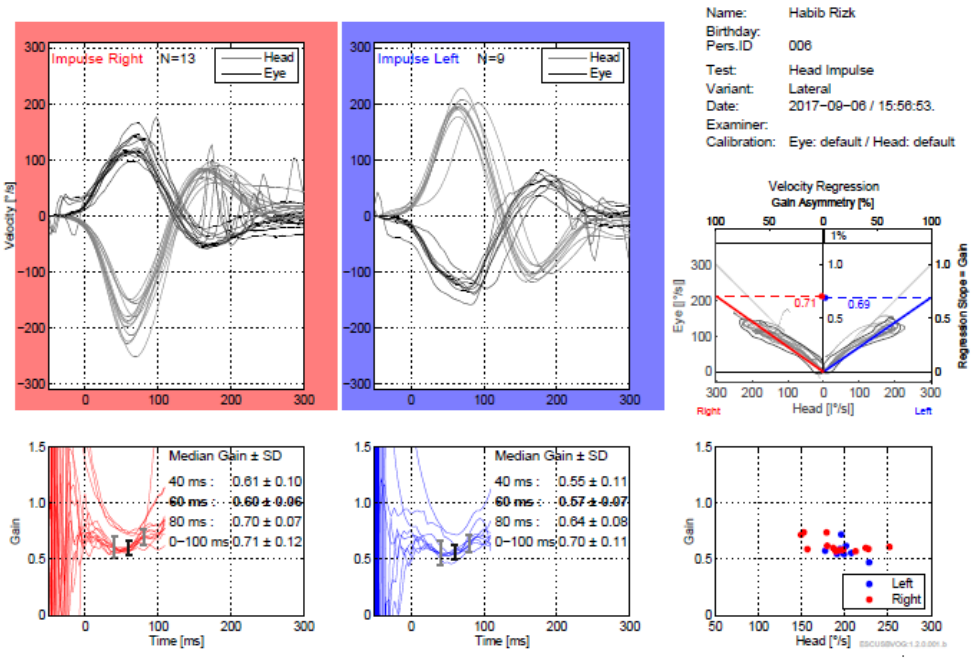




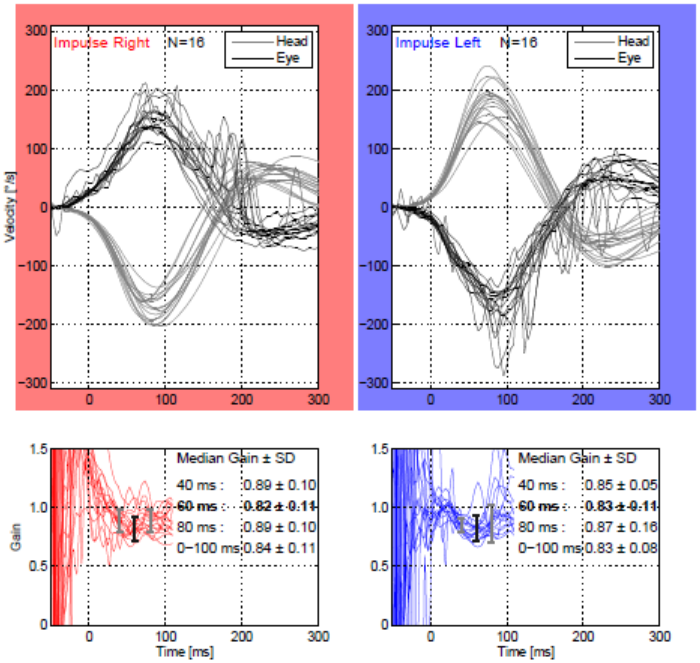
7/12/2017
Week 5

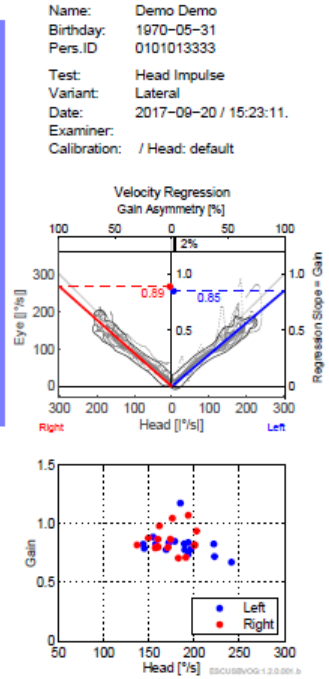


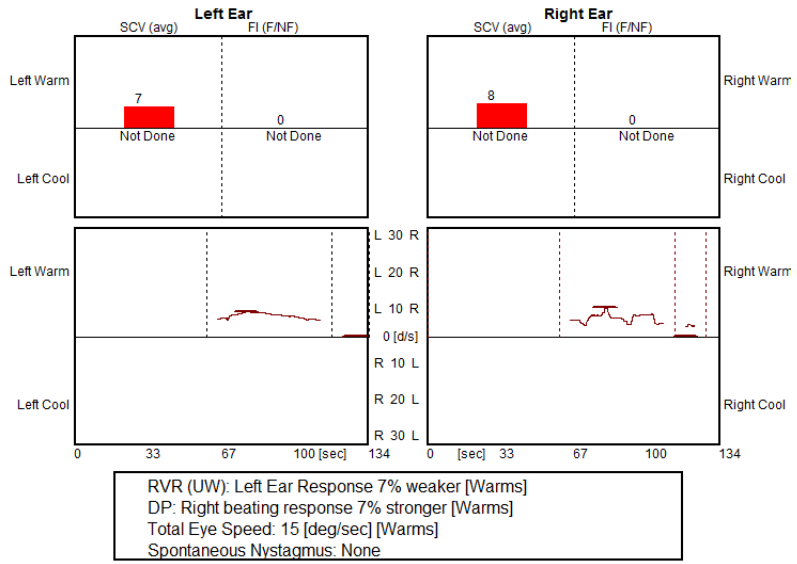
9/6/2017
Day 90



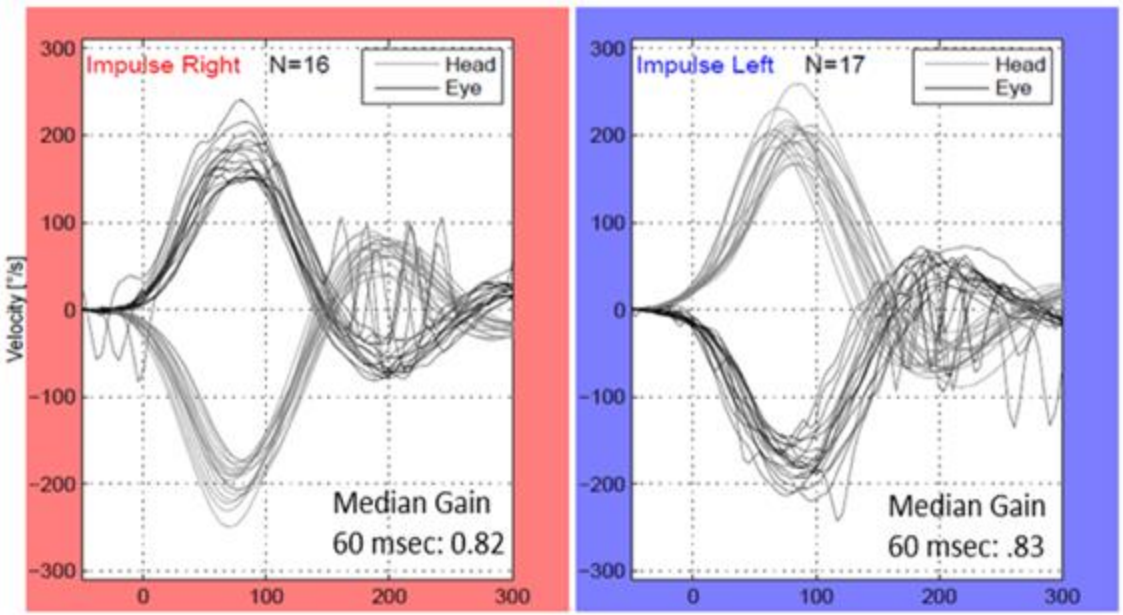
9/20/2019
3.5 Month







Month 8


Impulse Right
N=16
Head
Eye
Velocity [°/s]
300
200
100
0
-100
-200
-300
0
100
200
300
Median Gain
60 msec: 0.82
Impulse Left
N=17
Head
Eye
Median Gain
60 msec: .83

Bilateral Vestibular Deafferentation

Sequential:

- Bechterew's Phenomenon

Concomitant

