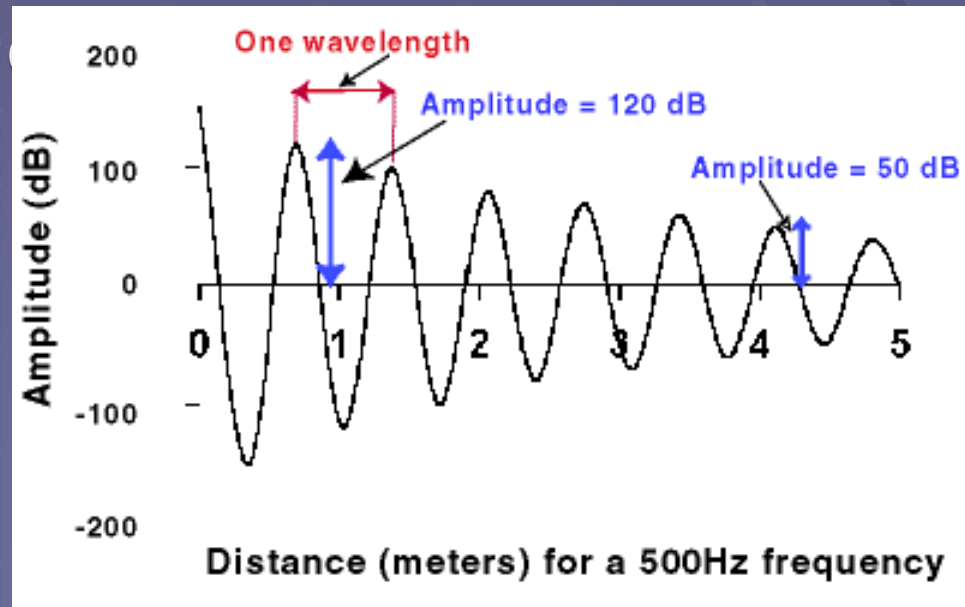


# The Special Senses

## 3) Auditory

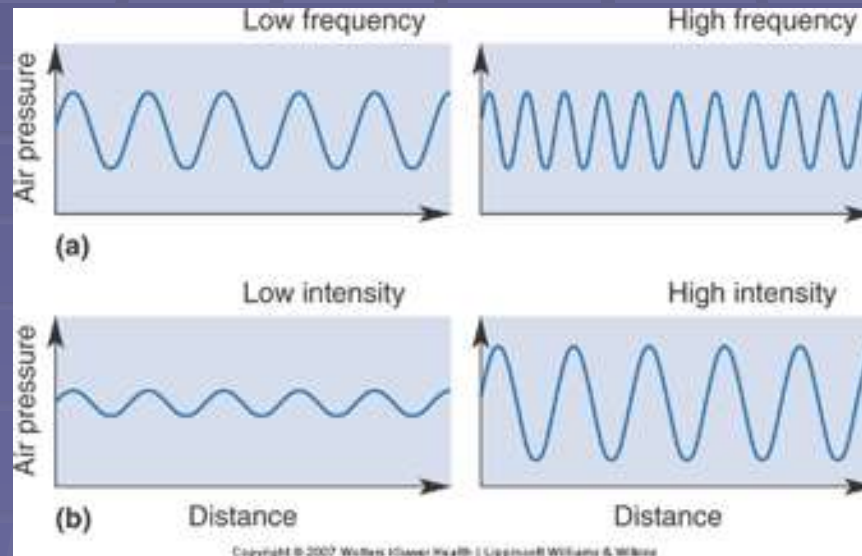
# The Nature of Sound

- All waves have basic characteristics:
  - Amplitude
  - Frequency
  - Wavelength
- These properties have particular names with reference to sound
  - Amplitude = loudness
  - Frequency = pitch

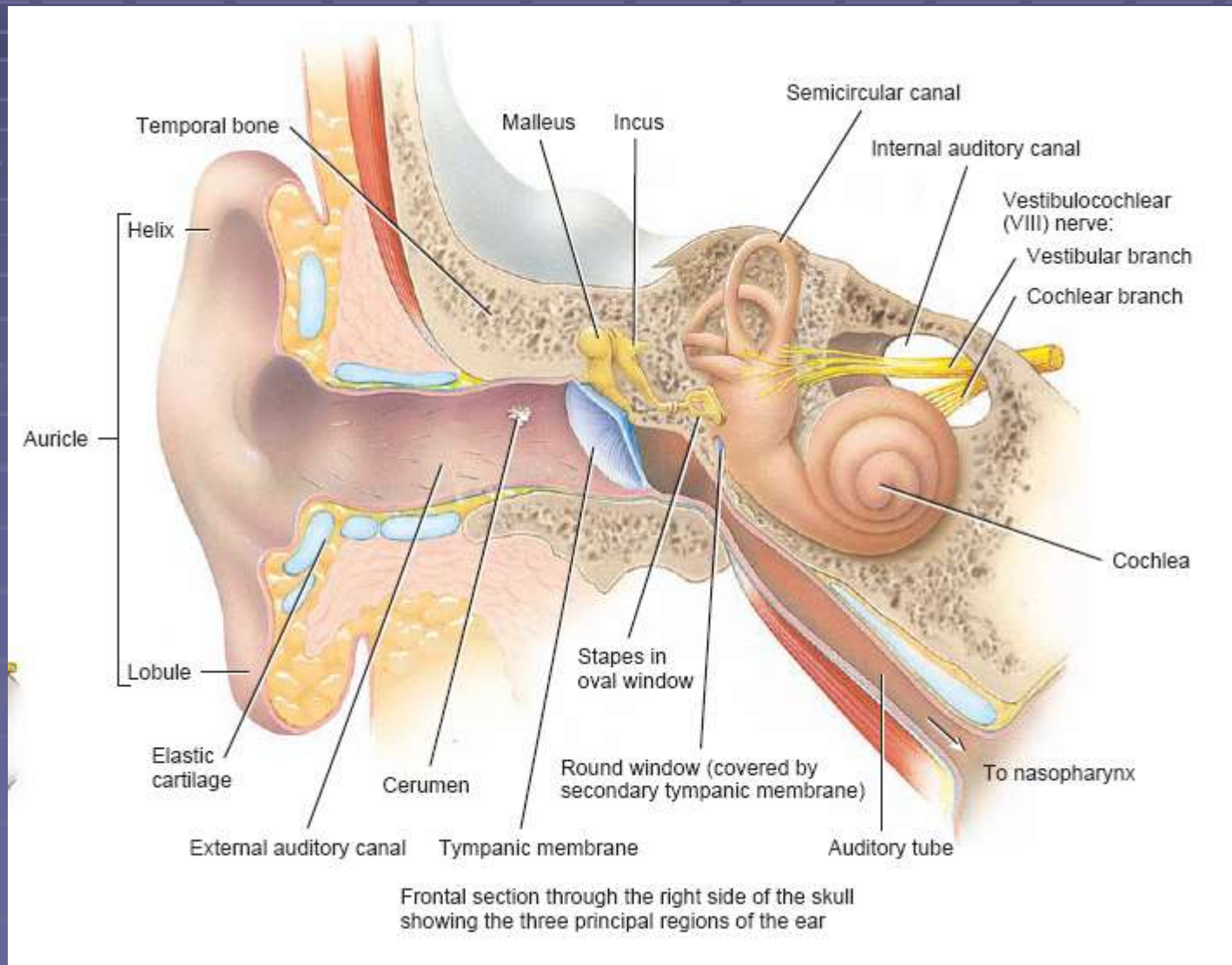


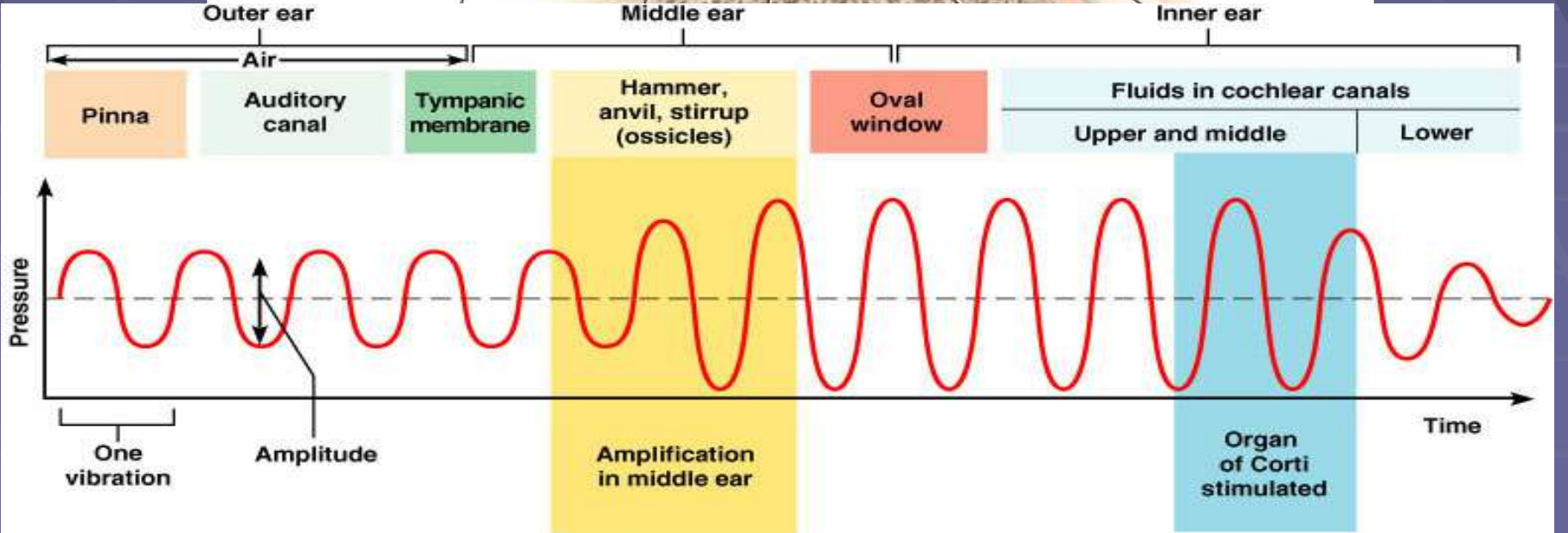
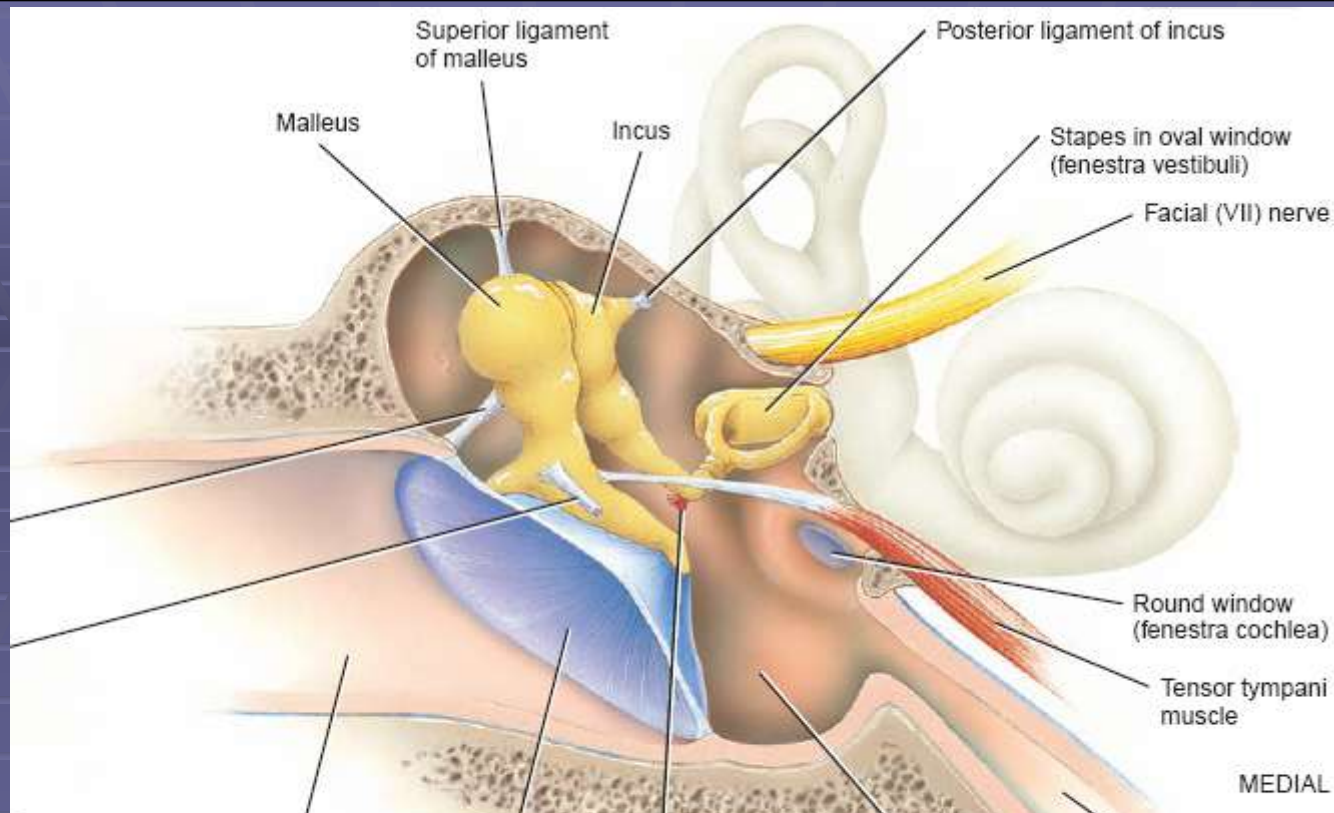
# The Nature of Sound

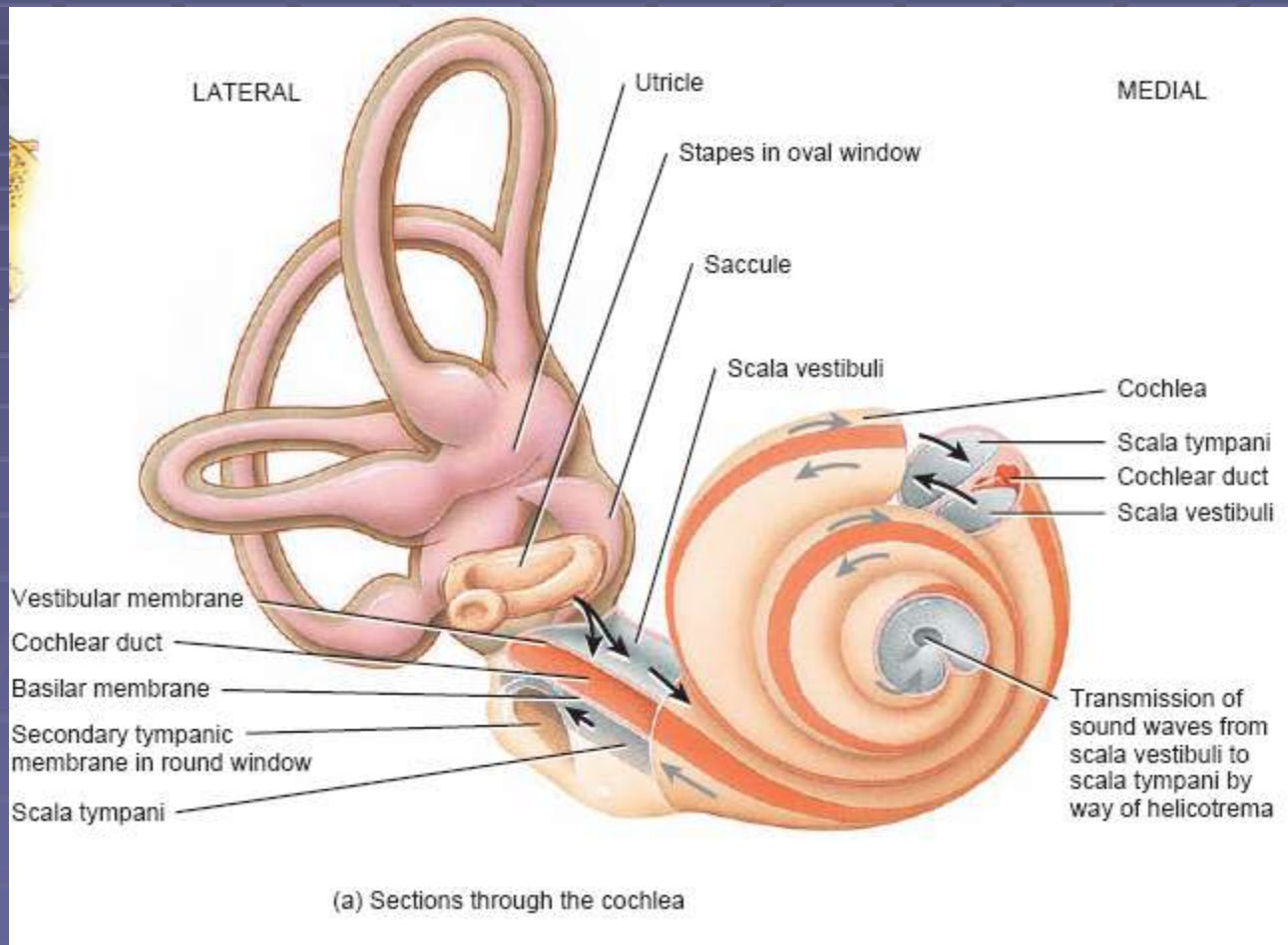
- Sound
  - Range: 20 Hz to 20,000 Hz
  - Pitch: High pitch = high frequency; low frequency = low pitch
  - Intensity: High intensity louder than low intensity