Vestibular Compensation

N Engl J Med 1952; 246:458-460 March 20, 1952

" When I recall how completely disabled I was by the initial impact of loss of vestibular function, I am amazed that I am so little troubled at present"

MEDICAL INTELLIGENCE

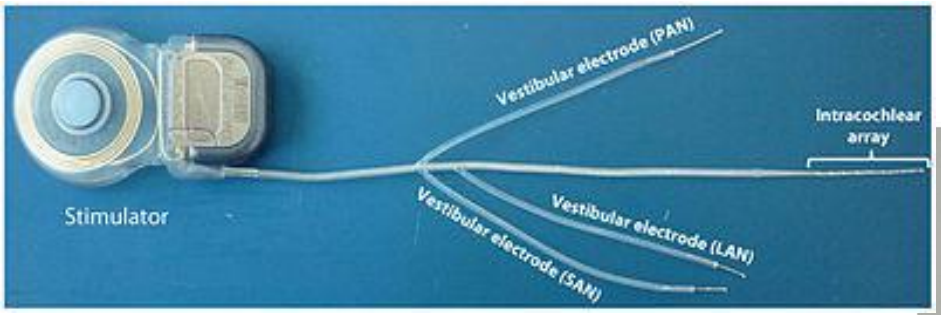
LIVING WITHOUT A BALANCING MECHANISM*

J. C.

BOSTON

" Is there any man-made machine designed like the human apparatus- with so many alternate systems to accomplish its end?"

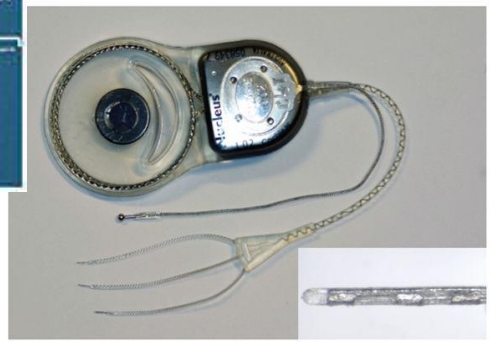
Vestibular Implants?