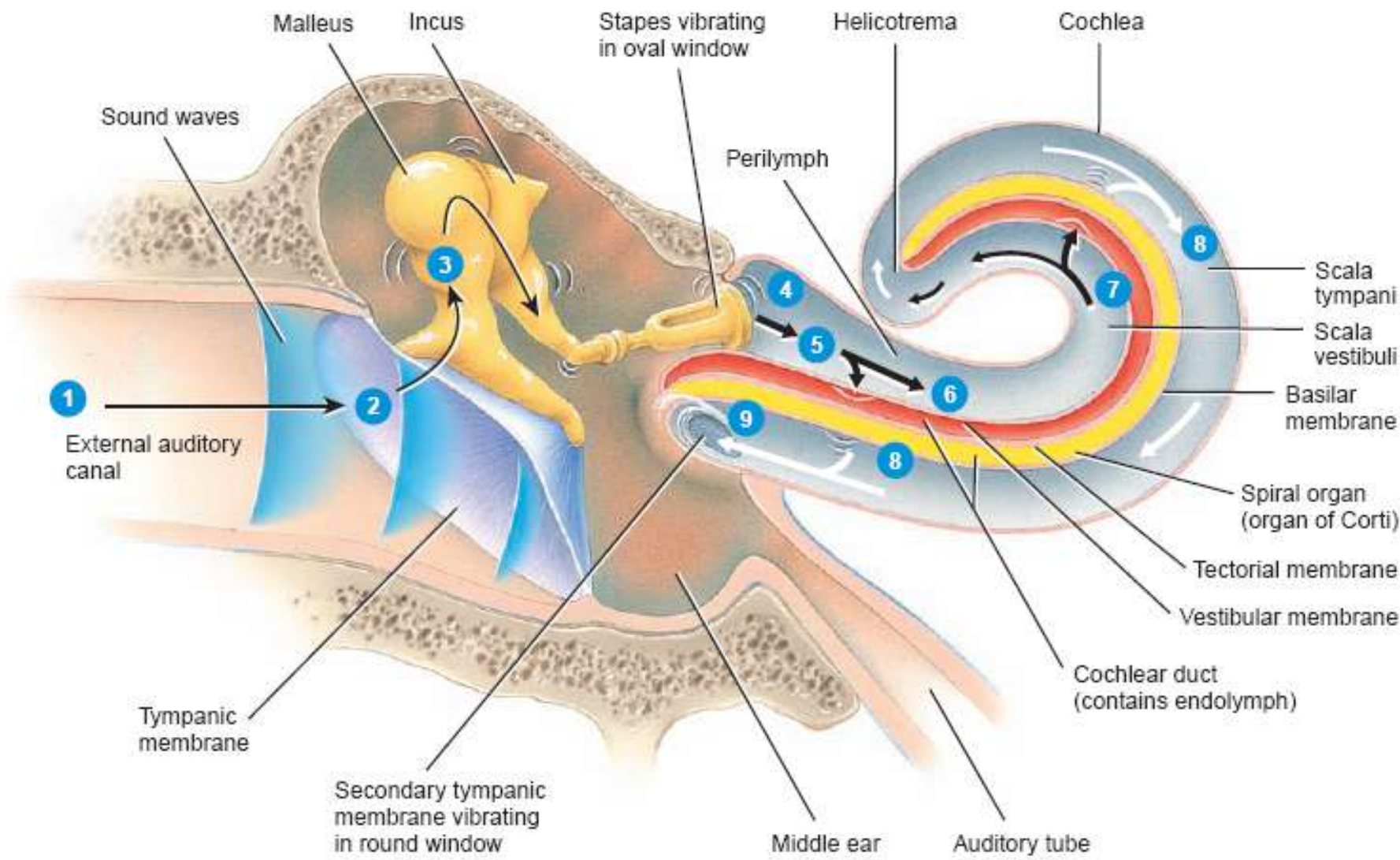


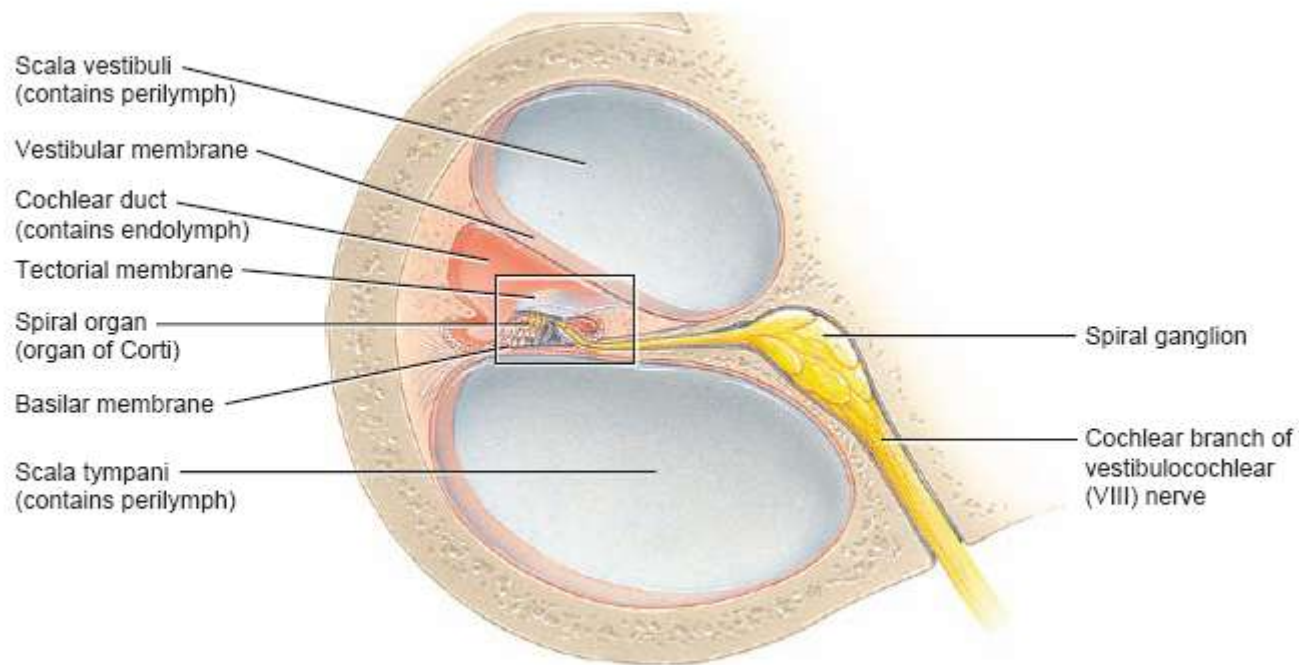
# Anatomy of the ear







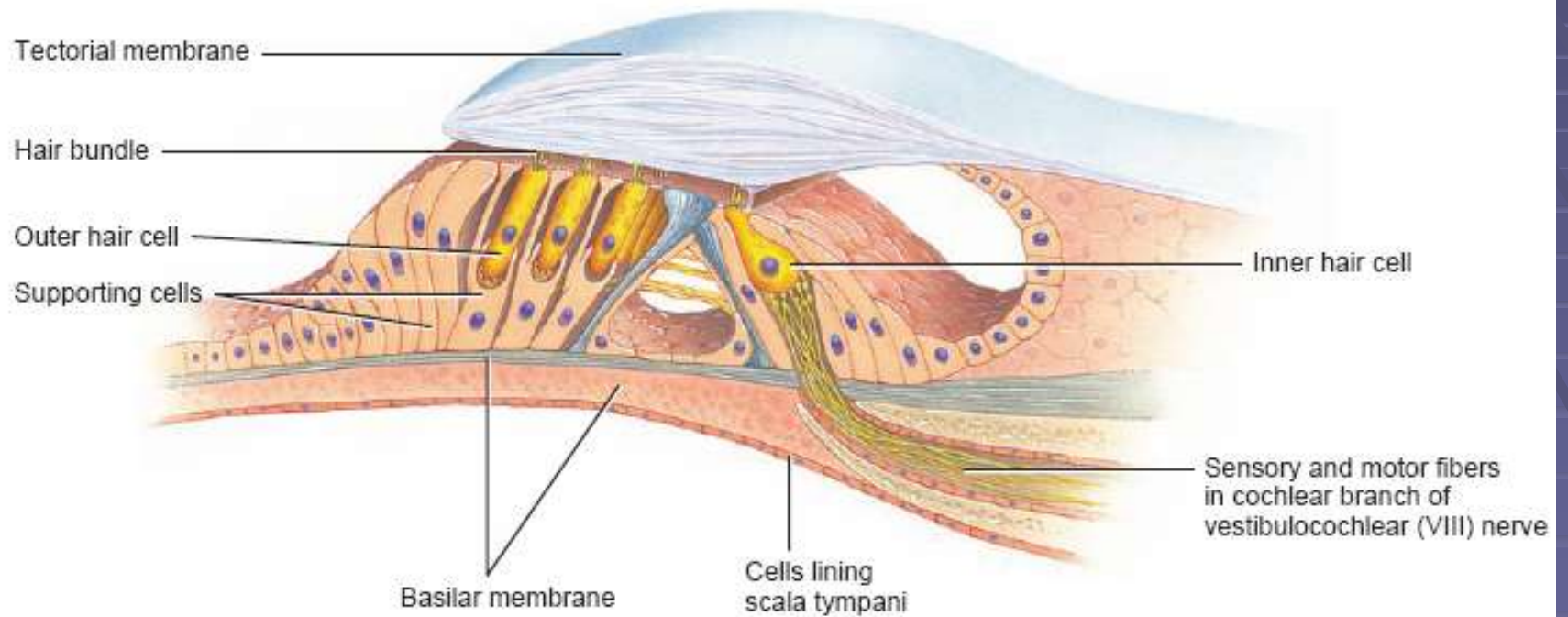




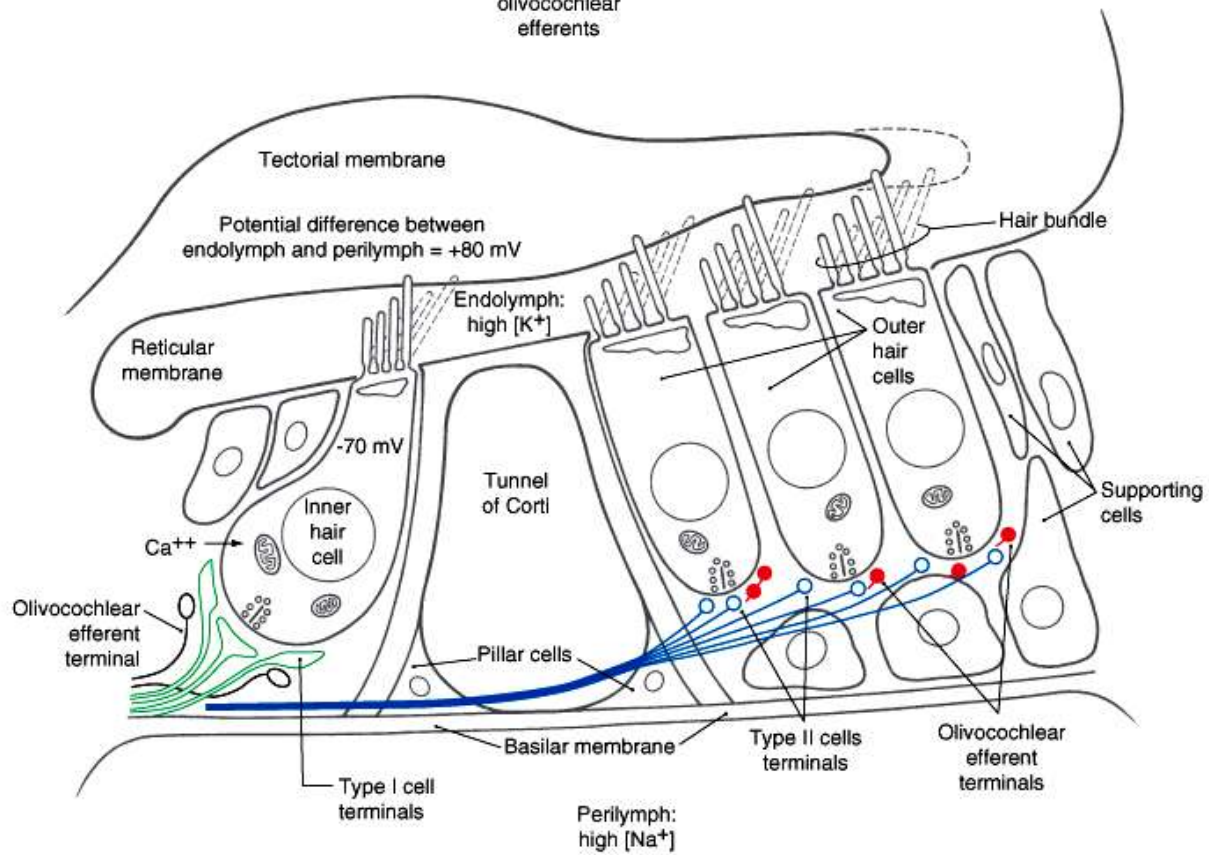
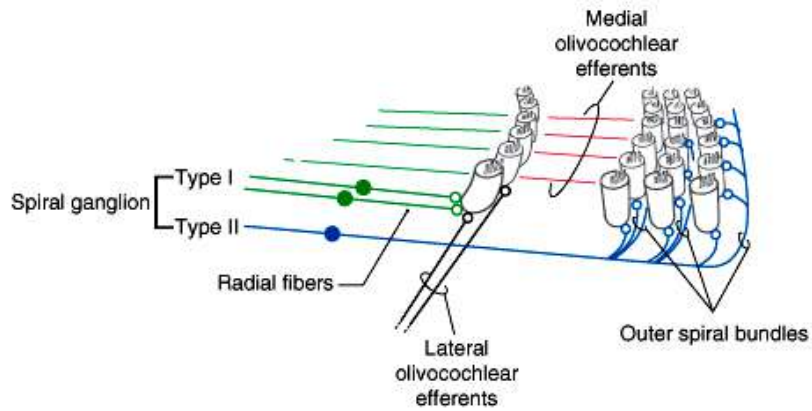
(c) Section through one turn of the cochlea

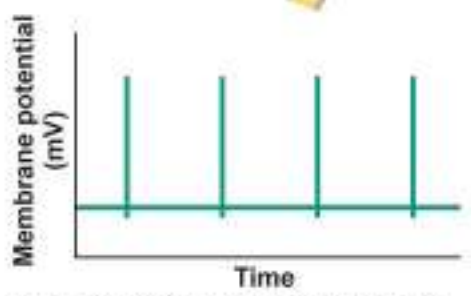
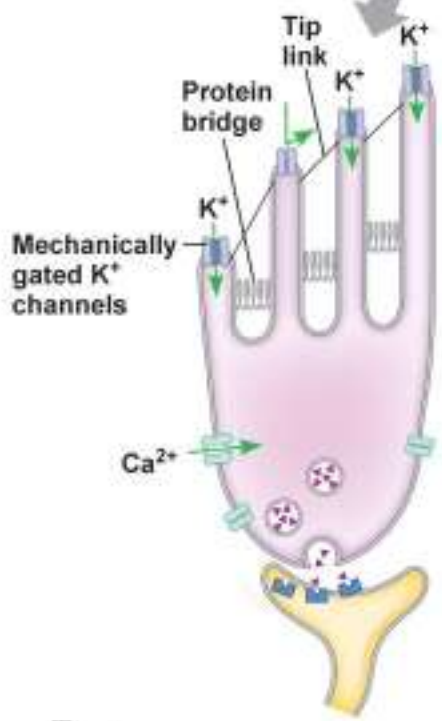
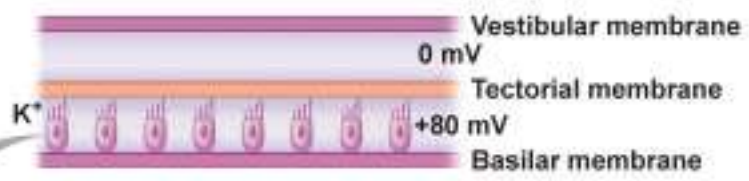


(c) Section through one turn of the cochlea

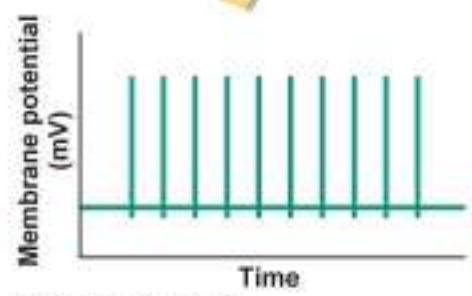
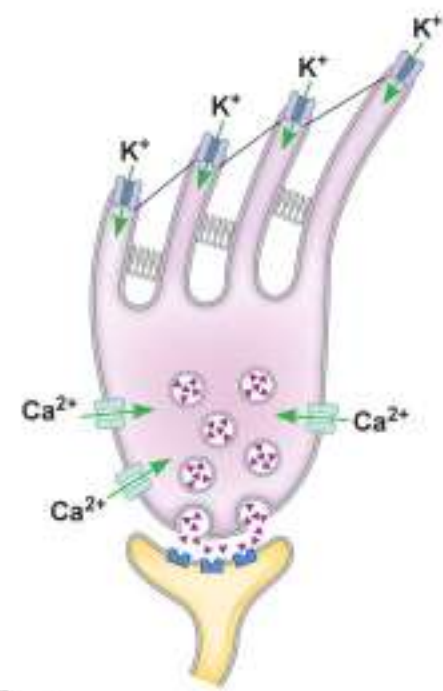


(d) Enlargement of spiral organ (organ of Corti)

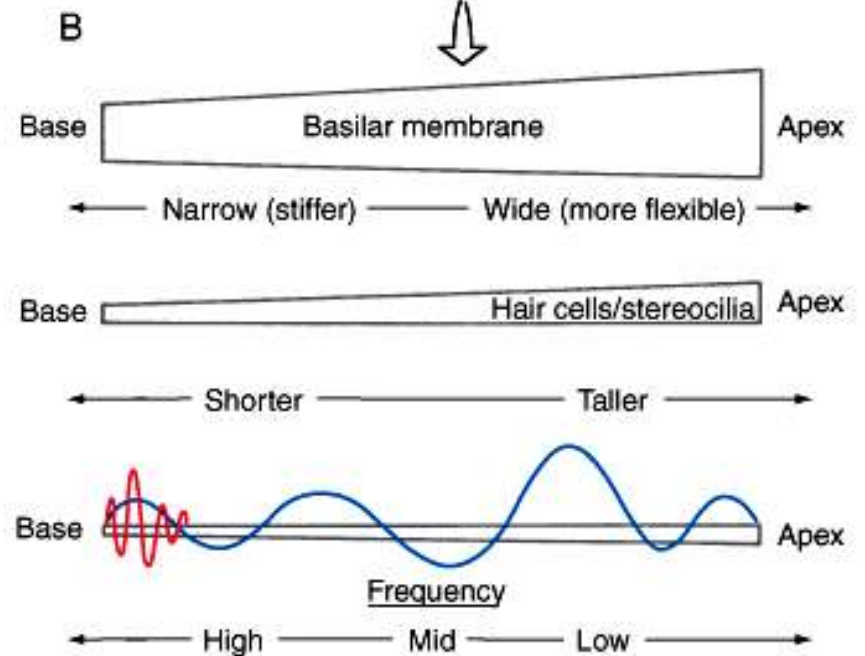
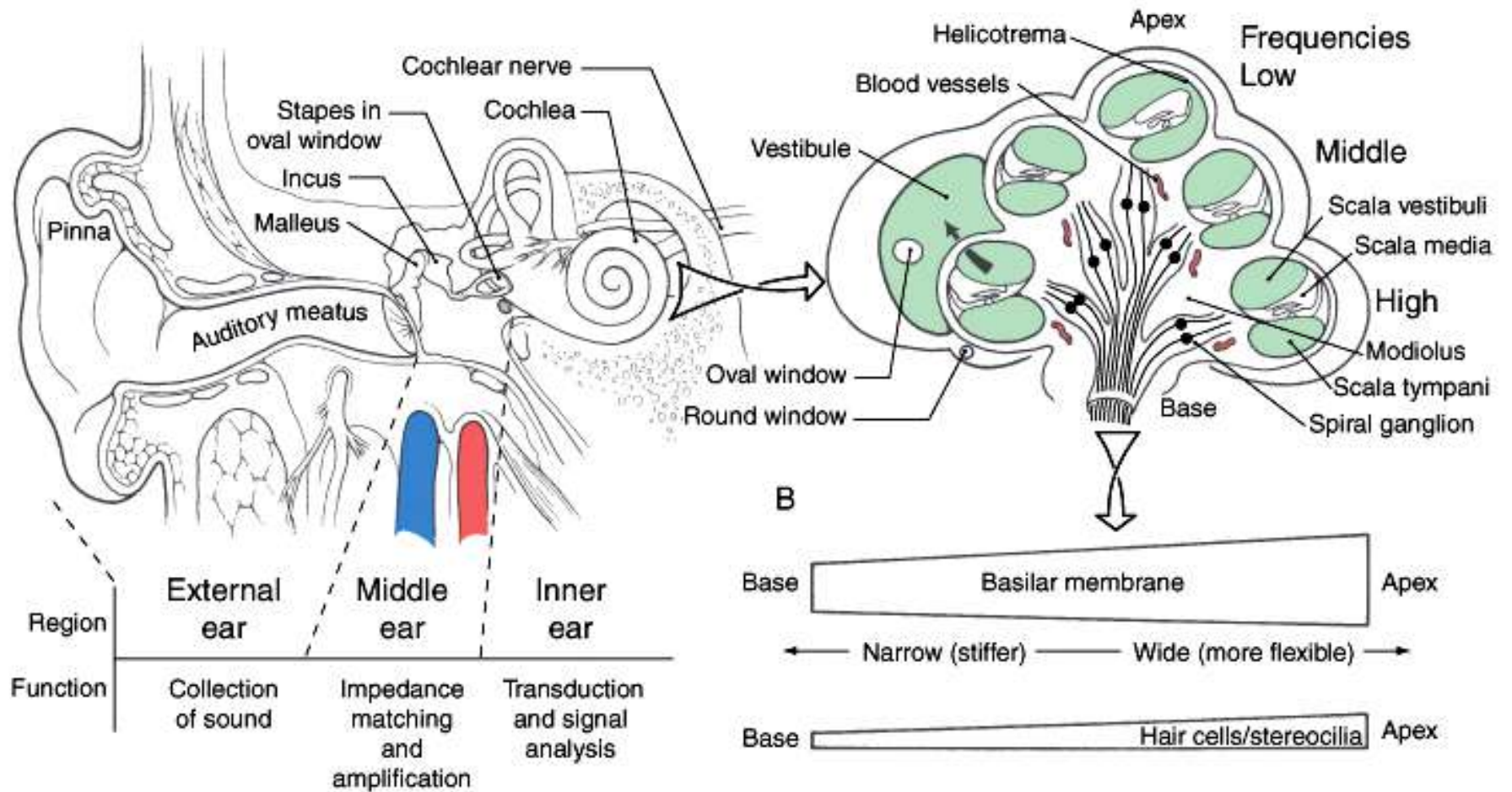




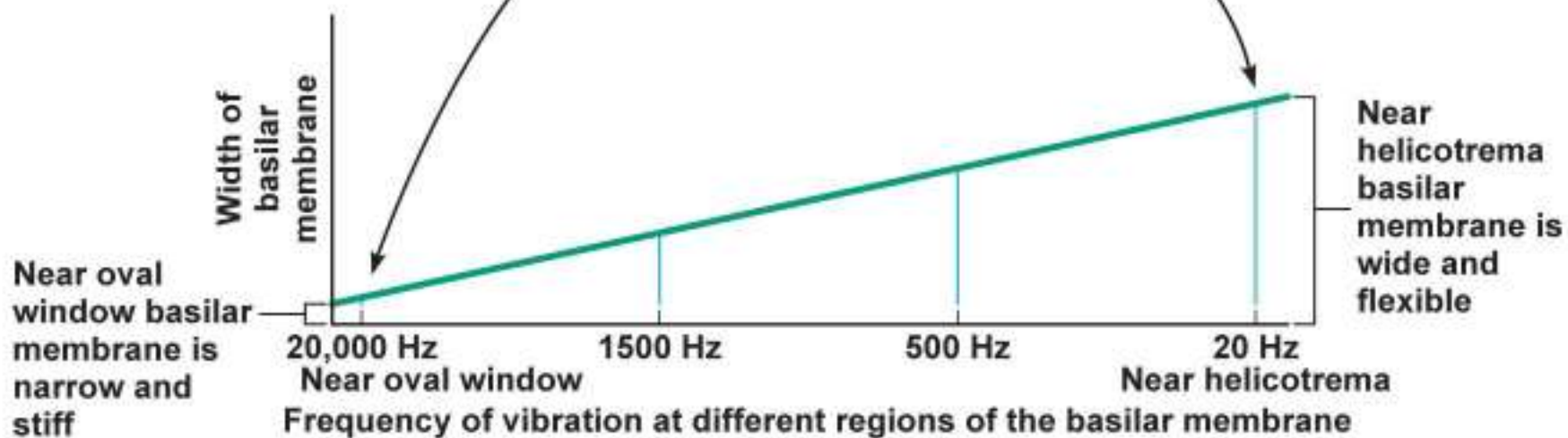
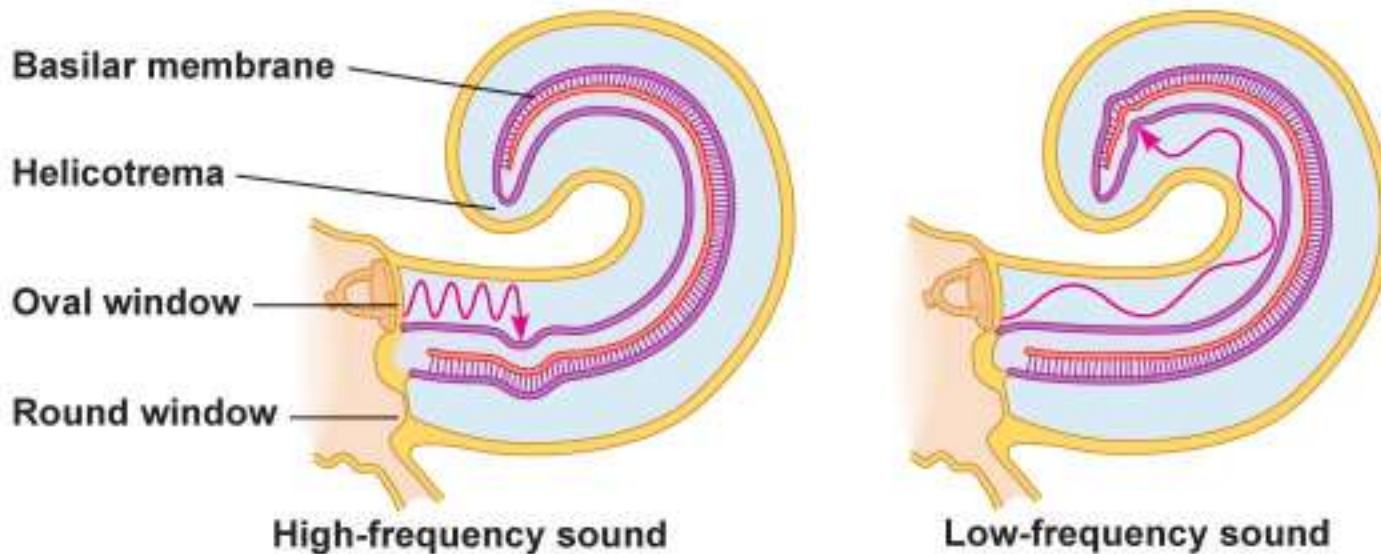
(a) At rest (partially depolarized)

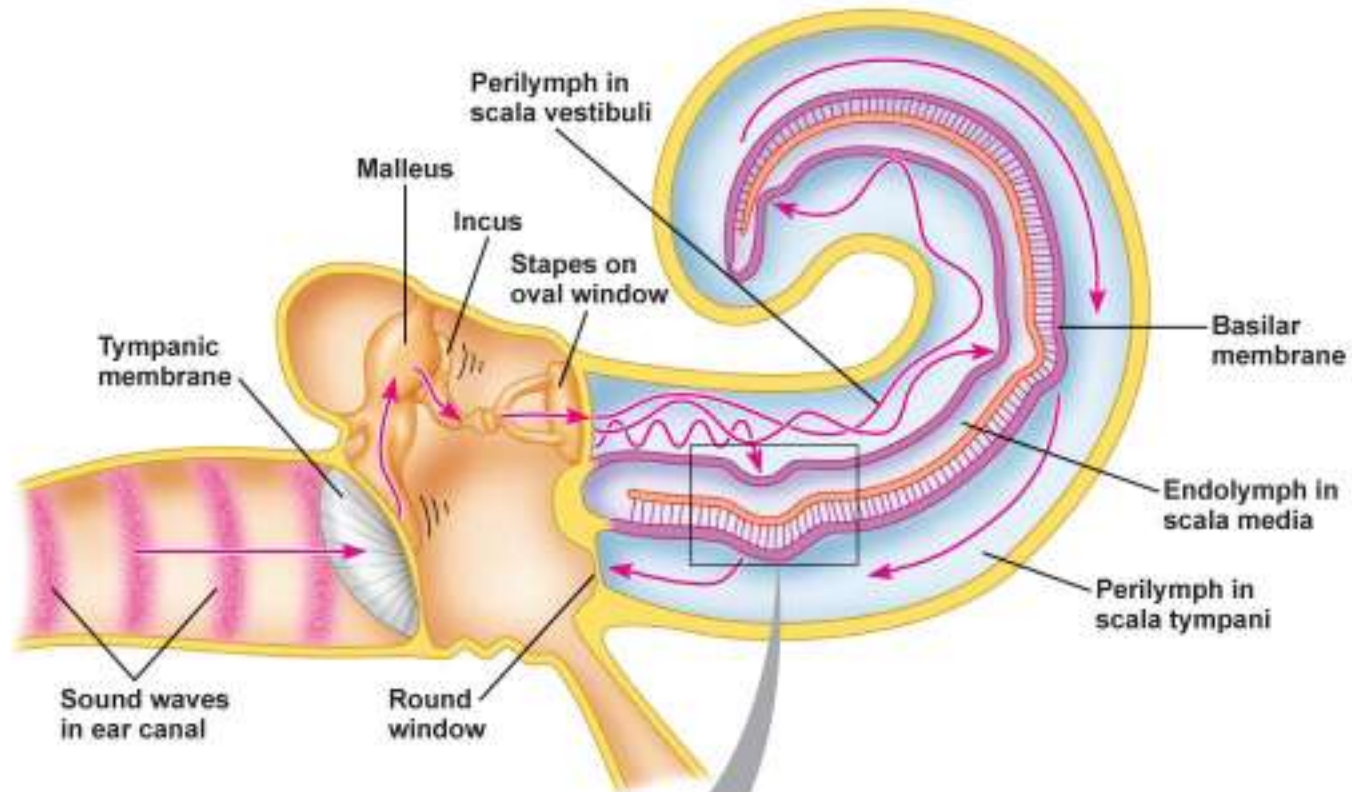


(b) Depolarized

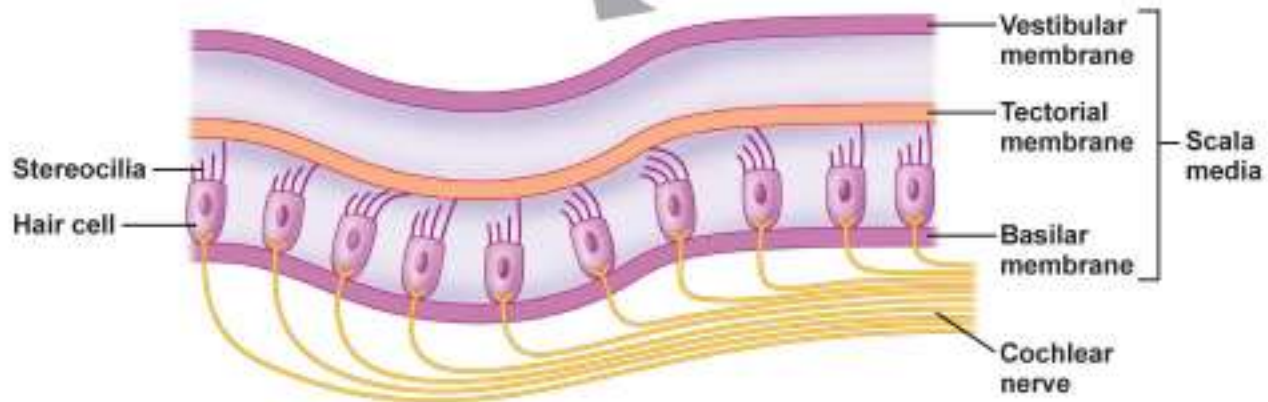


C

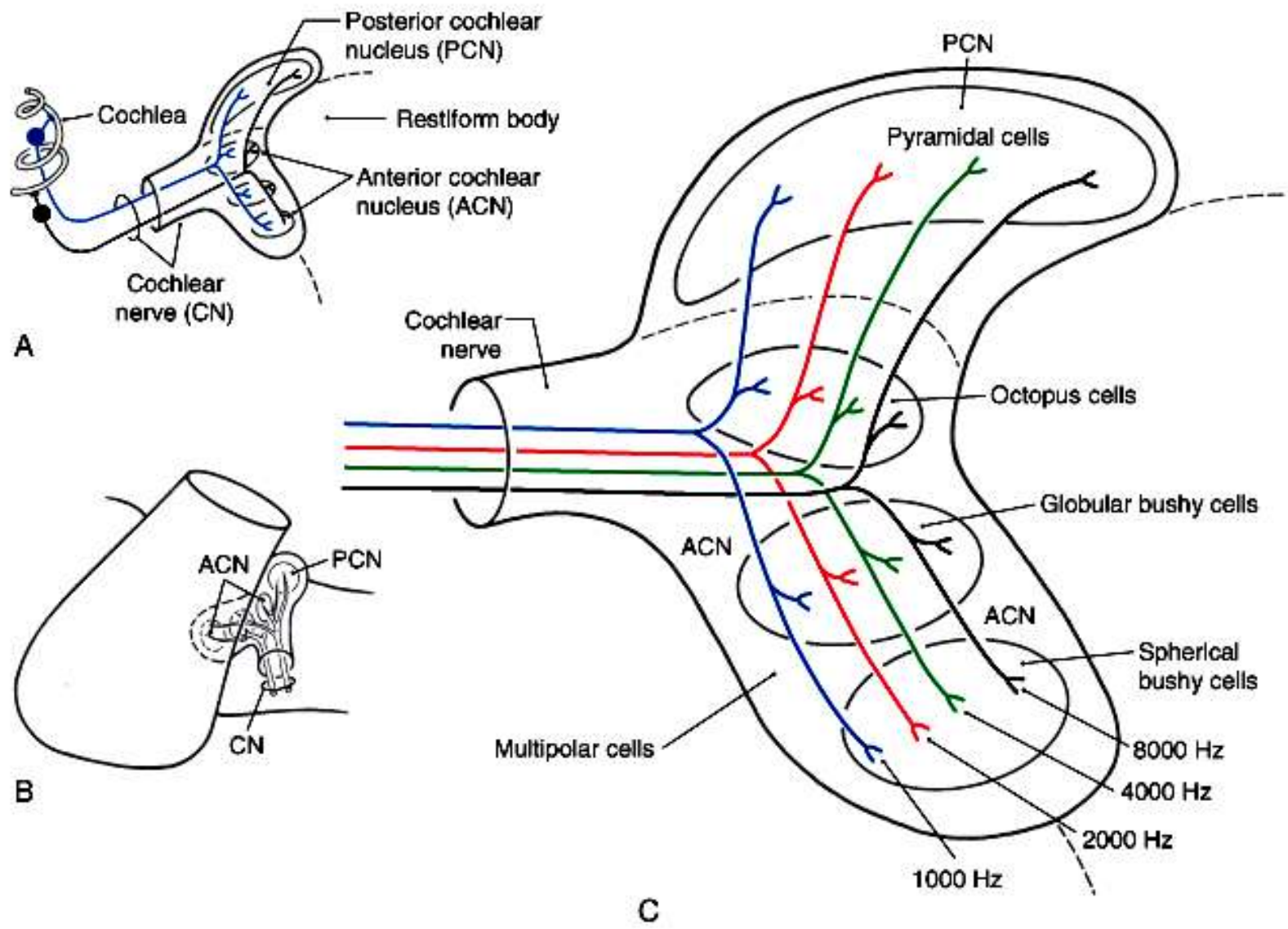




(a)

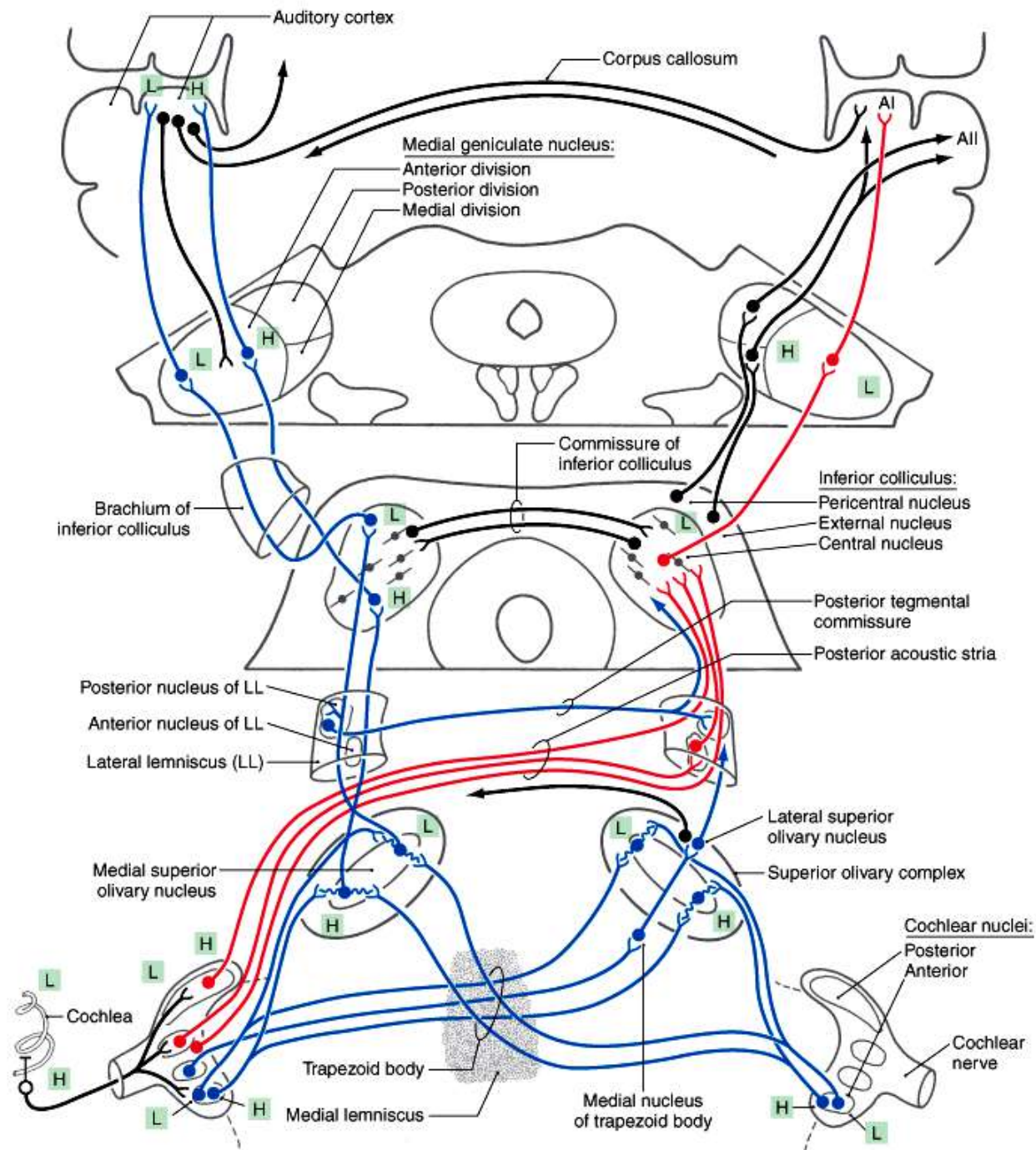


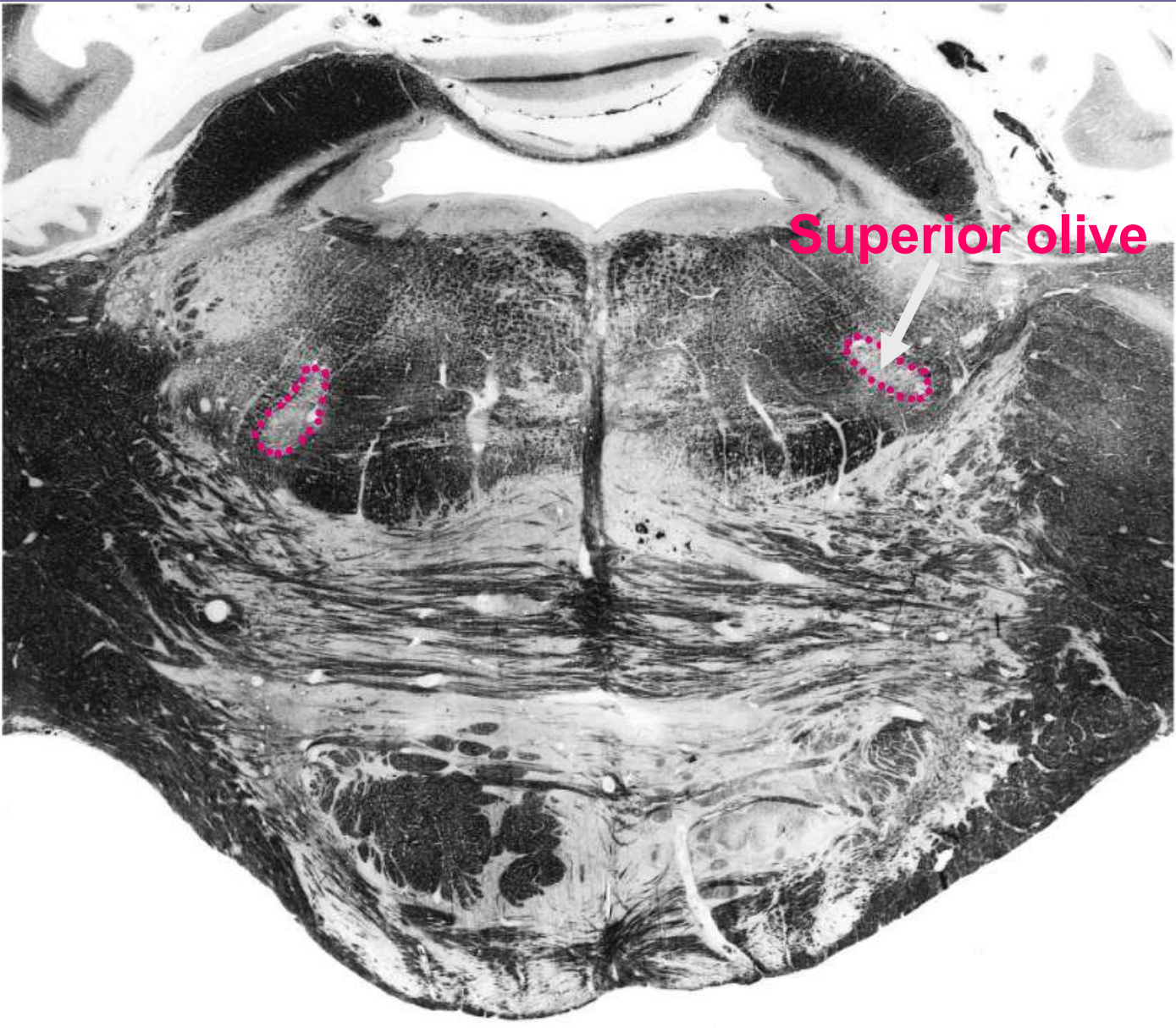
(b)