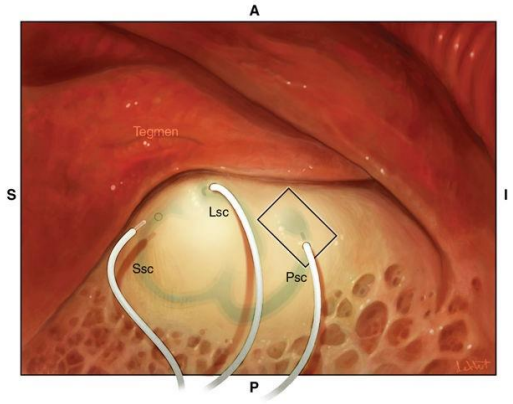
ORL 2015;77:227–240

DOI: 10.1159/000433554

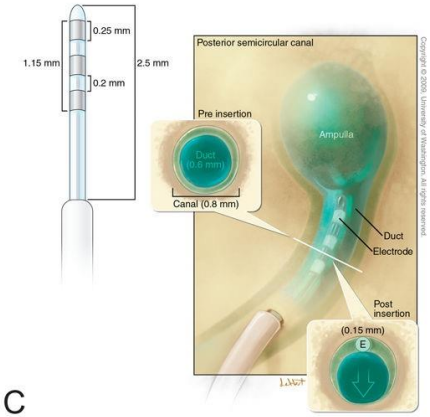
Guinand et al.: Vestibular Implants: 8 Years of Experience with Electrical Stimulation of the Vestibular Nerve in 11 Patients with Bilateral Vestibular Loss



A



B



C

Q2 Can the peripheral vestibular end-organ affect higher cortical functions?

PART II The vestibular system-cognition link

Aging of the Vestibular System Histology

Marked age-related degeneration in nearly every type of vestibular-related cell and neuron bundle

- Starting after the age of 65 years
- Saccule seems to be the most affected

Clinical Implications: Age-related declines in the ability to correctly detect head position and motion in space, to elicit vestibular spinal reflexes or to solve sensory conflicts

Tang PF, Woollacott M, Balance control in older adults. In: Bronstein AM,Brandt TW, Nitt J, eds. Clinical disorders of balance, posture and gait.2nd ed. London: Arnold, 2004:385-403

Aging of the Vestibular System
Clinical Correlation

Early studies did not show direct correlation between histology and abnormalities on paraclinical testing

More recent evidence comes with VEMPs and videoHIT showing high prevalence of abnormalities with age

1. *Agrawal Y, Zuniga MG, Davalos-Bichara M et al, Decline in semicircular canal and otolith function with age, Otol Neurotol, 2012; 33: 832-839*
2. *Agrawal Y, Schubert MC, Migliaccio AA et al, Evaluationof quantitative head impulse testing using search coils versus video-oculography in older individuals, Otol Neurotol, 2014; 35:283-288*

Central compensation occurs until a “tipping point” is reached

Aging of the Vestibular System Epidemiology

Incidence of Dizziness increases from 22% (65-69) to over 40% for adults over 80-84 (18 M people by 2050)

Cutson TM, Falls in the elderly, Ann Fam Physician, 1994, 49:149-156

65% of adults over 60 experience dizziness and loss of balance on a daily basis

Hobeika CP, Equilibrium and balance in the elderly, Ear Nose Throat J, 1999, 78:558-562

Approximately 20% of imbalance and 40-50% of dizziness is due to a vestibular problem

University of Iowa Health Care. Comprehensive Management of vestibular disorders, Currents, 2002,3 (2)

Aging of the Vestibular System Epidemiology

TABLE 1.1
Most Common Causes of Dizziness in Primary Care Practice[1]

Category	Percentage of Patients (%)	Examples
Peripheral vestibular disease	20–50	Benign paroxysmal positional vertigo, labyrinthitis, vestibular neuritis
Cardiovascular disease	10–30	Arrhythmia, congestive heart failure, vasovagal conditions (e.g., carotid sinus hypersensitivity)
Systemic infection	10–20	Systemic viral and bacterial infection
Psychiatric conditions	5–15	Depression, anxiety, hyperventilation
Metabolic disturbances	5–10	Hypoglycemia, hyperglycemia, electrolyte disturbances, thyrotoxicosis, anemia
Medications	5–10	Antihypertensives, psychotropic medications

Aging of the Vestibular System Epidemiology

Lifetime prevalence of vestibular vertigo 7.8%

One year prevalence of vertigo 4.9%

- 7.2% in 60-69 year olds
- 8.8% over the age of 80

Incidence of vertigo 1.5%

Neuhauser NK, von Brevern M, Radtke A et al. Epidemiology of vestibular vertigo: a neurotologic survey of the general population. Neurology, 2005; 65:898-904

Aging of the Vestibular System Epidemiology

35% of US adults over 40 have evidence of balance dysfunction on postural exam

85% of individuals over 80 have evidence of balance dysfunction

Agrawal Y, Carey JP, Della Santina CC, Schubert MC, Minor LB, Disorders of balance and vestibular function in US adults: data from the national health and nutrition examination survey, 2001-2004, Arch Intern Med, 2009;169:938-944

Dizziness is a major cause of absenteeism, partial or full disability and an increase in healthcare utilization

Diagnostic criteria for presbyvestibulopathy (PVP)

Each of the criteria A through D have to be fulfilled

A. Chronic vestibular syndrome (at least 3 months duration) with at least 2 of the following symptoms:[1]

1. Postural imbalance or unsteadiness
2. Gait disturbance
3. Chronic dizziness
4. Recurrent falls

B. Mild[2] bilateral peripheral vestibular hypofunction documented by at least 1 of the following:

1. VOR gain measured by video-HIT between 0.6 and 0.8[3] bilaterally
2. VOR gain between 0.1 and 0.3 upon sinusoidal stimulation on a rotatory chair (0.1 Hz, Vmax = 50-60°/sec)[4]
3. Reduced caloric response (sum of bithermal maximum peak SPV on each side between 6 and 25°/sec)[5]

C. Age ≥ 60 years[6]

D. Not better accounted for by another disease or disorder[7]

Presbyvestibulopathy

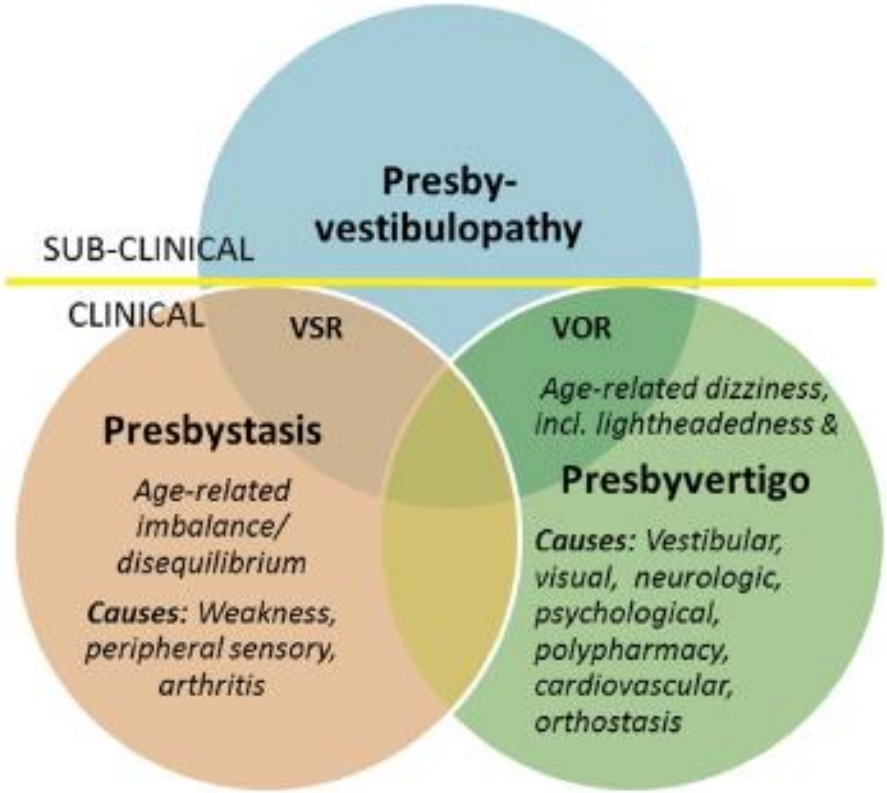
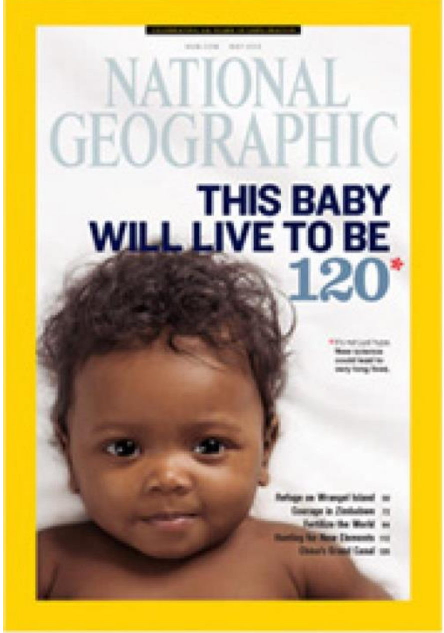


FIG. 1.1 Typology of age-related dizziness, imbalance, and vestibular loss. *VOR*, vestibulo-ocular reflex; *VSR*, vestibulospinal reflex.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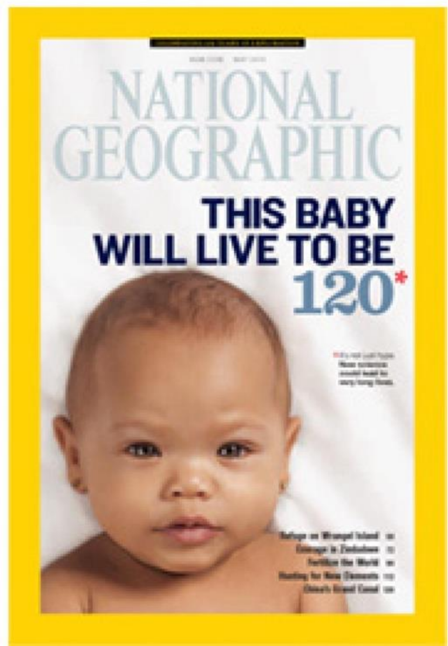
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Vestibular System and Cognition