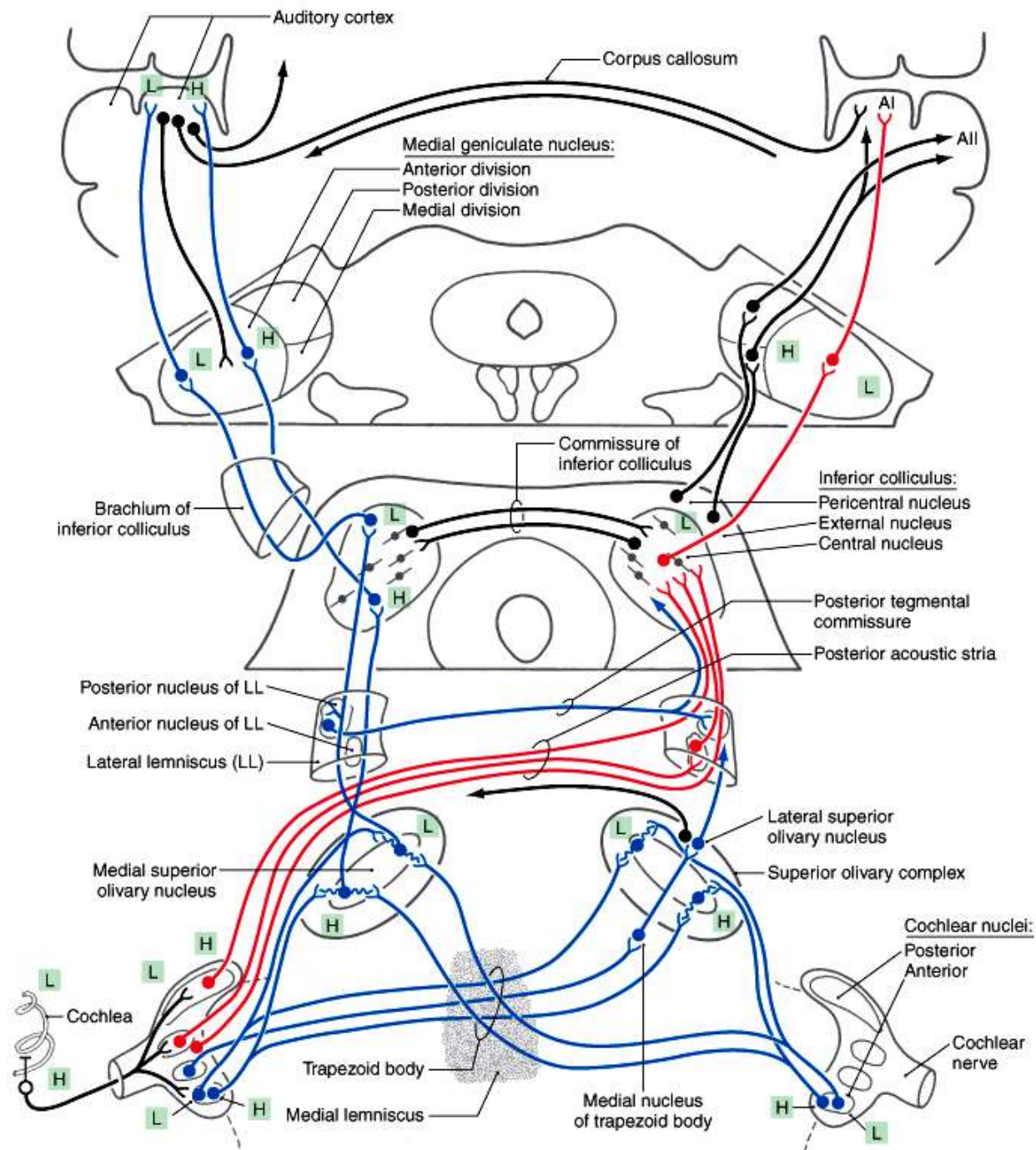


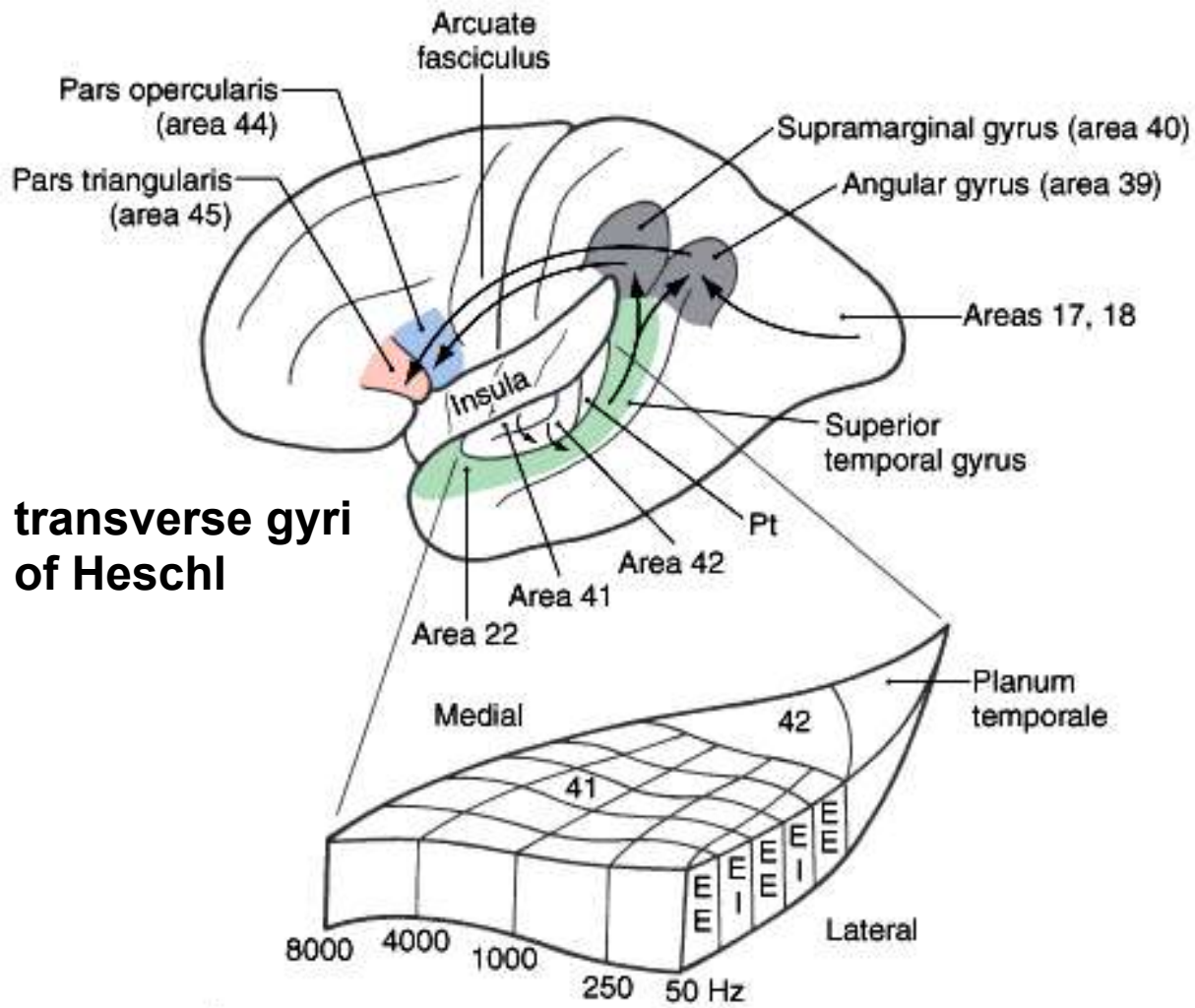






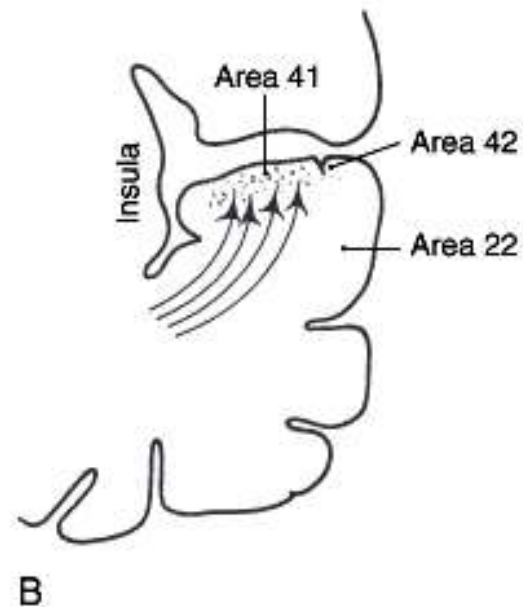


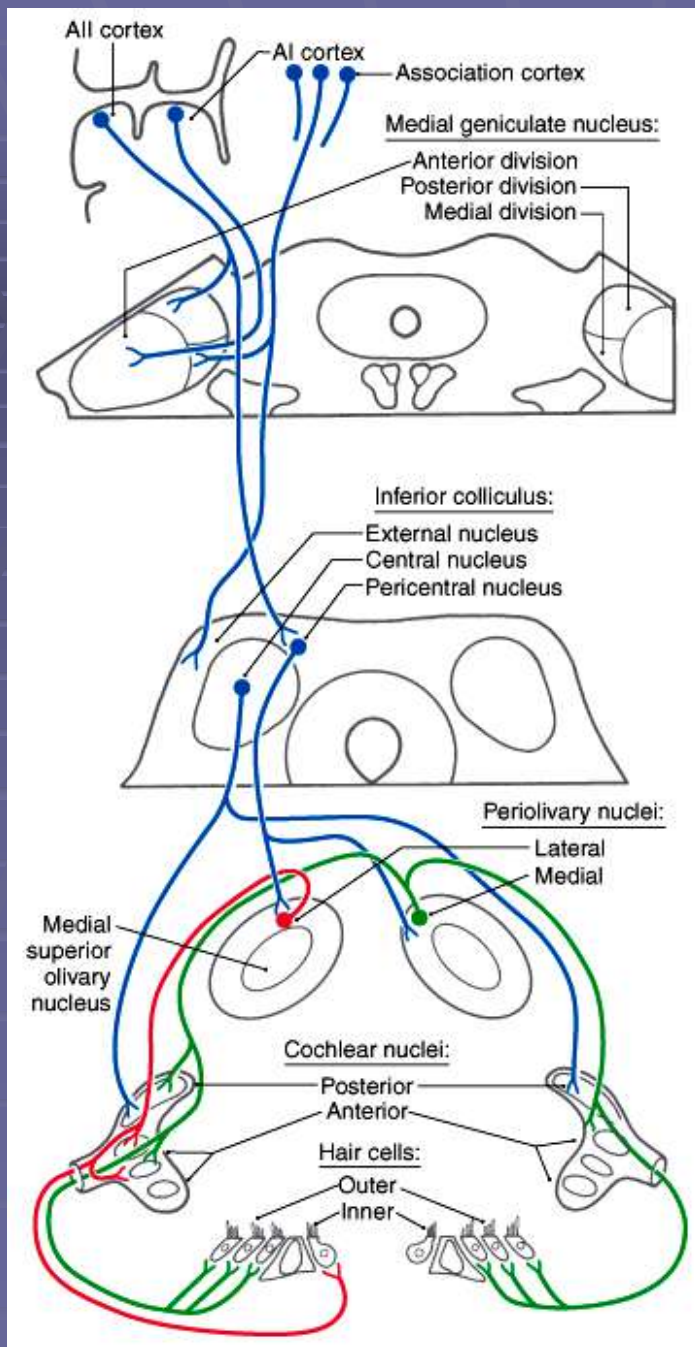




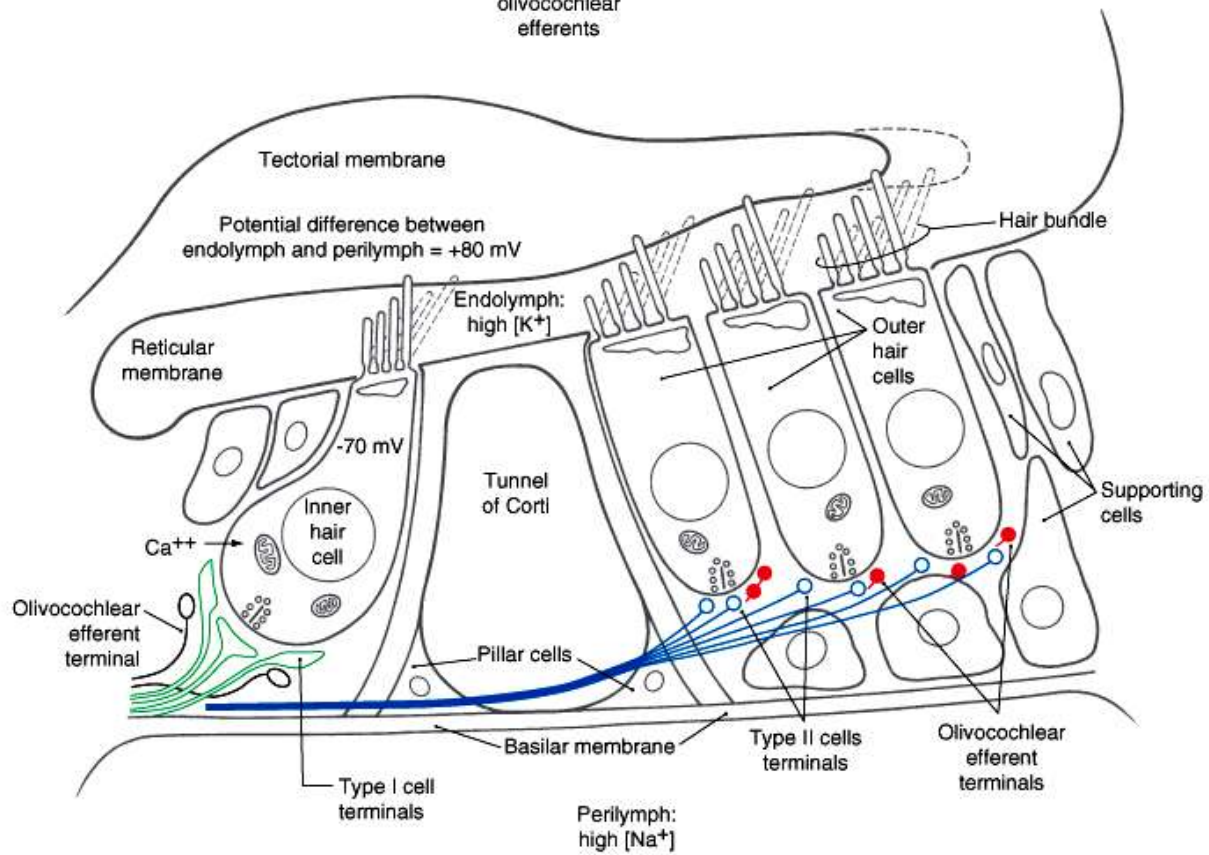
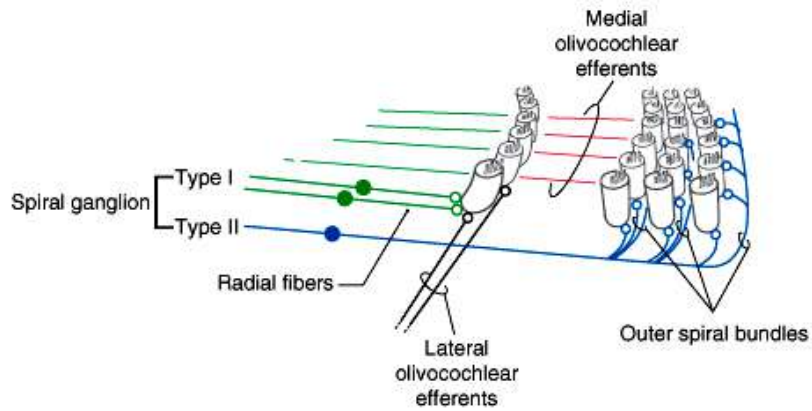
**transverse gyri  
of Heschl**

**A**



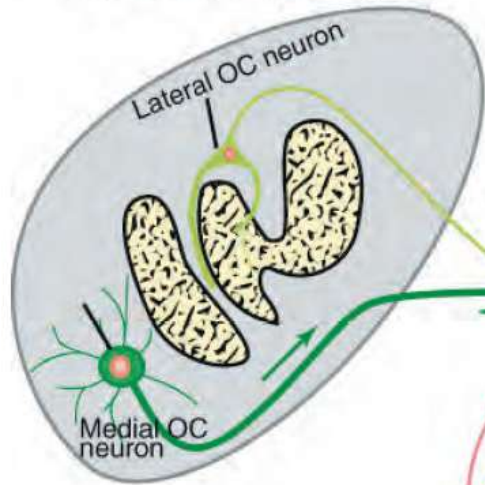


## Olivocochlear descending feedback loop



# Organ of Corti

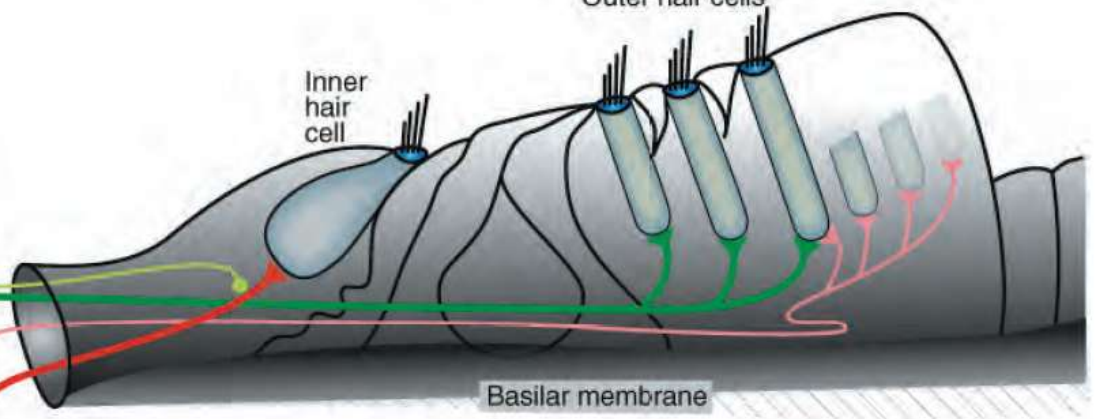
## Superior olivary complex



## Outer hair cells

## Inner hair cell

## Basilar membrane

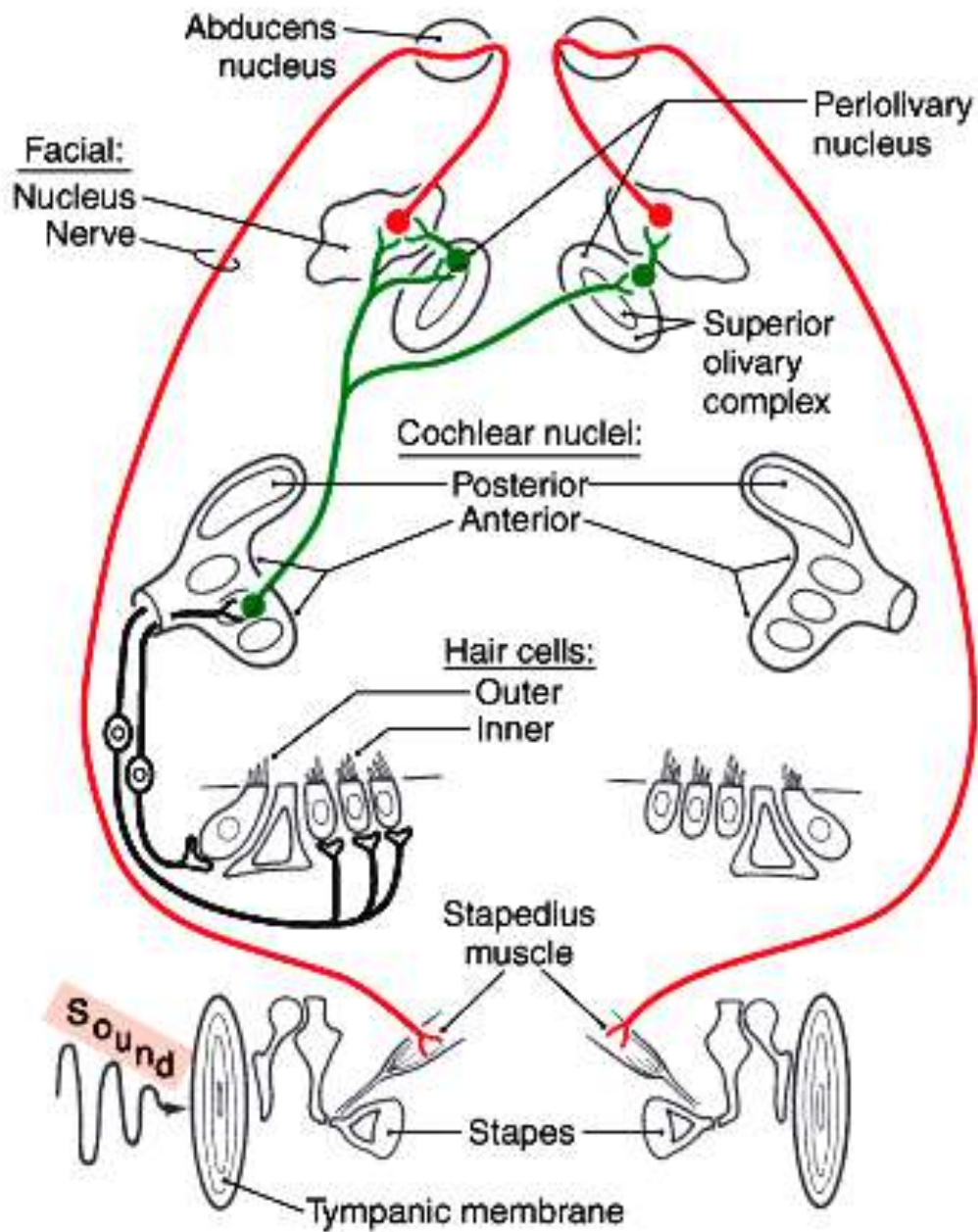


## Type I neuron

## Type II neuron

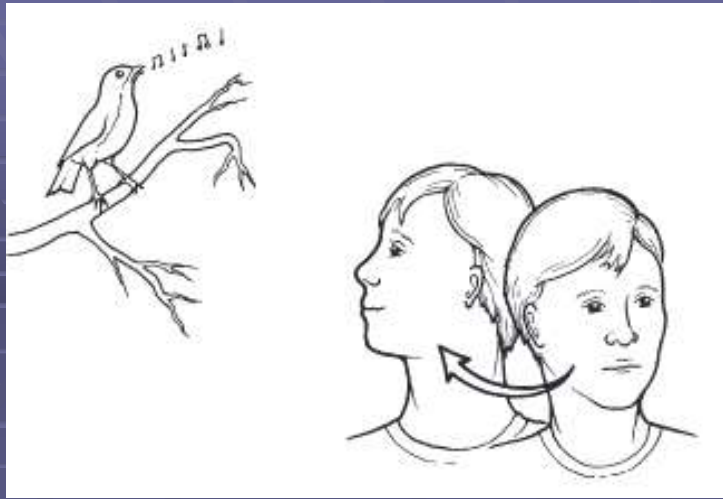
## Auditory nerve



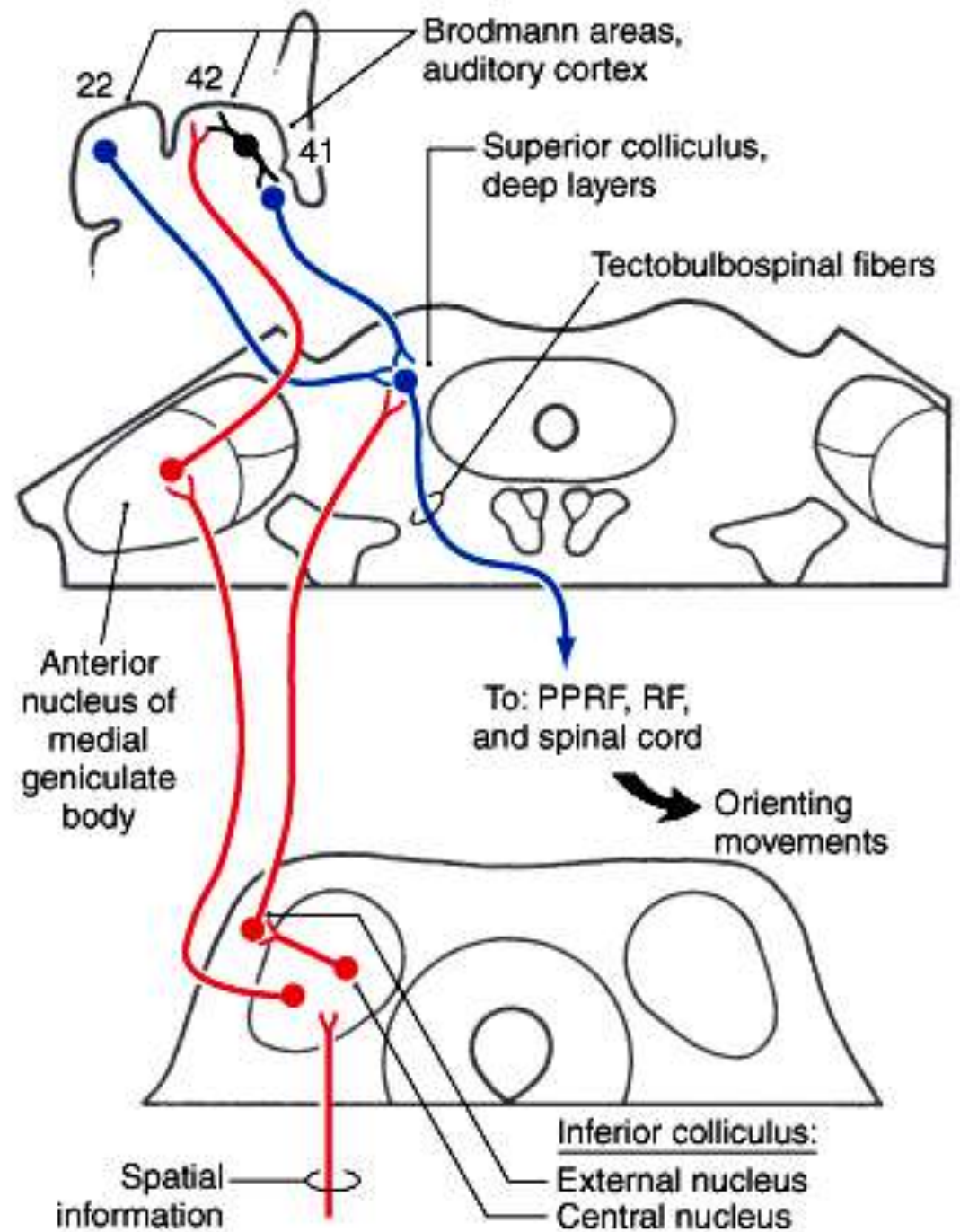


## Middle ear reflex arc





## Acoustic startle reflex



# Types of Deafness

## ■ Conductive Deafness

- Due to impaired sound transmission in external and middle ear
- Impacts all sound frequencies
- Causes:
  - Plugging of the EAC with cerumen or foreign body
  - Otitis externa and otitis media
  - Perforation of eardrum
  - osteosclerosis

# Types of Deafness

- **Sensorineural Deafness**
  - Due to loss of cochlear hair cells
  - Problems with CN VIII
  - Lesions within the Central Auditory Pathway
  - Impairs the ability to hear certain pitches (permanent)
  - Causes:
    - Aminoglycosides
    - Prolonged exposure to noise
- Tumors and vascular damage (Pontine auditory hallucinosis)

# Pontine auditory hallucinosis

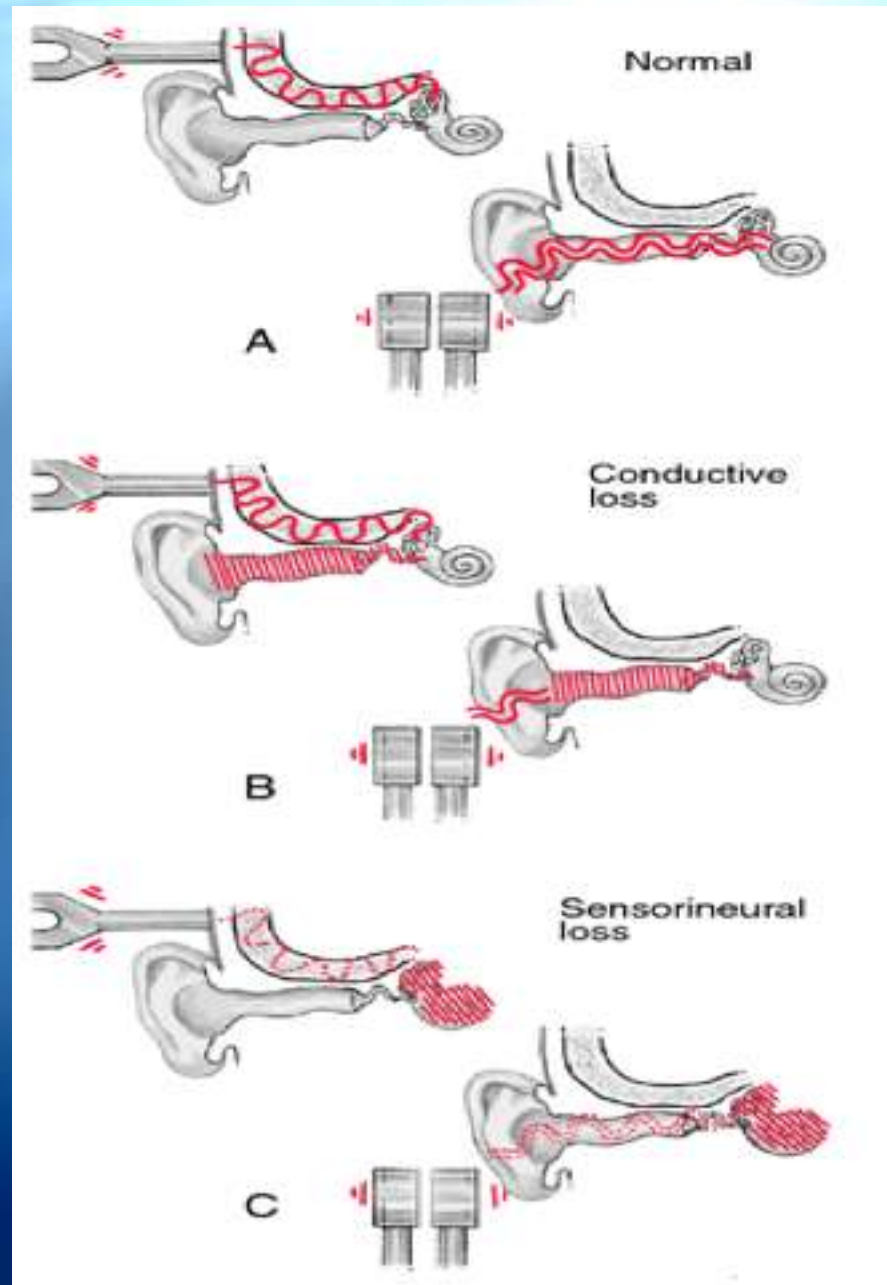
- perceived auditory events that sounds like and an orchestra out of tune, buzzing insects, or strands of music, which are accompanied by more typical symptoms of pontine lesions of auditory , such as cranial nerve deficits and long tract signs. A perception of noise or sounds may also be experienced by patients with temporal lobe seizures or a temporal lobe lesion that damages auditory cortices

# Tinnitus

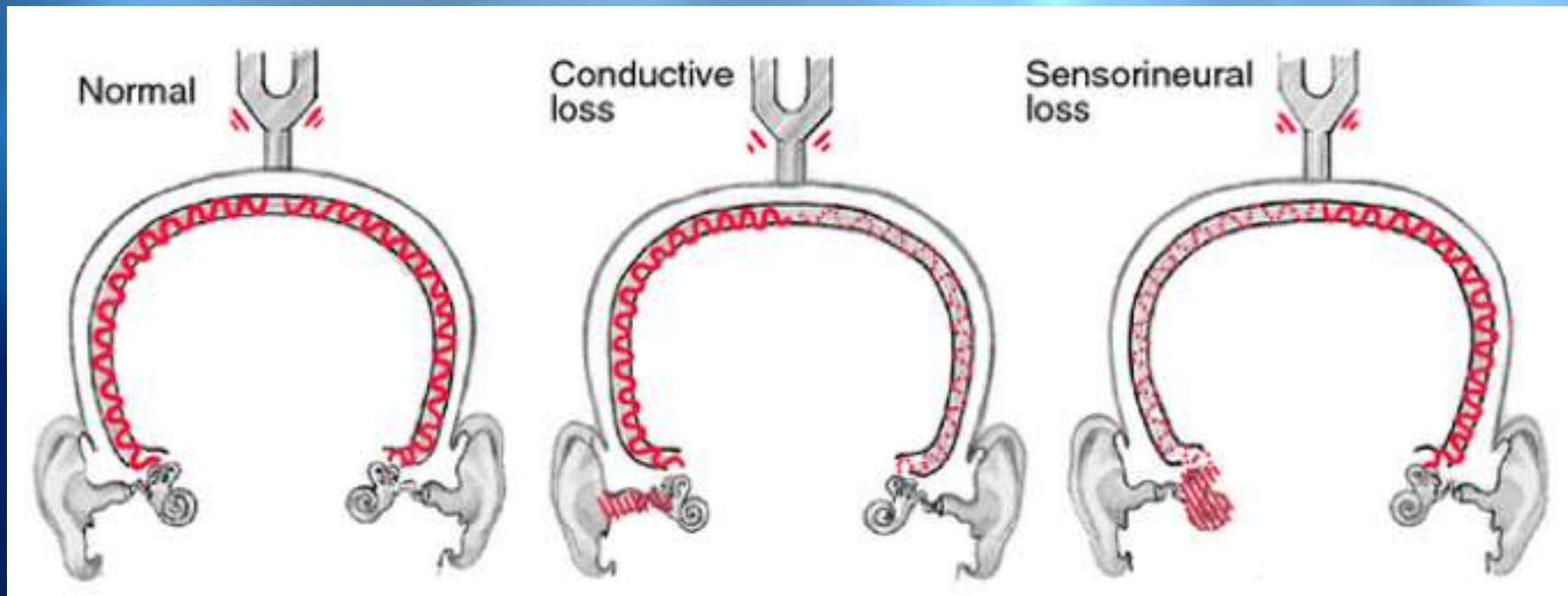
Some of these causes include high blood pressure, diabetes, listening to loud music, a tumor, thyroid conditions, and medications / antidepressants, sedatives, antibiotics, anti-inflammatories, and aspirin.

# Rinne and Weber Tests

# Rinne's test



# Weber's test

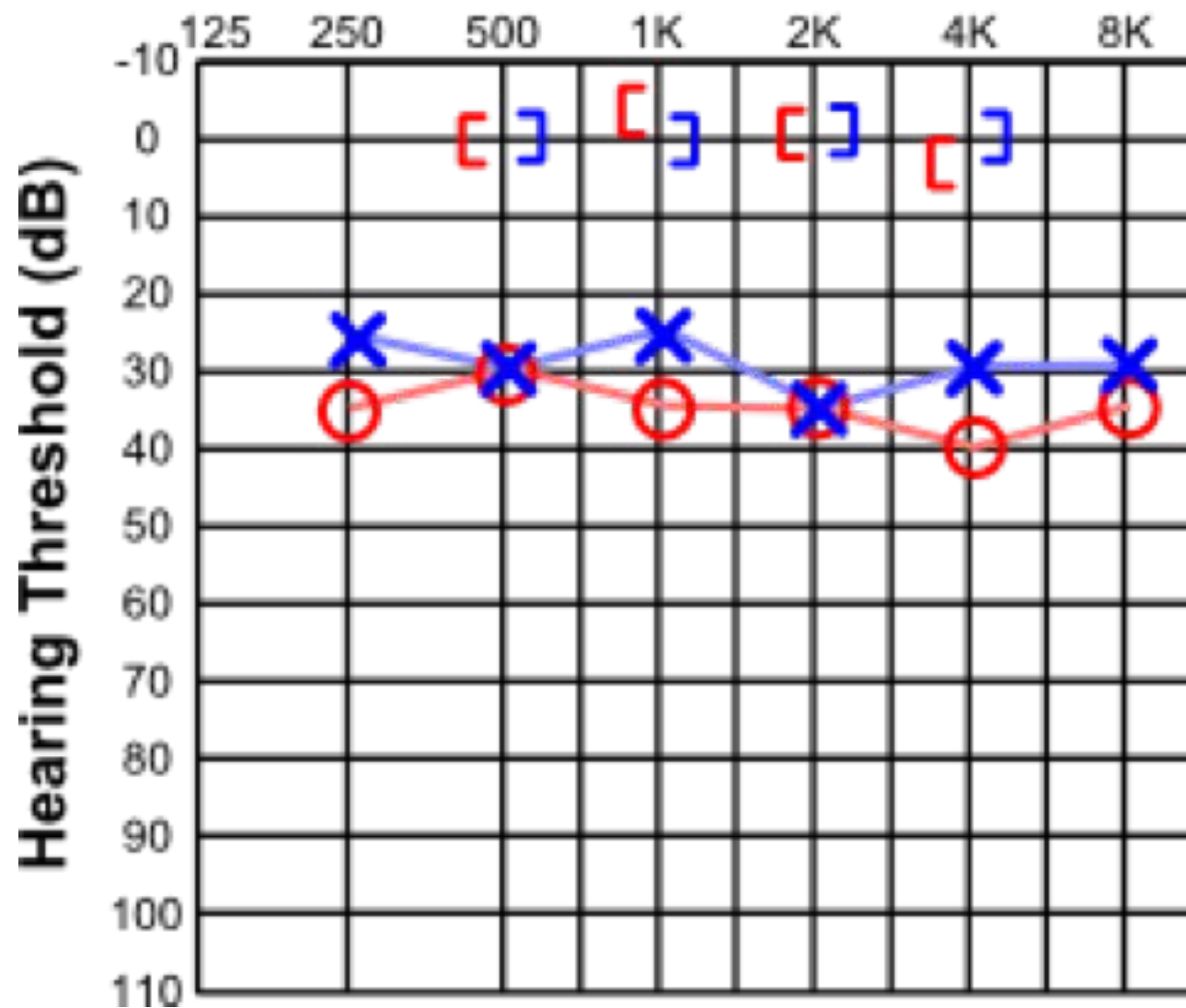




**Table 13-1 Common Tests with a Tuning Fork to Distinguish between Sensorineural and Conduction Deafness.**

	<b>Weber</b>	<b>Rinne</b>	
Method	Base of vibrating tuning fork placed on vertex of skull.	Base of vibrating tuning fork placed on mastoid process until subject no longer hears it, then held in air next to ear.	
Normal	Hears equally on both sides.	Hears vibration in air after bone conduction is over.	Positive Rinne
Conduction deafness (one ear)	Sound louder in diseased ear because masking effect of environmental noise is absent on diseased side.	Vibrations in air not heard after bone conduction is over.	Negative Rinne
Sensorineural deafness (one ear)	Sound louder in normal ear.	Vibration heard in air after bone conduction is over, as long as nerve deafness is partial.	

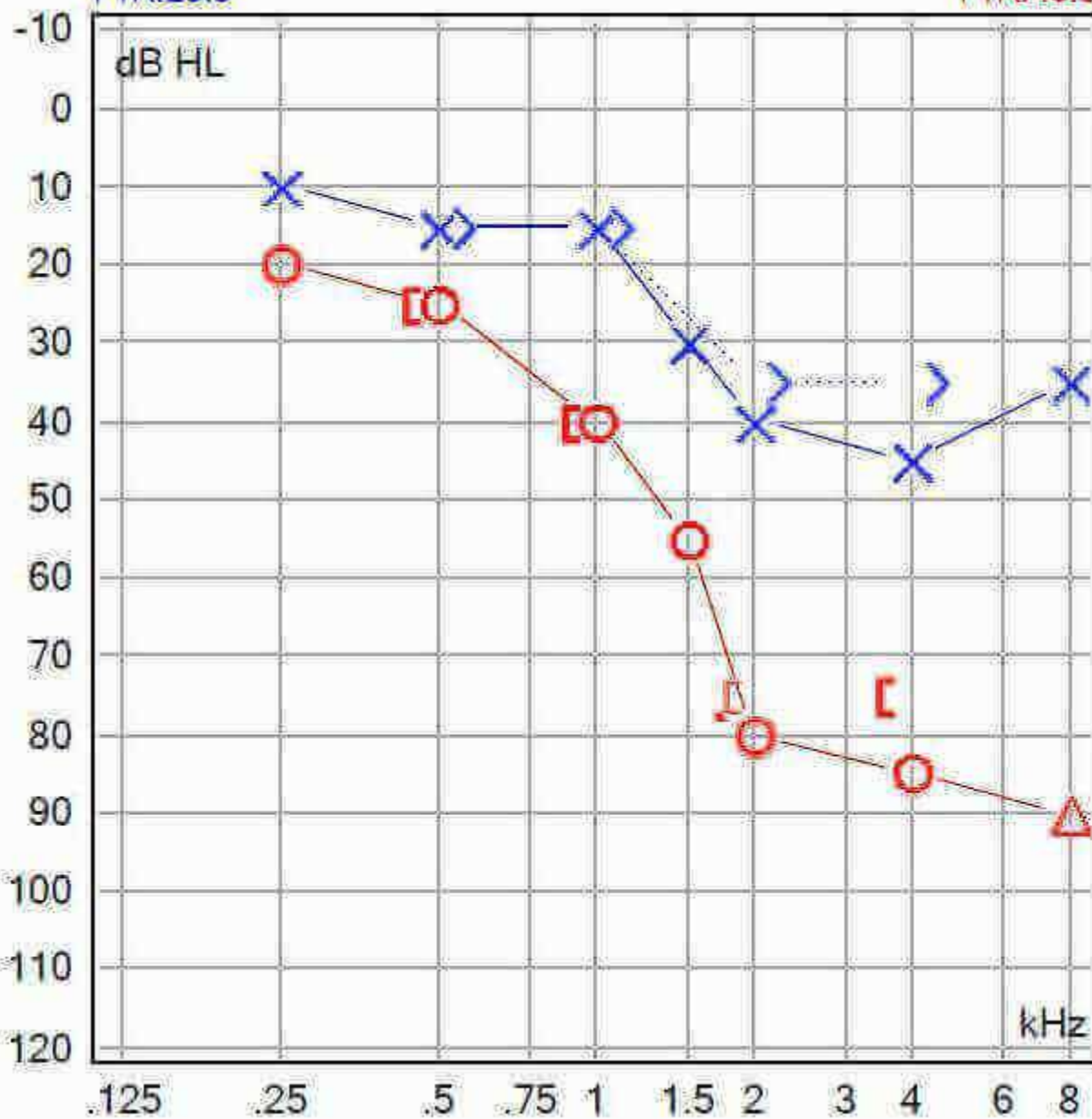
# Frequency (Hz)