Older studies:

- Memory impairment and Perilymphatic fistulae
- Concentration difficulties and gentamicin ototoxicity

Bilateral vestibular nerve section (NF2) and reduction in spatial memory and spatial navigation as well as reduced hippocampal volumes

Brandt T, Schautzer F, Hamilton DA, Brüning R, Markowitsch HJ, Kalla R, Strupp M, Vestibular loss causes hippocampal atrophy and impaired spatial memory in humans, Brain, 2005, 128:2732-2741

Van der Zaag HJ, van Leeuwen RB, Dizziness causes absence from work, Acta Neurol Belg, 2015, 115: 345-349

- 400 patients
- 70% absenteism
- 12% completely disabled
- 51% worked less because of the dizziness

Vestibular System and Cognition

Box 1. Summary of 3 US population-based studies reporting a significant association between reduced vestibular function and poorer cognitive performance.

Study population	Year	N	Finding
Baltimore Longitudinal Study of Aging (BLSA)	2013-2014	183	Reduced saccular function associated with significantly poorer scores on neurocognitive tests of visuospatial ability in cross-sectional analyses; no significant relationship between saccular function and tests of language, executive function, and verbal memory
National Health and Nutrition Examination Survey (NHANES)	1999-2002	1303	Vestibular impairment (based on modified Romberg test) associated with significantly poorer Digit Symbol Subsitution Score in cross-sectional analyses
National Health Interview Survey (NHIS)	2008	20,950	Vestibular vertigo (based on questionnaire responses) significantly associated with 4-fold increased odds of "difficulty remembering and confusion" in cross-sectional analyses

Bigelow, R. T., Semenov, Y. R., Du Lac, S., Hoffman, H. J., & Agrawal, Y. (2015). Vestibular vertigo and comorbid cognitive and psychiatric impairment: The 2008 national health interview survey. *Journal of Neurology, Neurosurgery & Psychiatry, 87*(4), 310–319.

Bigelow, R. T., Semenov, Y. R., Trevino, C., Ferrucci, L., Resnick, S. M., Simonsick, E. M., … Agrawal, Y. (2015). Association between visuospatial ability and vestibular function in the baltimore longitudinal study of aging. *Journal of the American Geriatrics Society, 63*(9), 1837–1844.

Semenov, Y. R., Bigelow, R. T., Xue, Q., Lac, S. D., & Agrawal, Y. (2015). Association between vestibular and cognitive function in US adults: Data from the national health and nutrition examination survey. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences, 71*(2), 243–250.

Vestibular System and Cognition

Hippocampal atrophy in Meniere's disease

1. *Kremmyda O, Hüffner K, Flanagin VL, Hamilton DA, Linn J, Strupp M, Brandt T, Beyond dizziness: Virtual navigation, spatial anxiety and hippocampal volume in bilateral vestibulopathy, Frontiers in Human Neuroscience, 10, 139*
2. *Popp P, Wulff M, Finke K, Rühl M, Brandt T and Dieterich M, Cognitive deficits in patients with chronic vestibular failure, J Neurol, 2017, 264:554-563*

20% of patients presenting with vertigo have reproducible and significant cognitive error in arithmetic abilities, short term memory and performance in specific visual environments

1. *Risey J and Briner W, Dyscalculia in Patients with Vertigo, J Vest Res, 1990, 1:31-37*
2. *Hanes DA and McCollum G, Cognitive-vestibular interactions: a review of patient difficulties and possible mechanisms, J Vest Res, 2006, 16:75-91*

Vestibular System and Cognition

Alzheimer's disease patients: twice as likely to have vestibular impairment than healthy controls

AD patients with vestibular impairment have disproportionate reduction in spatial cognition compared to AD without vestibular loss

Agrawal Y, Smith PF and Rosenberg PB, Vestibular impairment, cognitive decline and Alzheimer's disease: balancing the evidence, Aging and Mental Health. Doi:10.1080/13607863.2019.1566813