# ANSI S3.6 2004

PTA:23.3

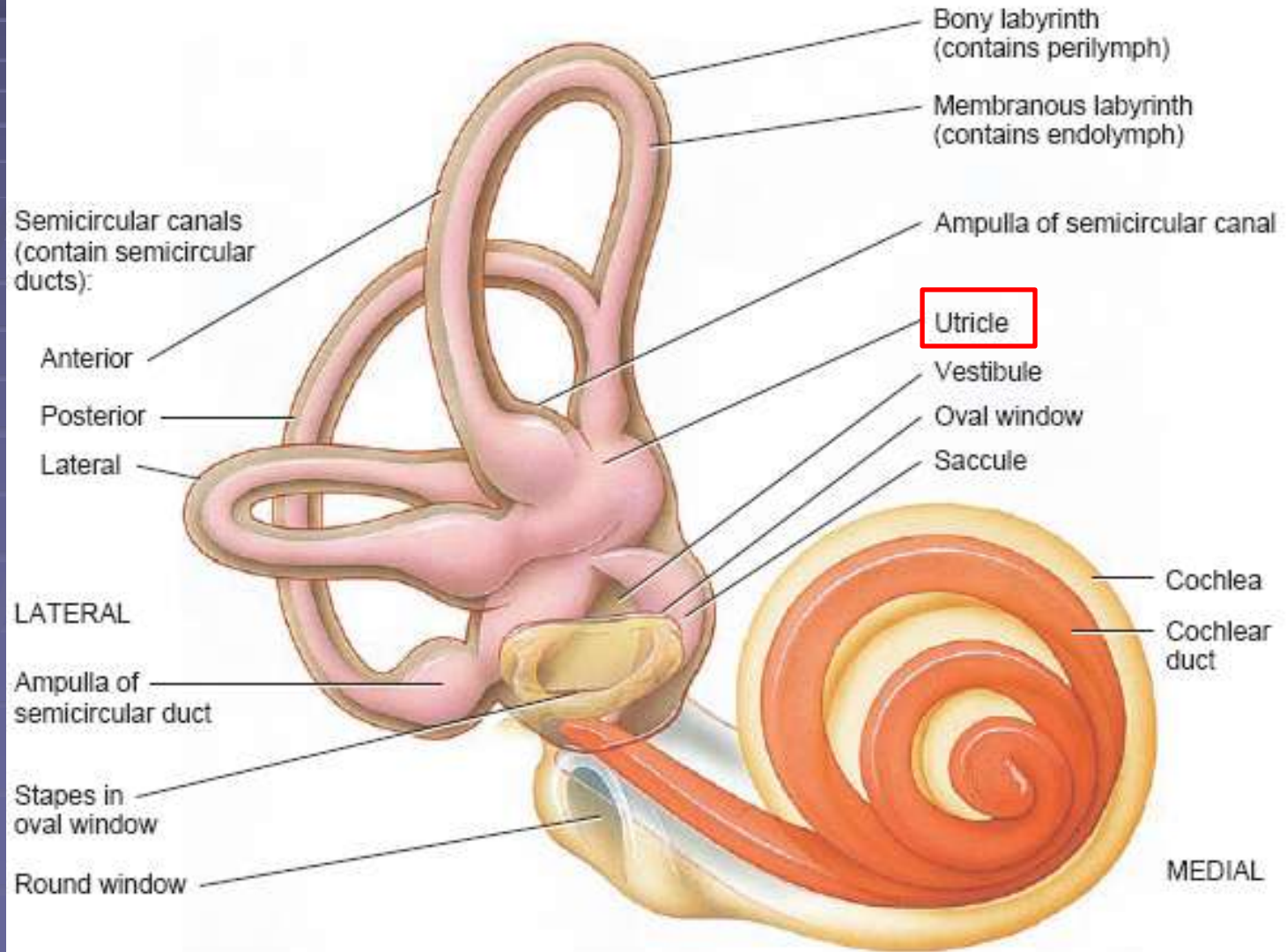
PTA:48.3



# The Vestibular System

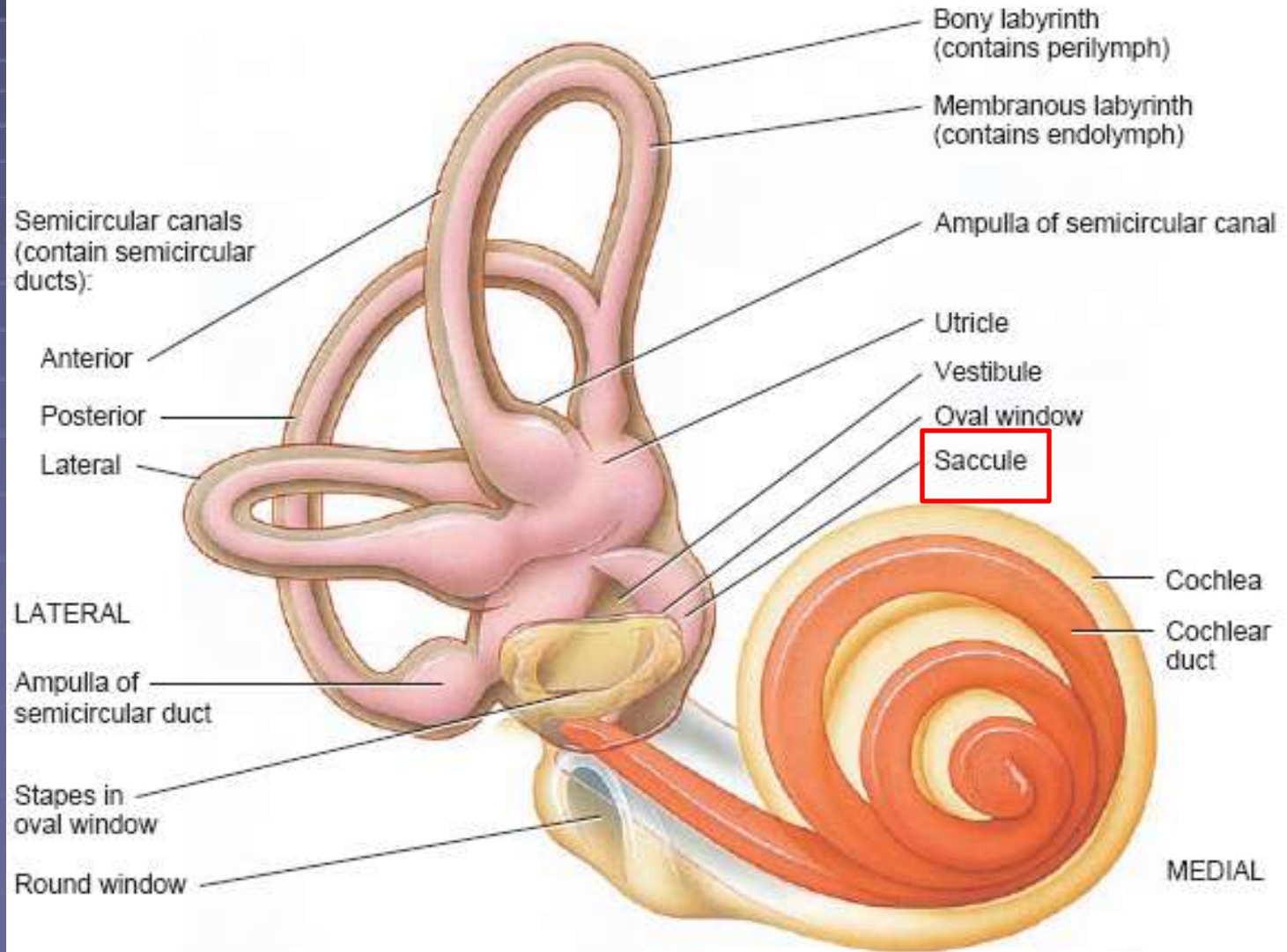
# Anatomy of the ear

Internal ear



# Anatomy of the ear

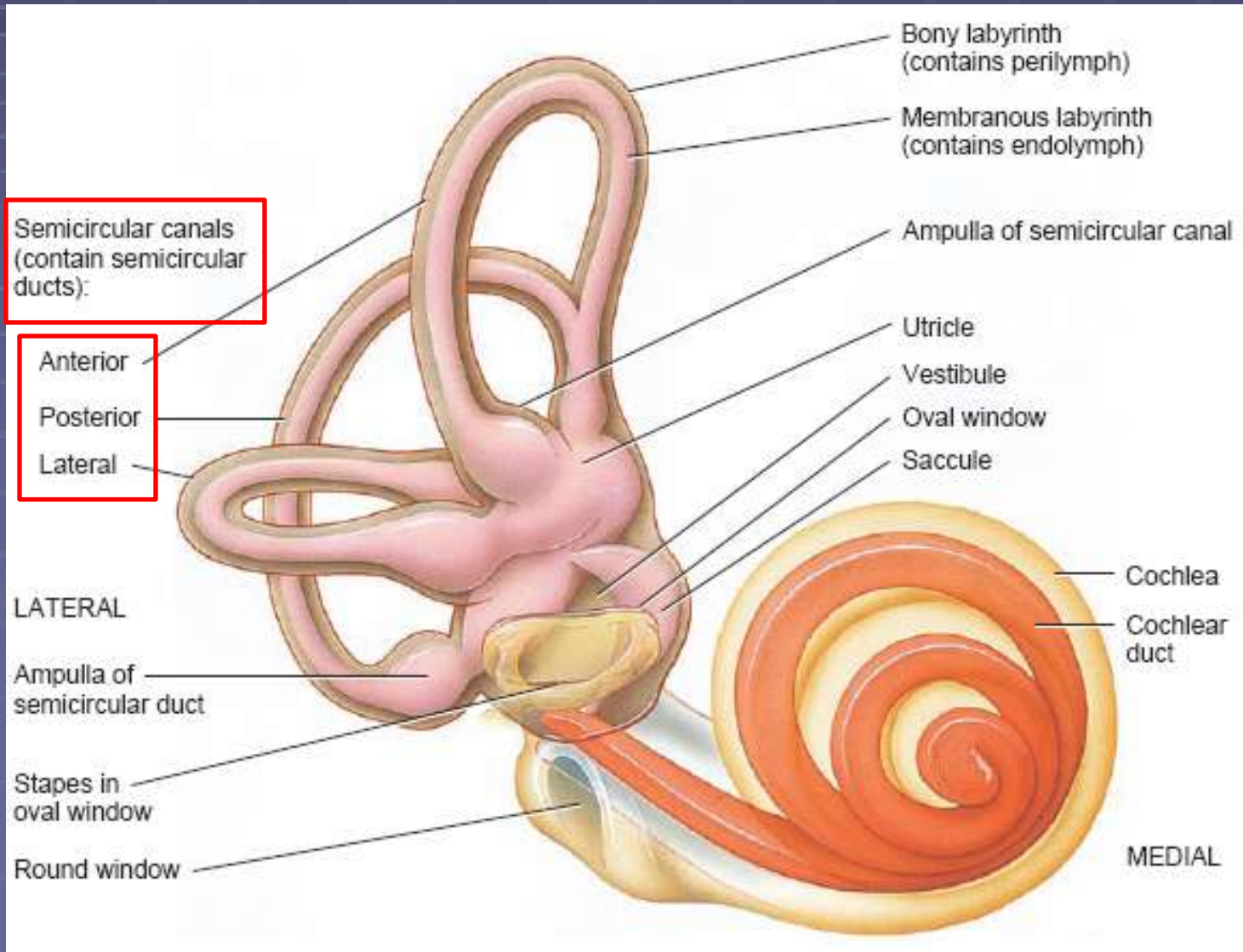
Internal ear



# Anatomy of the ear



Internal ear

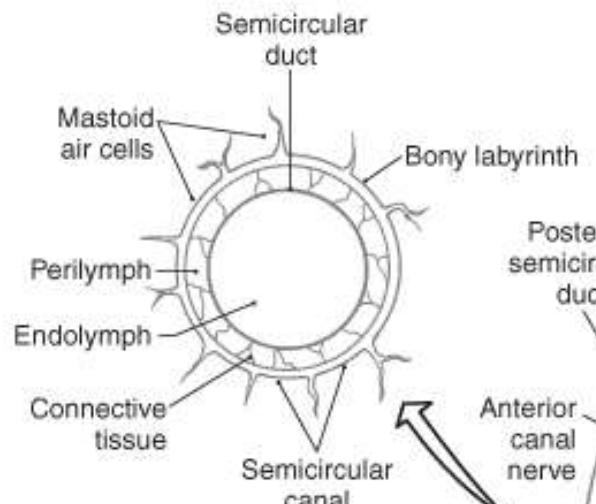
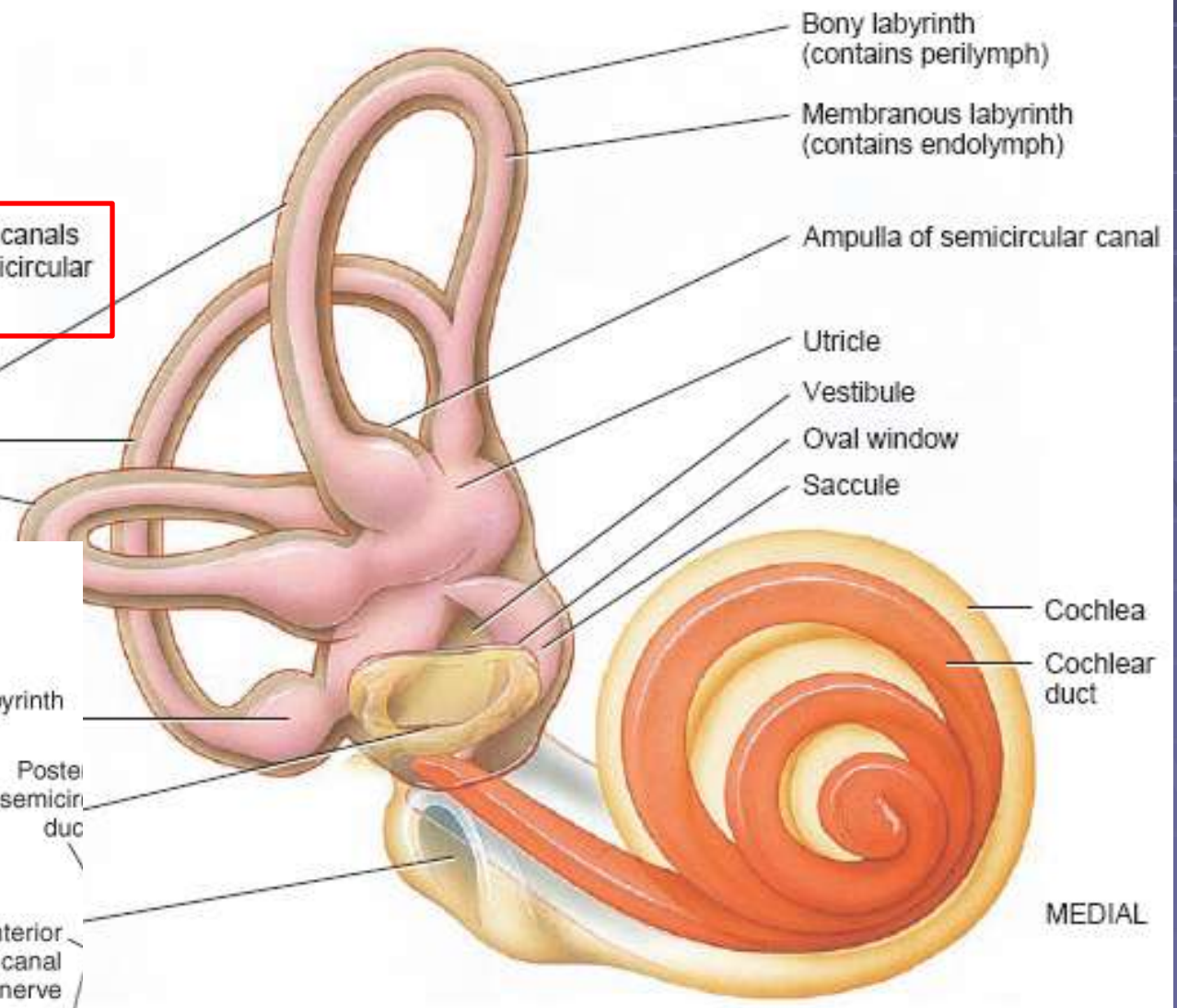


# Anatomy of the ear

Internal ear

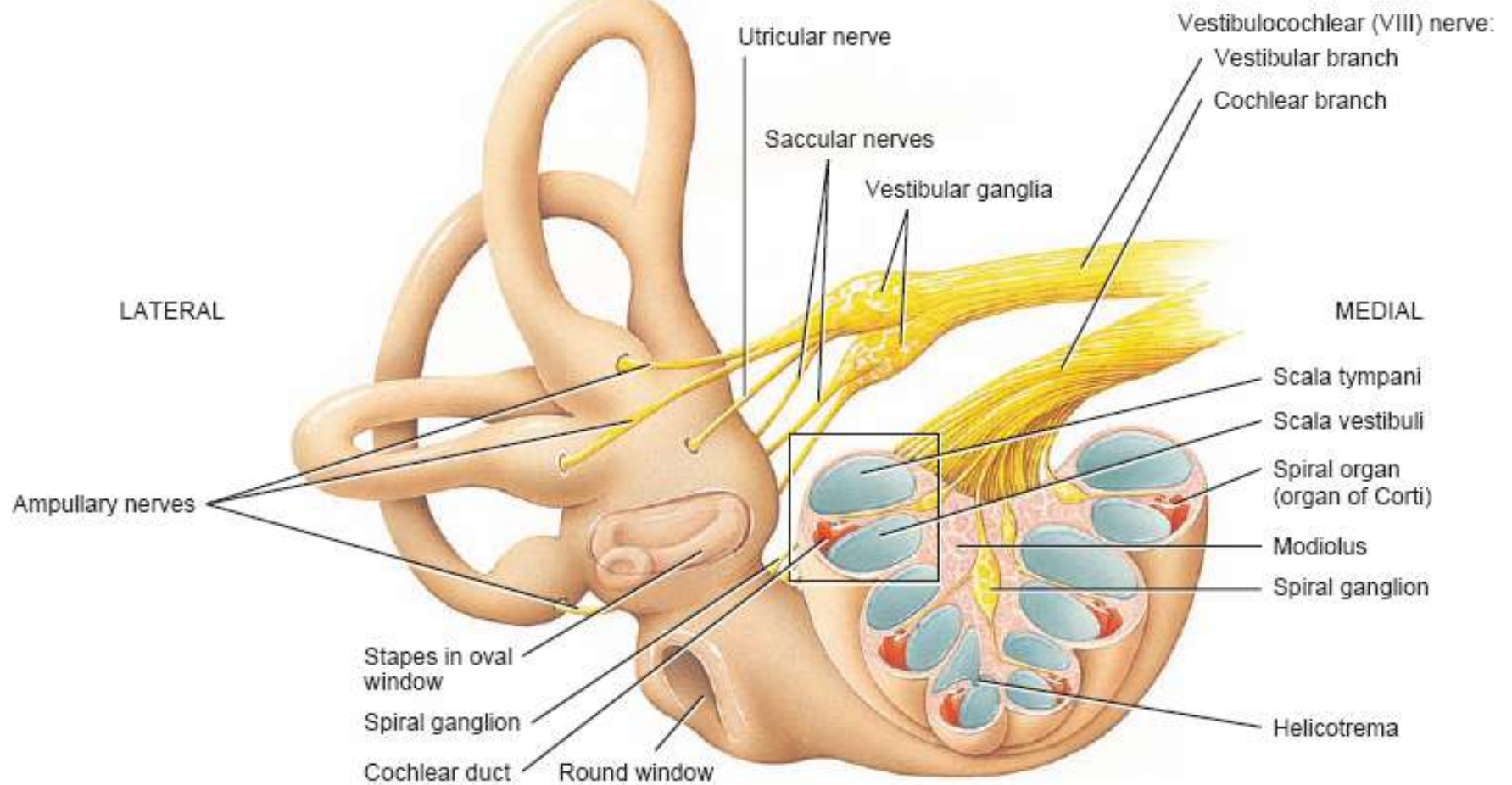
Semicircular canals (contain semicircular ducts):

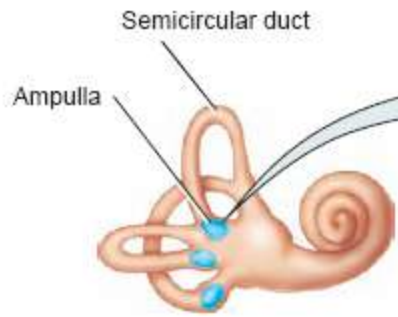
- Anterior
- Posterior
- Lateral



MEDIAL

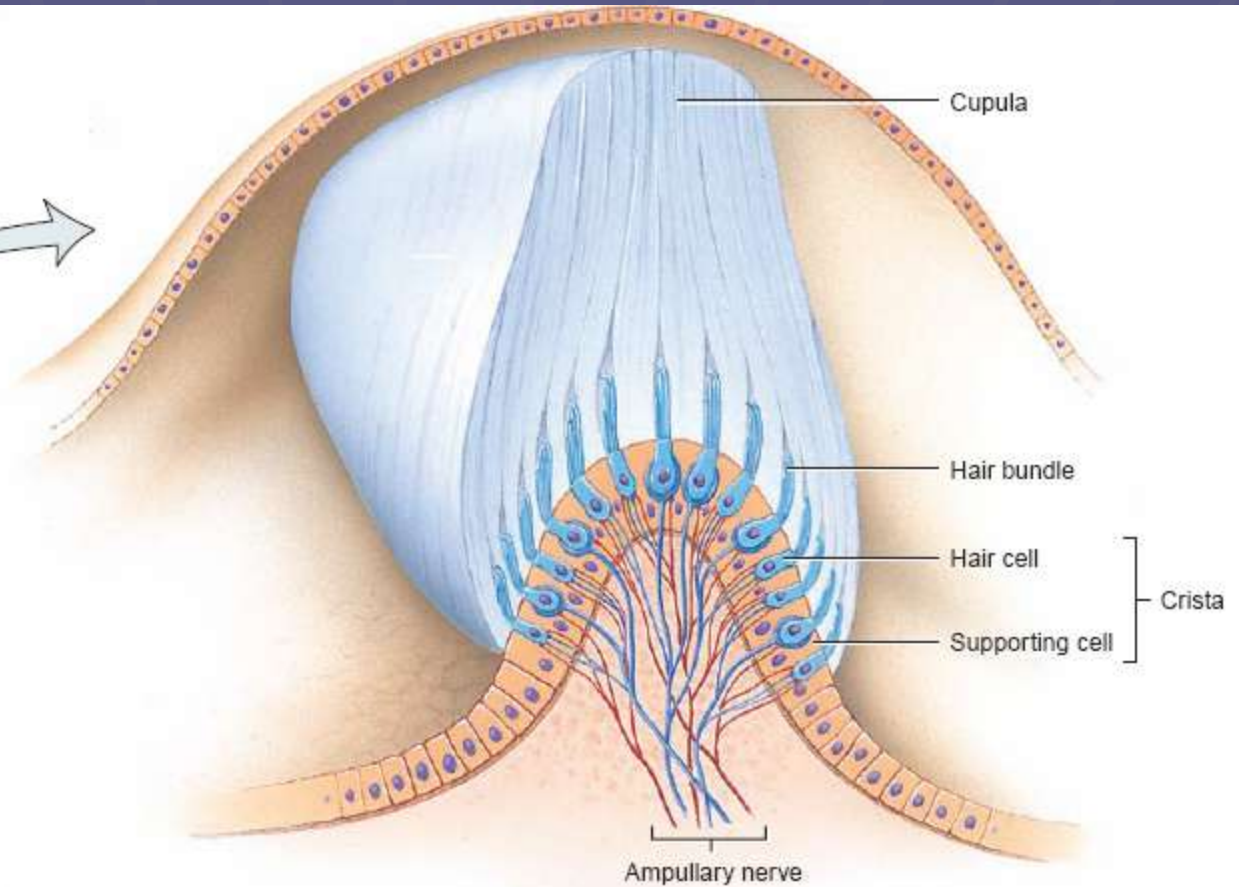






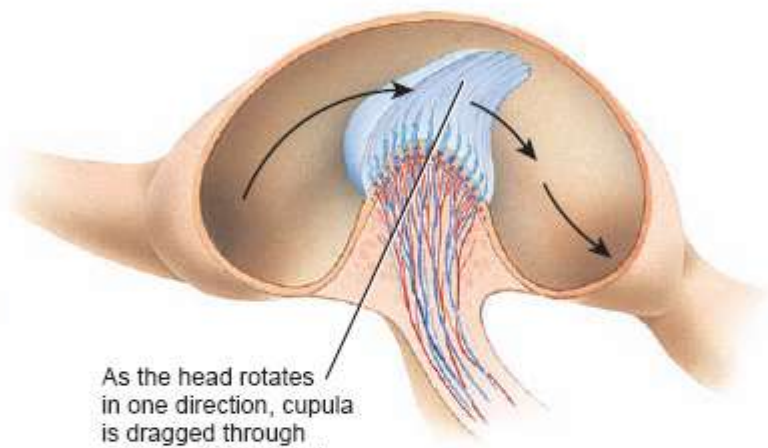
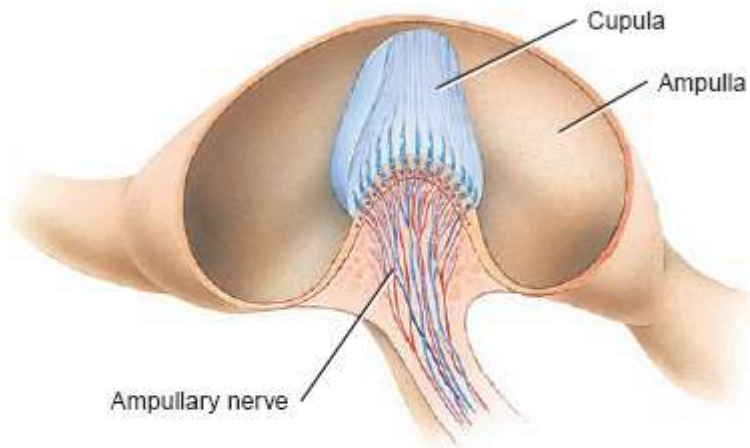
Location of ampullae of semicircular ducts (contain cristae)