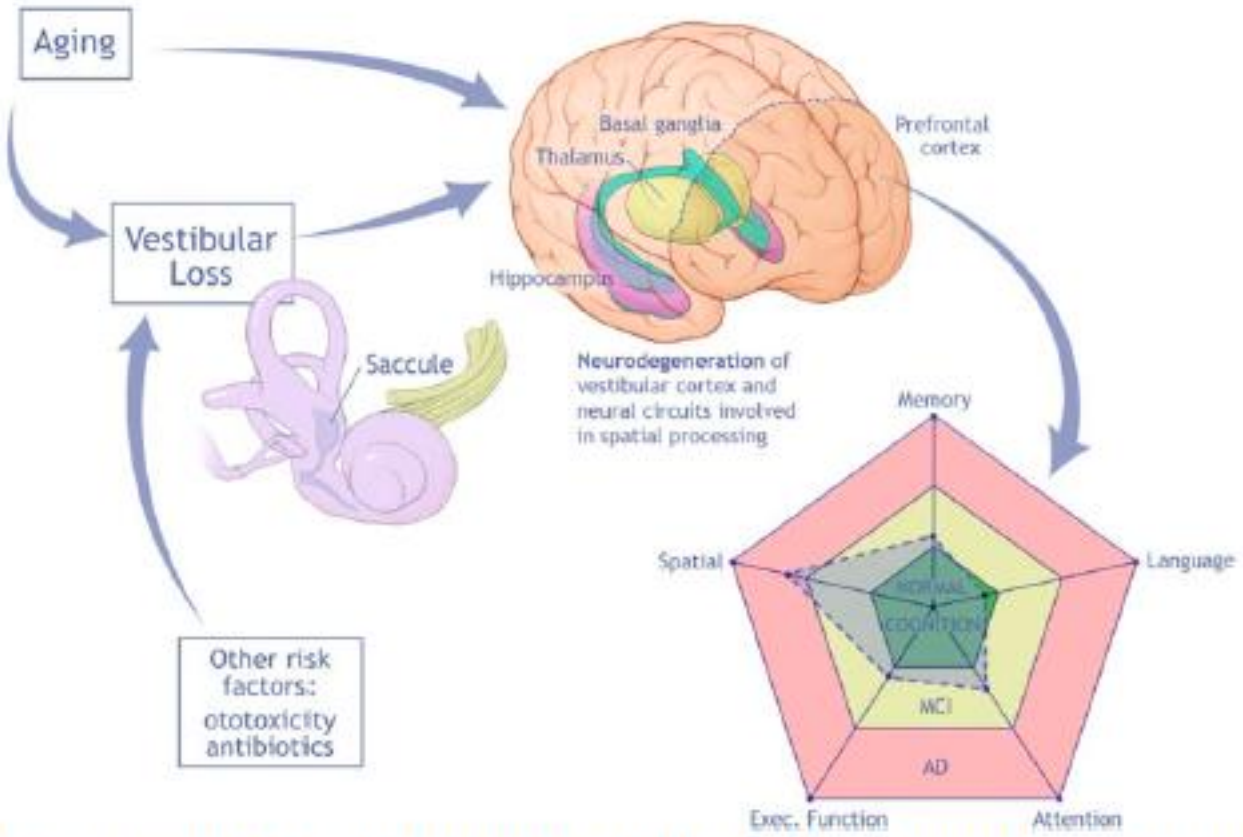
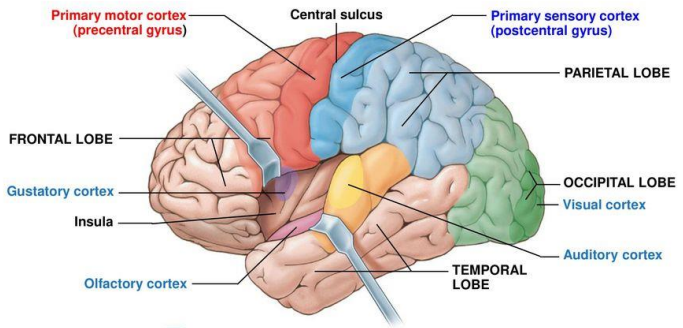


Figure 1. Conceptual model of impact of aging on vestibular function (notably saccular function), which contributes to neurodegeneration of neural circuits involved in vestibular processing and deterioration specifically in spatial cognitive ability.

Peripheral vestibular system provides major inputs to cholinergic neurons in medial temporal region, specifically degraded in AD

- **Vestibular impairment may participate in pathogenesis of AD**
- **Vestibular Impairment may cause a spatial phenotype of AD where there is higher risk of spatial disorientation, wandering and increase fall risk**

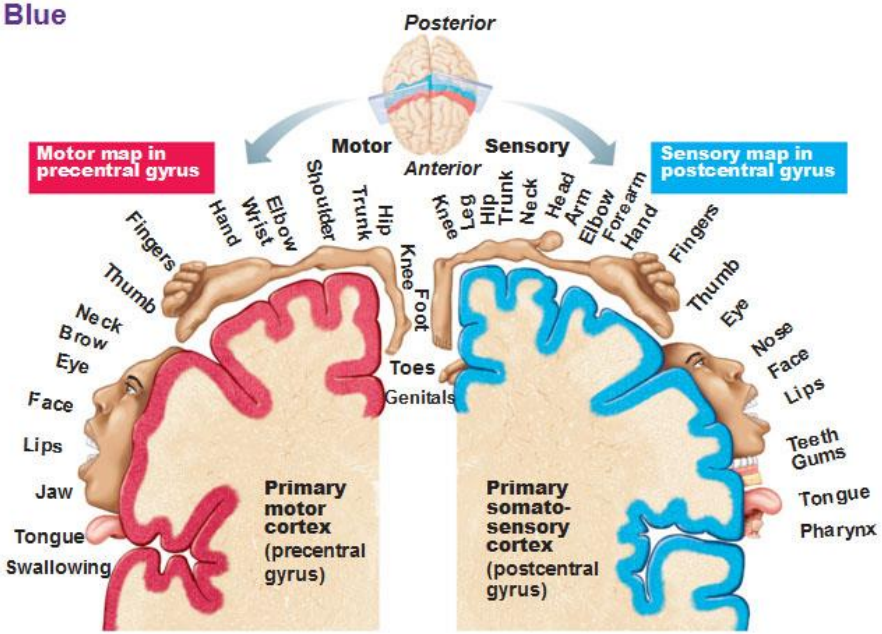
Figure 14-15a Motor and Sensory Regions of the Cerebral Cortex



a Major anatomical landmarks on the surface of the left cerebral hemisphere. The lateral sulcus has been pulled apart to expose the insula.