**Key:**  
Sensory fiber  
Motor fiber



(a) Details of a crista

# Ampulla of Semicircular canal



(b) Position of a cupula with the head in the still position (left) and when the head rotates (right)

# Hair Cell Activation

Rotational head movements



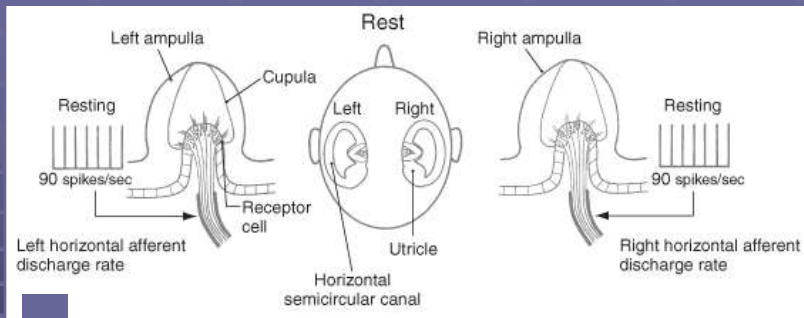
Angular accelerations



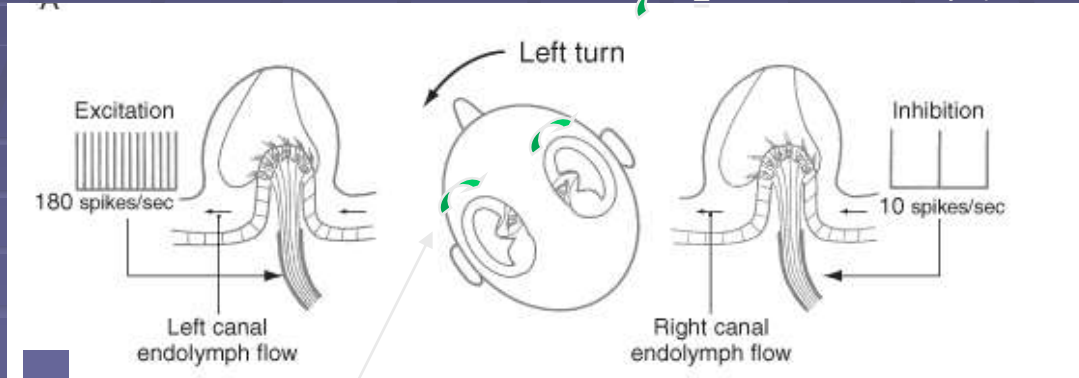
Displace endolymph in membranous ducts

Push cupula to one side or other

Displace stereocilia/kinocilium of  
hair cells in same direction

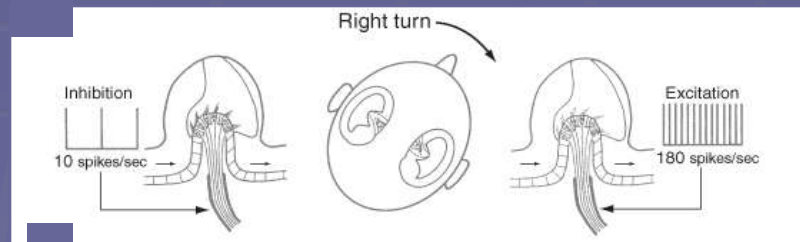


— Movement of endolymph



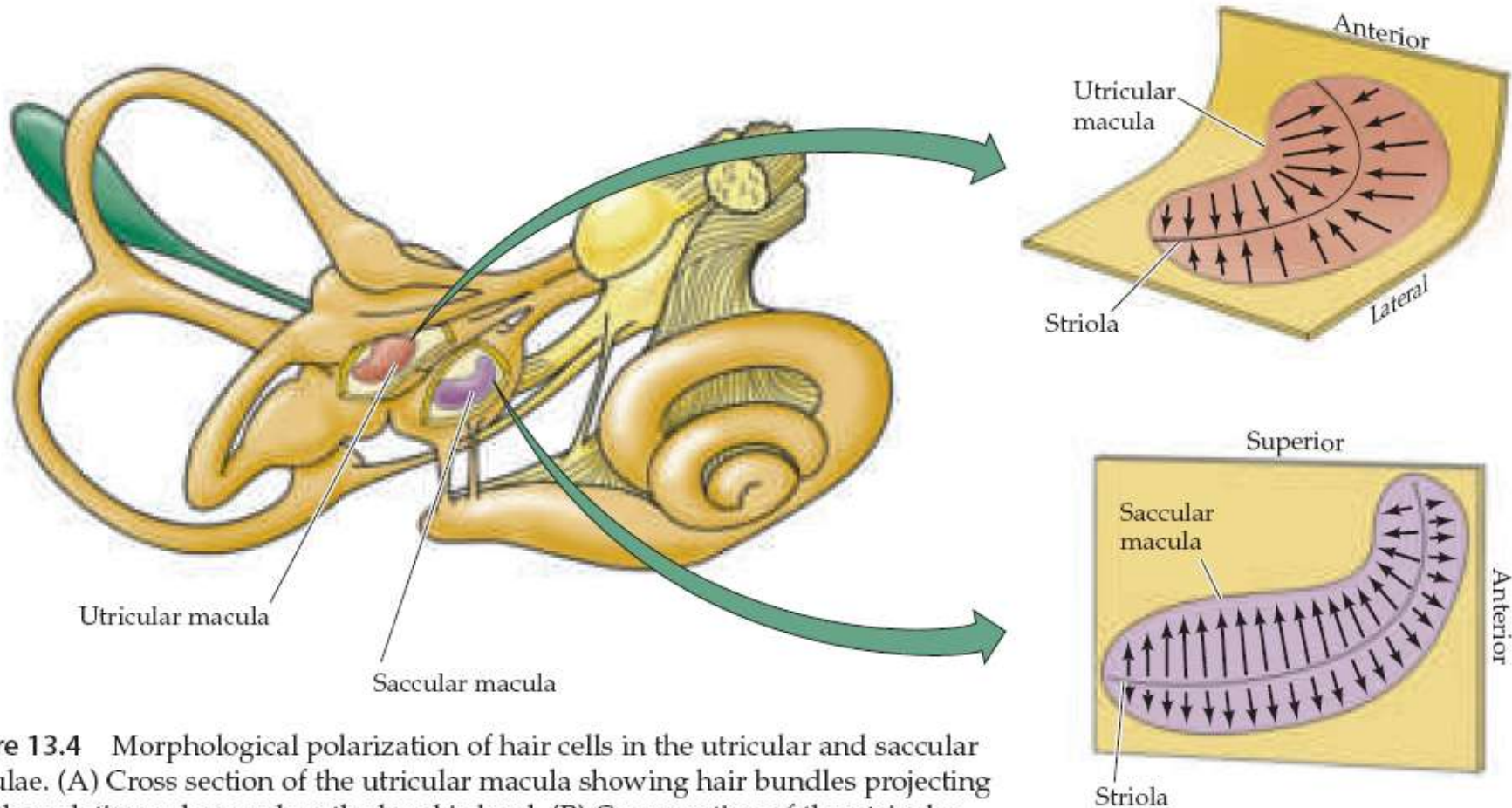
Movement of endolymph toward ampulla

Movement of endolymph away from ampulla



# Macula and otolith organ

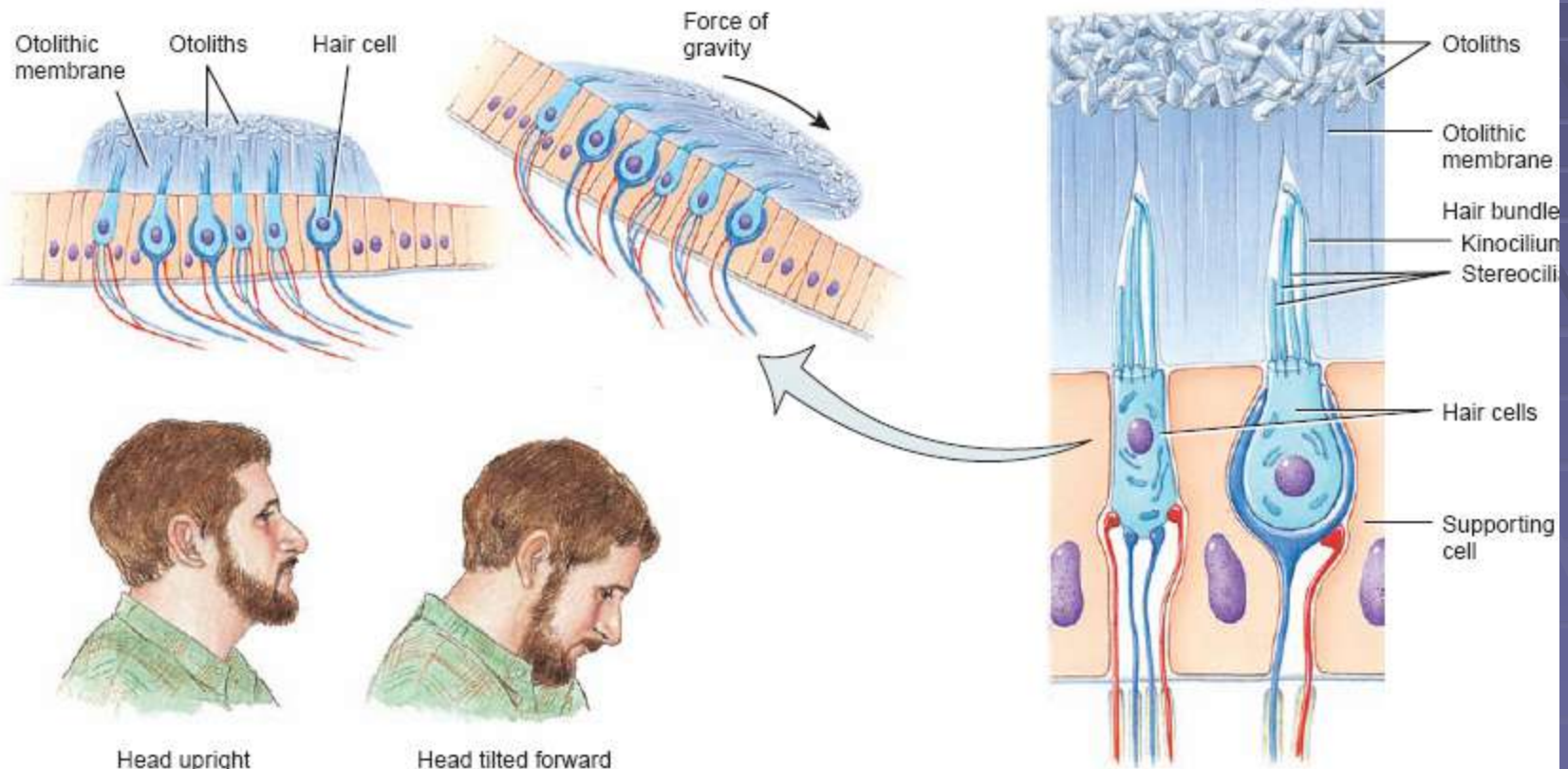
(C)



**Figure 13.4** 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 and otolith organ

(a) Overall structure of a section of the macula

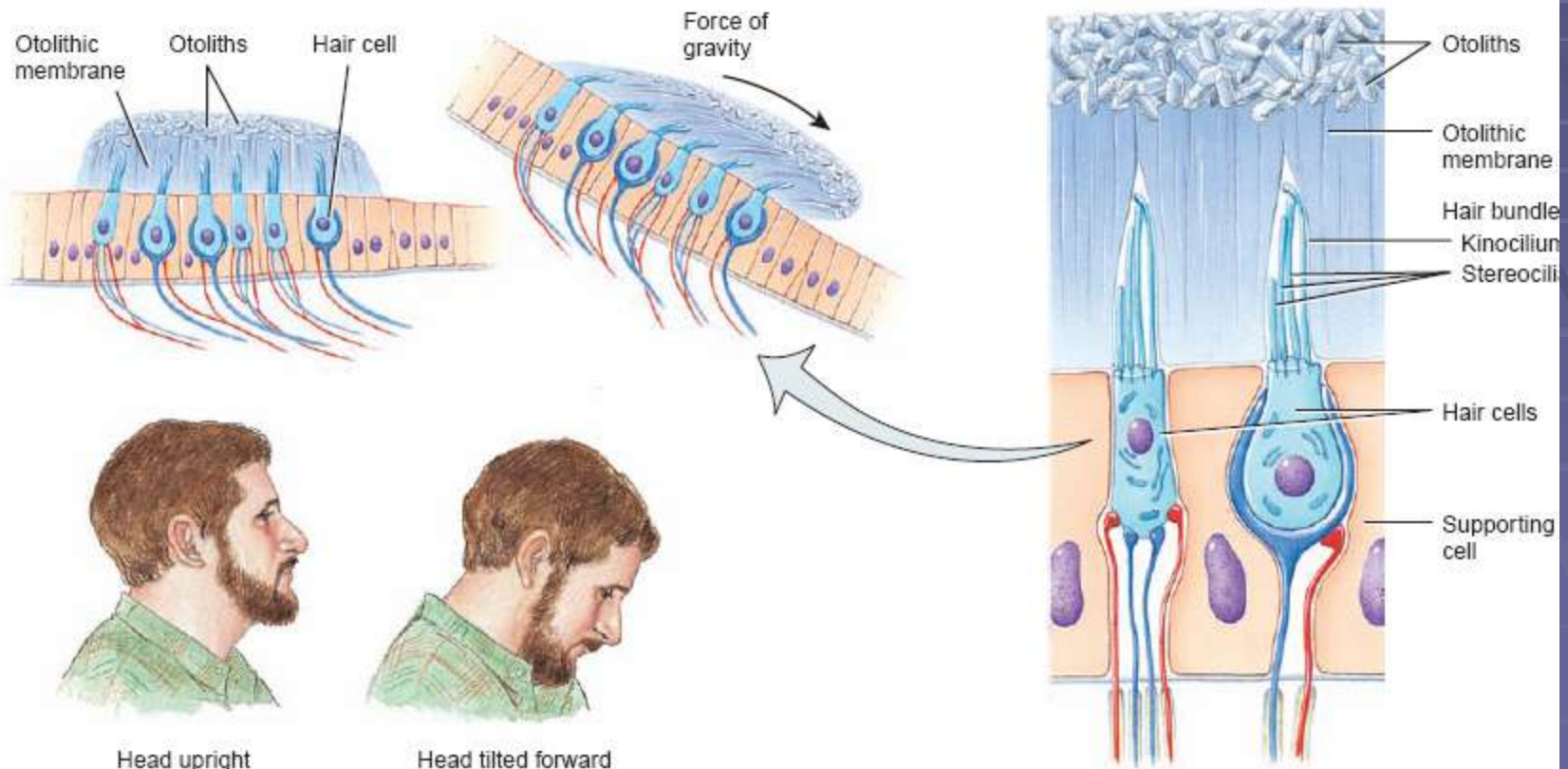


(c) Position of macula with head upright (left) and tilted forward (right)

(b) Details of two hair cells

# Macula and otolith organ

(a) Overall structure of a section of the macula



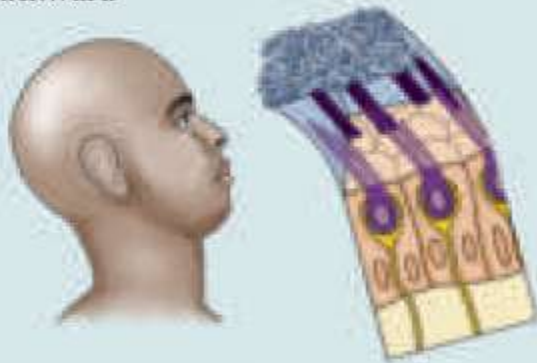
(c) Position of macula with head upright (left) and tilted forward (right)