Homunculus of Primary Somatosensory Cortex in Blue

Note that each hemisphere receives info from the opposite side of the body



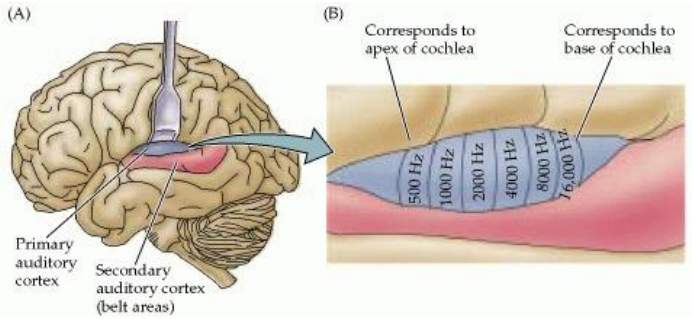
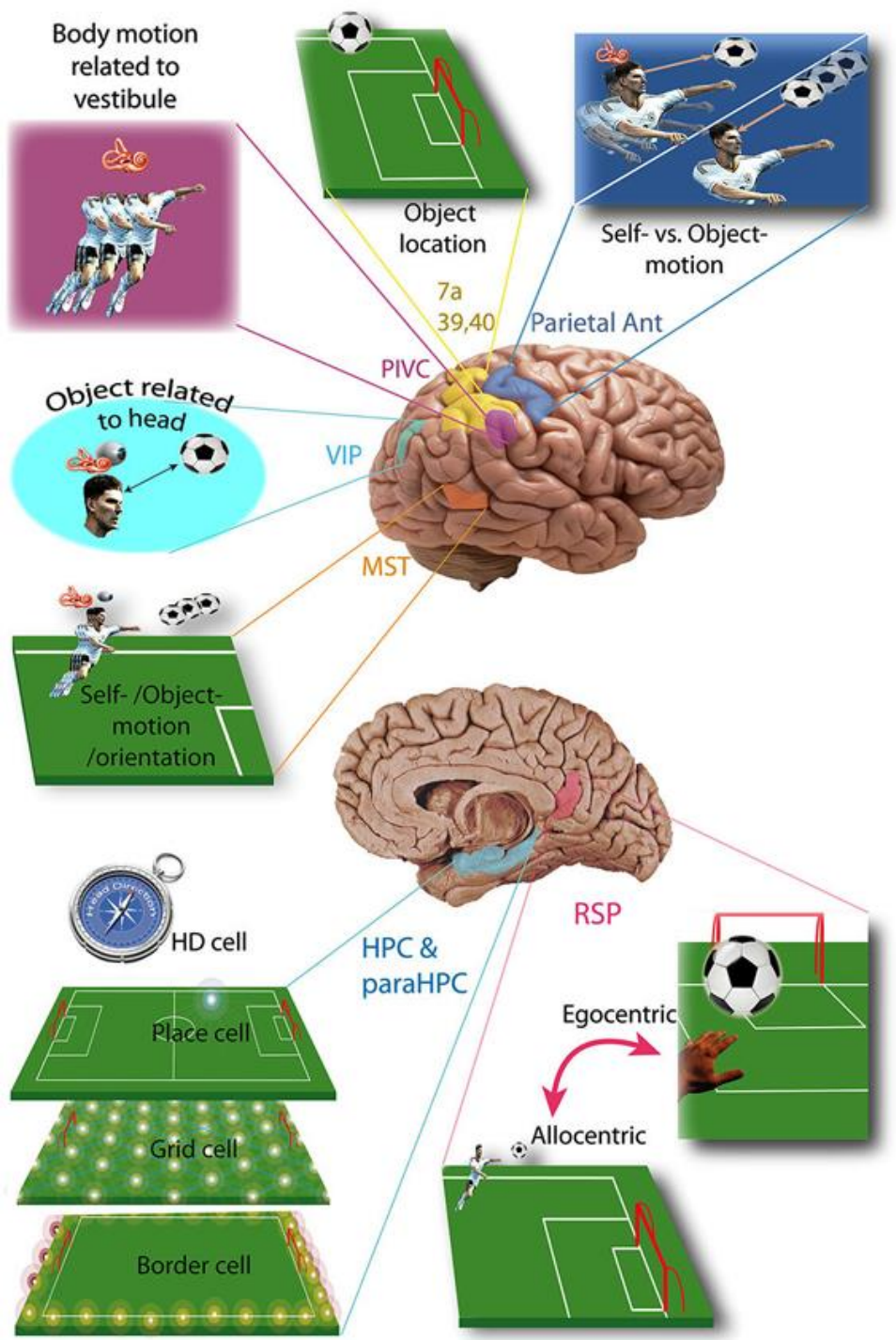


Figure 13.14

The human **auditory cortex**. (A) Diagram showing the brain in left lateral view, including the depths of the lateral sulcus, where part of the **auditory cortex** occupying the superior temporal gyrus normally lies hidden. The primary **auditory cortex** (A1) is shown in blue; the surrounding belt areas of the **auditory cortex** are in red. (B) The primary **auditory cortex** has a tonotopic organization, as shown in this blowup diagram of a segment of A1.



Area 7 – Visuo-Motor Coordination
Point of convergence between vision and proprioception to determine where moving objects are in relation to the body

Area 39 – Angular gyrus, considered by some to be part of Wernicke's area
It is also involved in a number of processes related to language, number processing and spatial cognition, memory retrieval, attention, and theory of mind.
Area 40 – Supramarginal gyrus, involved with language processing. Lesions in it an result in receptive aphasia

Vestibular cortices and spatial cognition. Vestibular cortices involved in spatial cognition, illustrated in a soccer player: HD cell, head direction cell; HPC, hippocampus; MST, medial superior temporal area; ParaHPC, parahippocampal cortex; Parietal Ant, parietal anterior cortex; PIVC, parieto-insular vestibular cortex; RSP, retrosplenial cortex; VIP, ventral intraparietal area; 7a, 39, 40 Brodmann area.

Wide dissemination of vestibular signals: maybe an evolutionary process to create a neural network with sparse coding

Sparse coding:

- Low energy cost
- Large storage capacity
- Rapid learning ability,
- Tolerance to degradation

Higher vestibular function

We are taught to clinically think in terms of peripheral versus central based on anatomical lesions

There is a lot of overlap in clinical presentations

Brandt T, Strupp M and Dieterich M, Toward a concept of disorders of "higher vestibular function", Front Integ Neuroscience, 2014,8 doi:10.3389/fnint.2014.00047

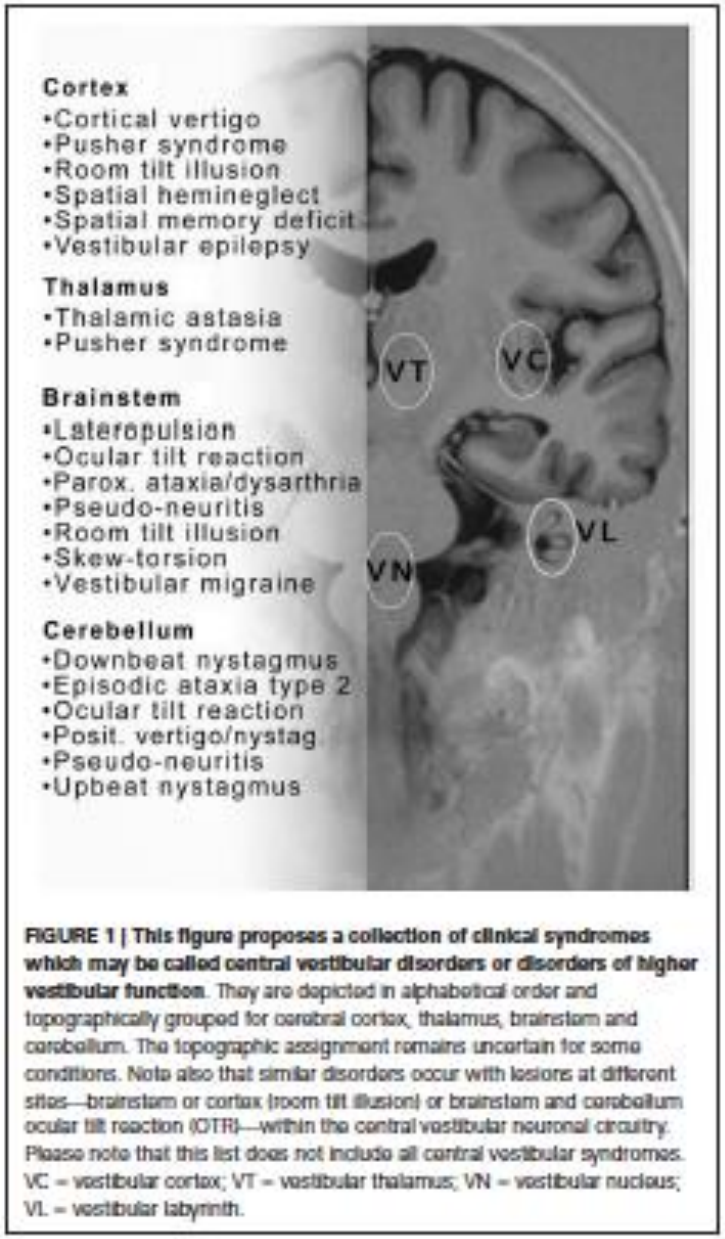


FIGURE 1 | This figure proposes a collection of clinical syndromes which may be called central vestibular disorders or disorders of higher vestibular function. They are depicted in alphabetical order and topographically grouped for cerebral cortex, thalamus, brainstem and cerebellum. The topographic assignment remains uncertain for some conditions. Note also that similar disorders occur with lesions at different sites—brainstem or cortex (room tilt illusion) or brainstem and cerebellum ocular tilt reaction (OTR)—within the central vestibular neuronal circuitry. Please note that this list does not include all central vestibular syndromes. VC = vestibular cortex; VT = vestibular thalamus; VN = vestibular nucleus; VL = vestibular labyrinth.

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Thank you
Questions?