(b) Details of two hair cells

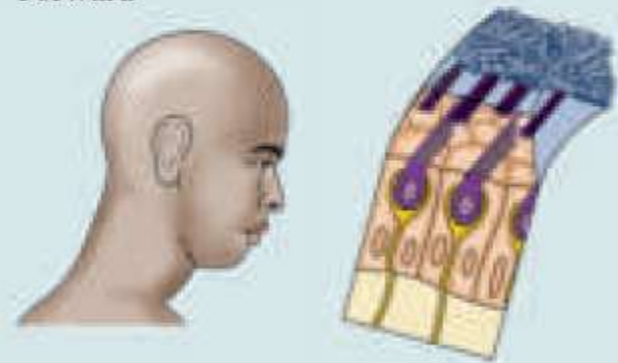


**Head tilt; sustained**

Backward

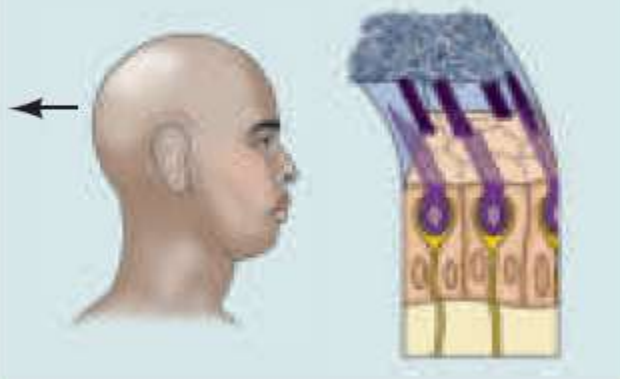


Forward

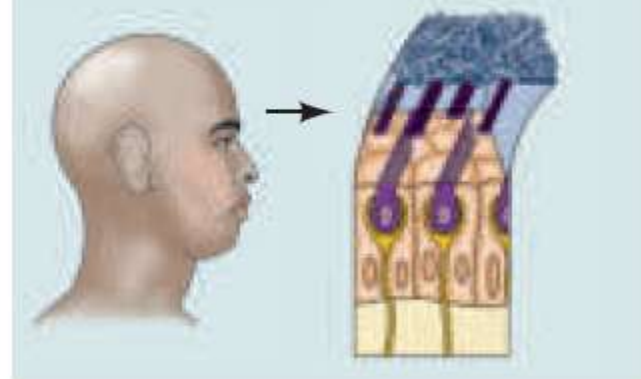


**No head tilt; transient**

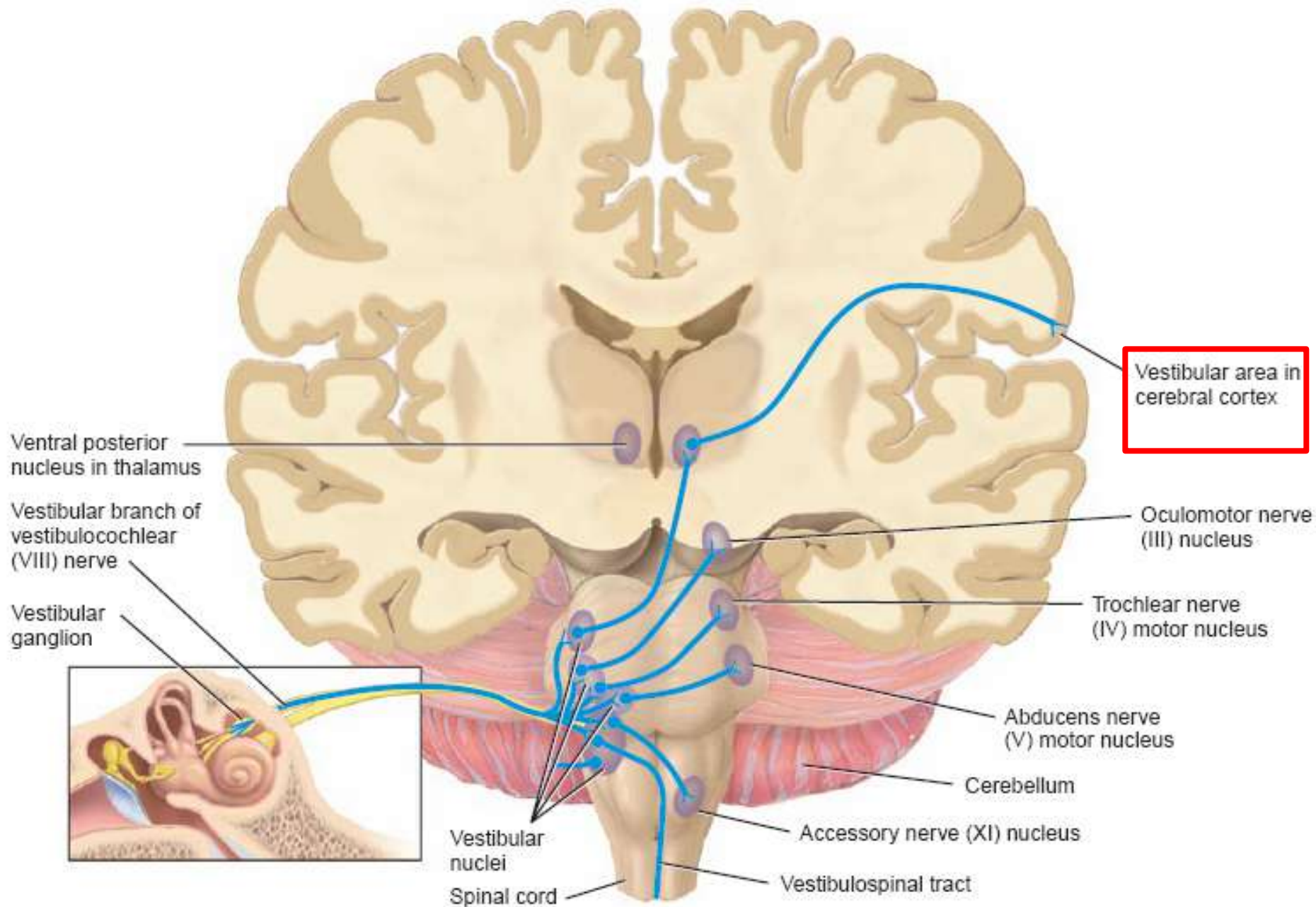
Forward acceleration

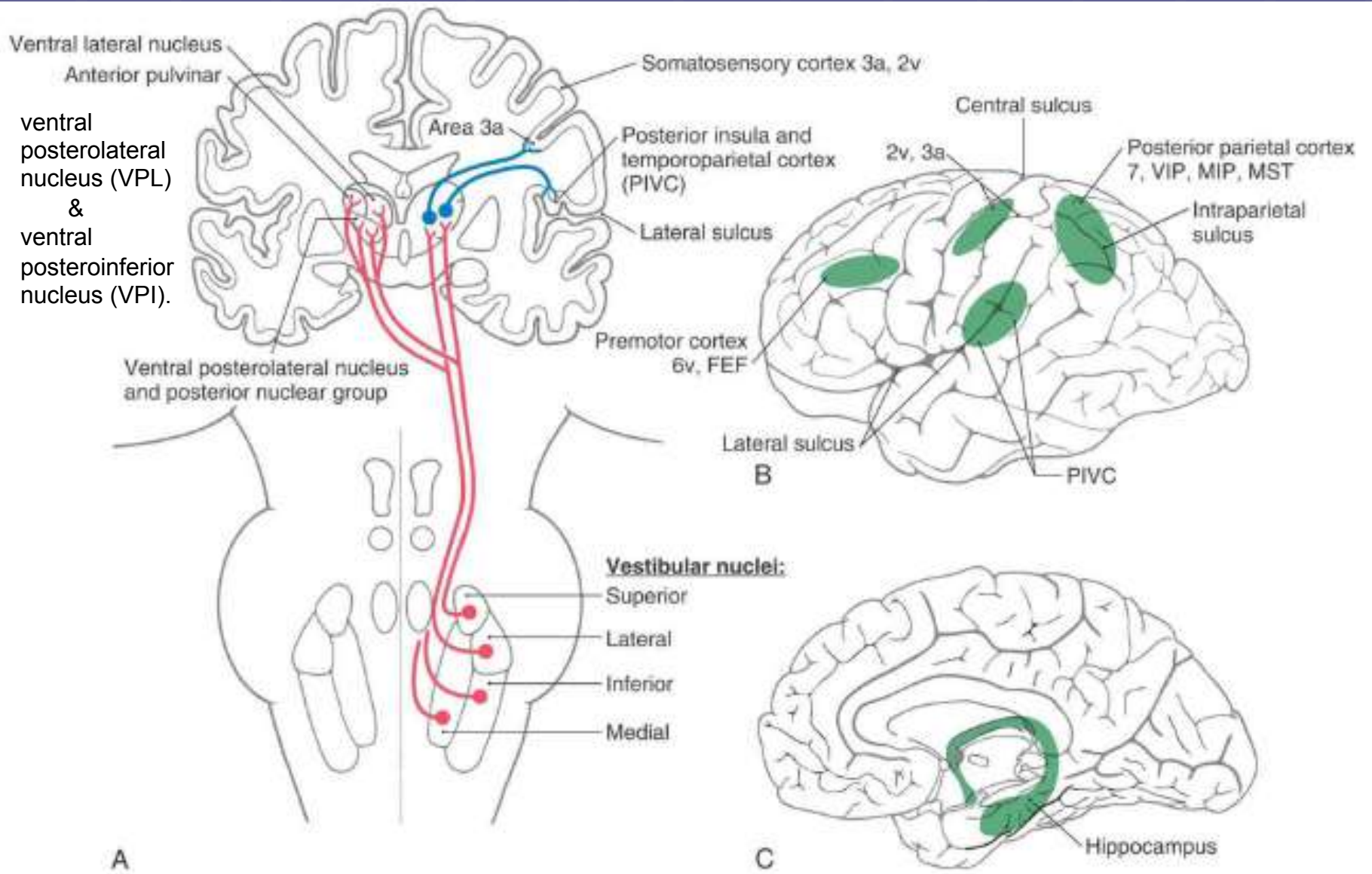


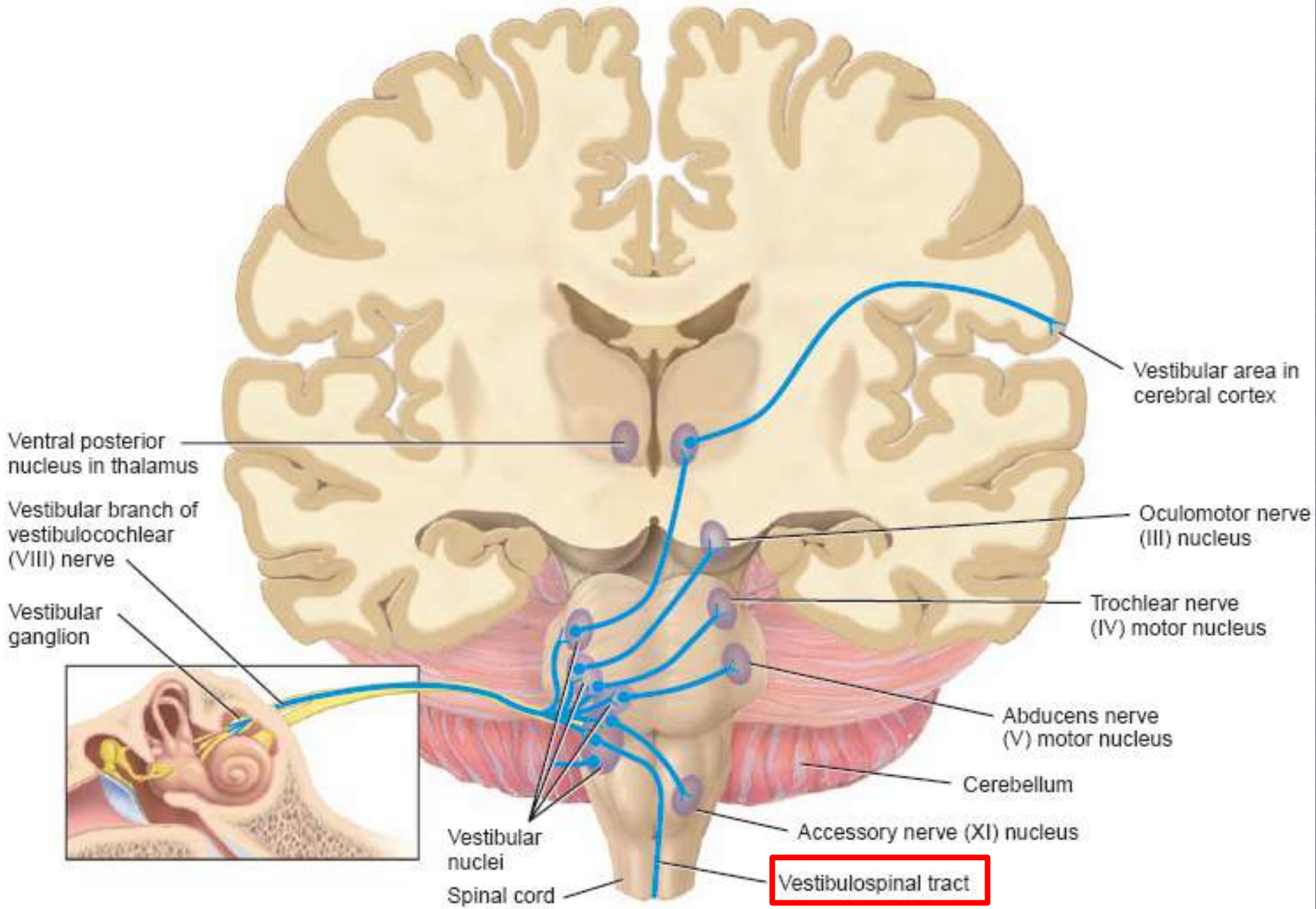
Deceleration

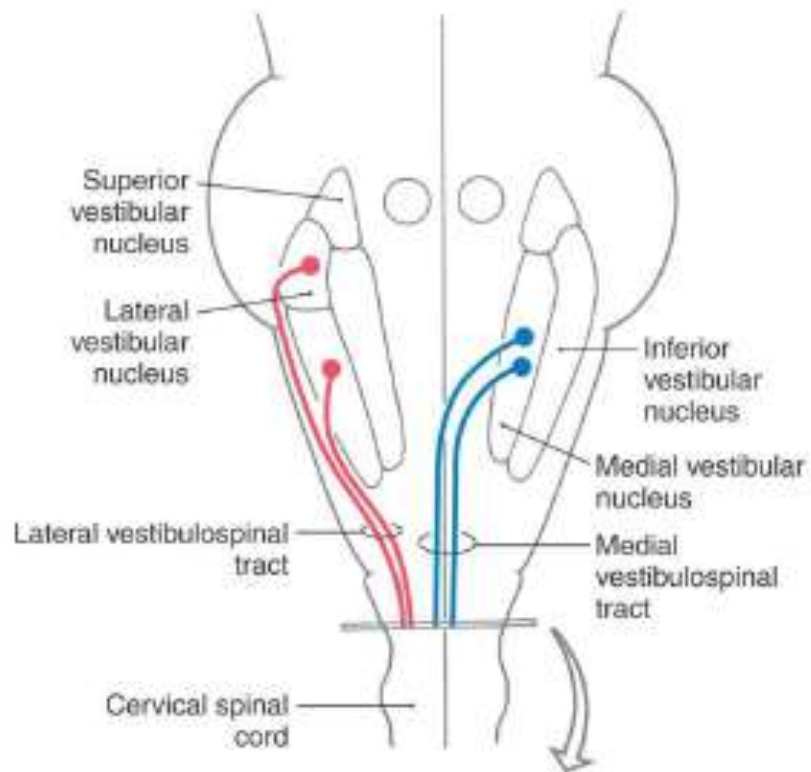


# VESTIBULAR PATHWAY

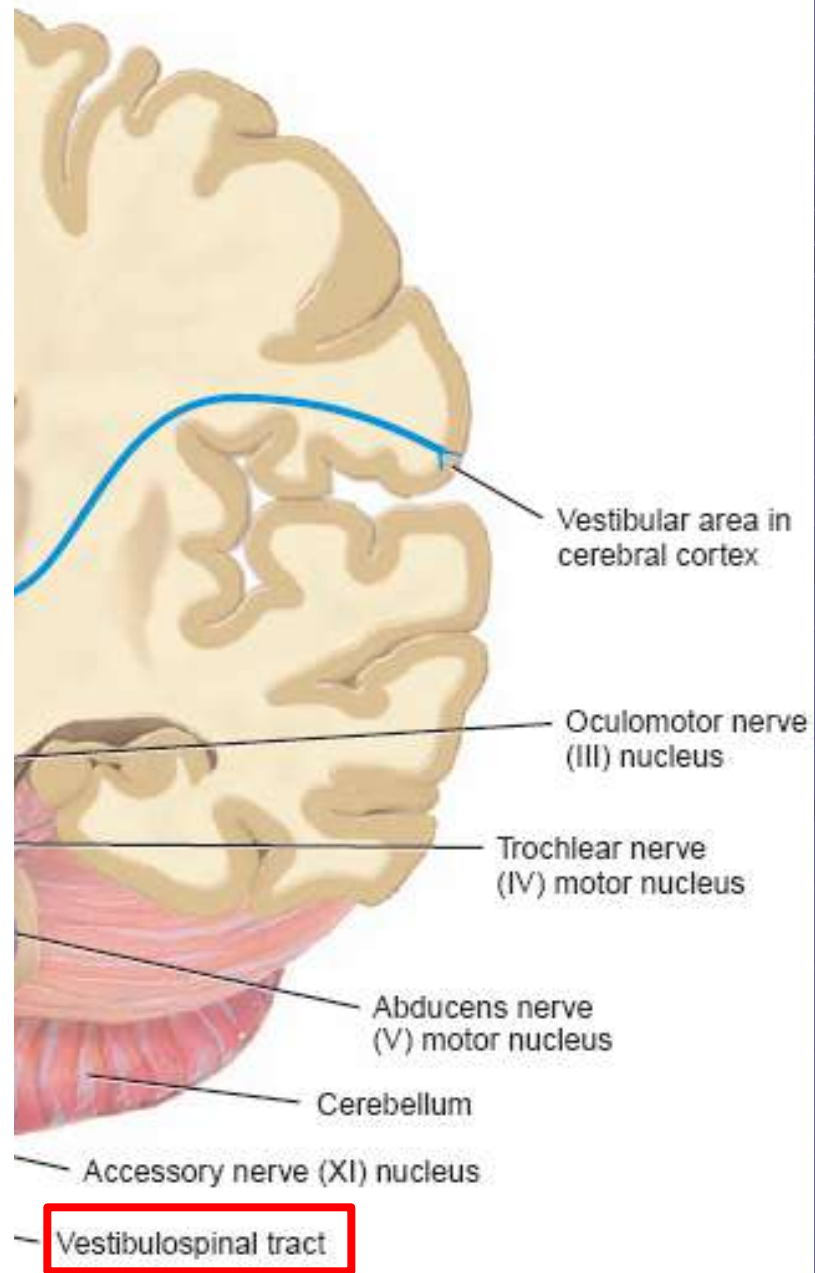
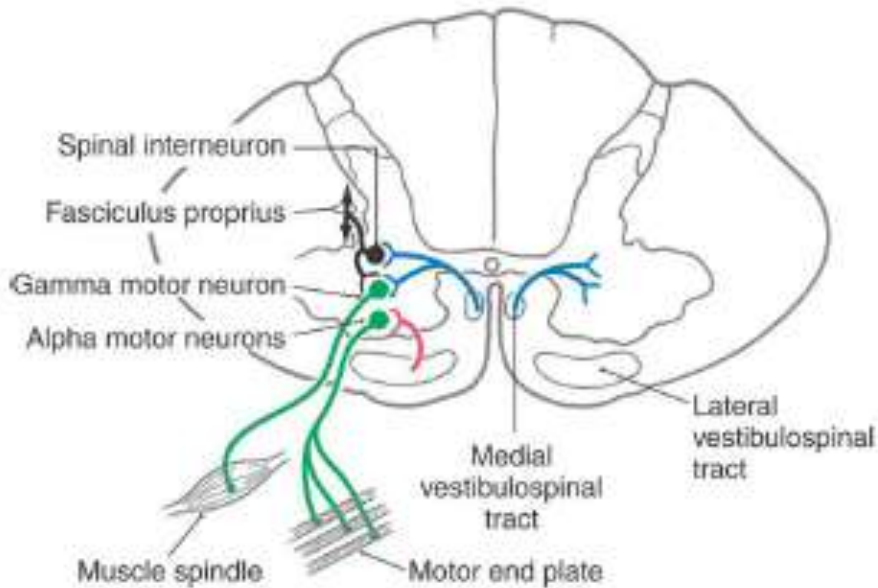








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# Vestibulospinal Network:

Influences muscle tone & produces reflexive postural adjustments of the head and body

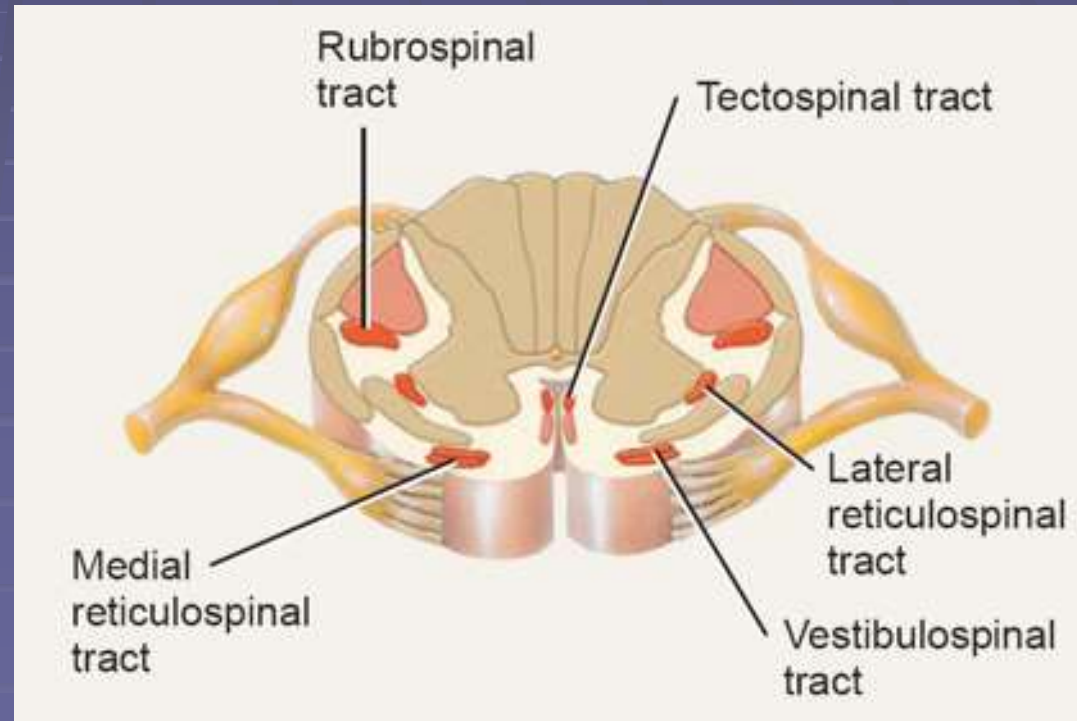
vestibular nucleus

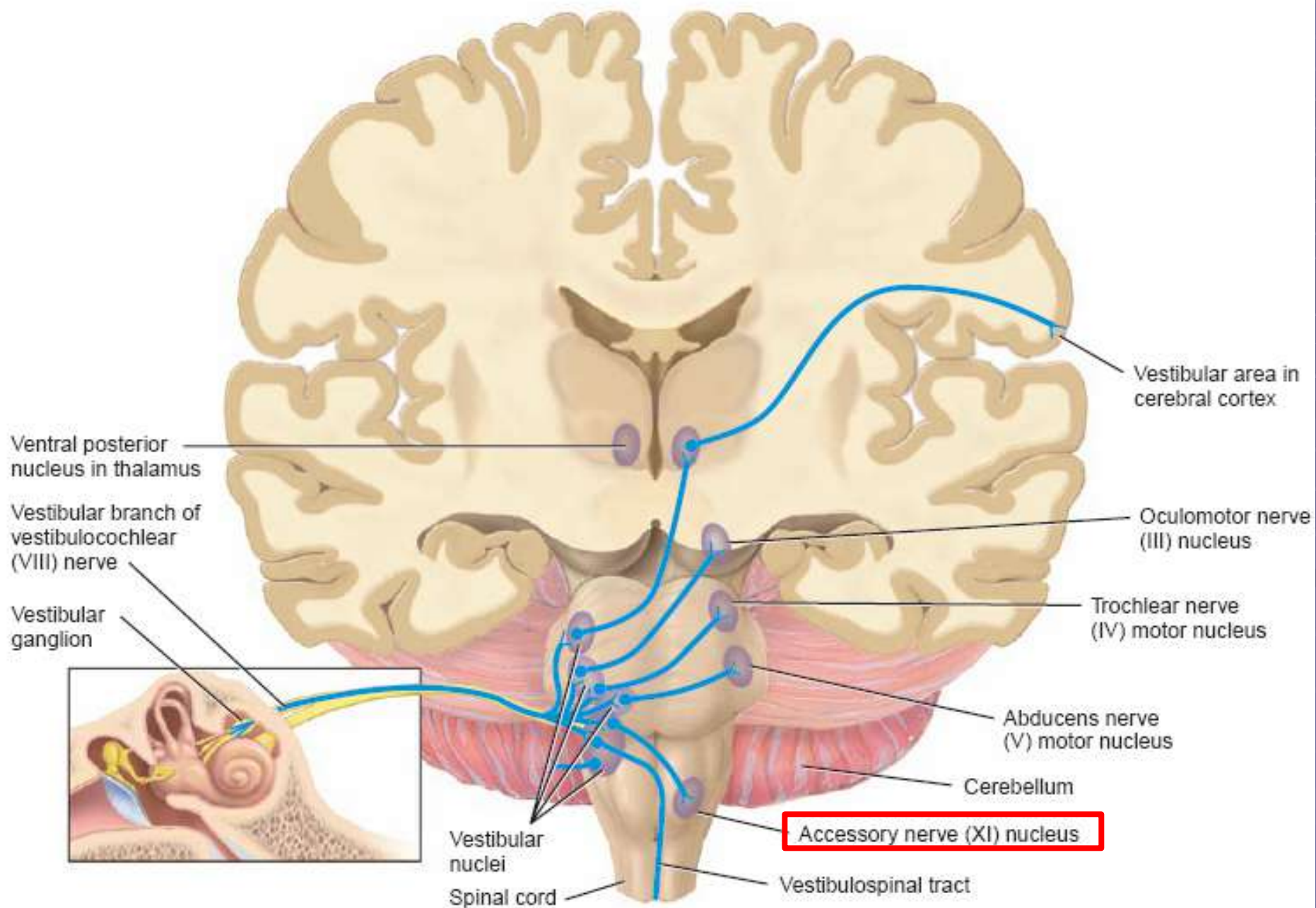
Projects to all levels of the spinal cord

Terminate on alpha & gamma motor neurons

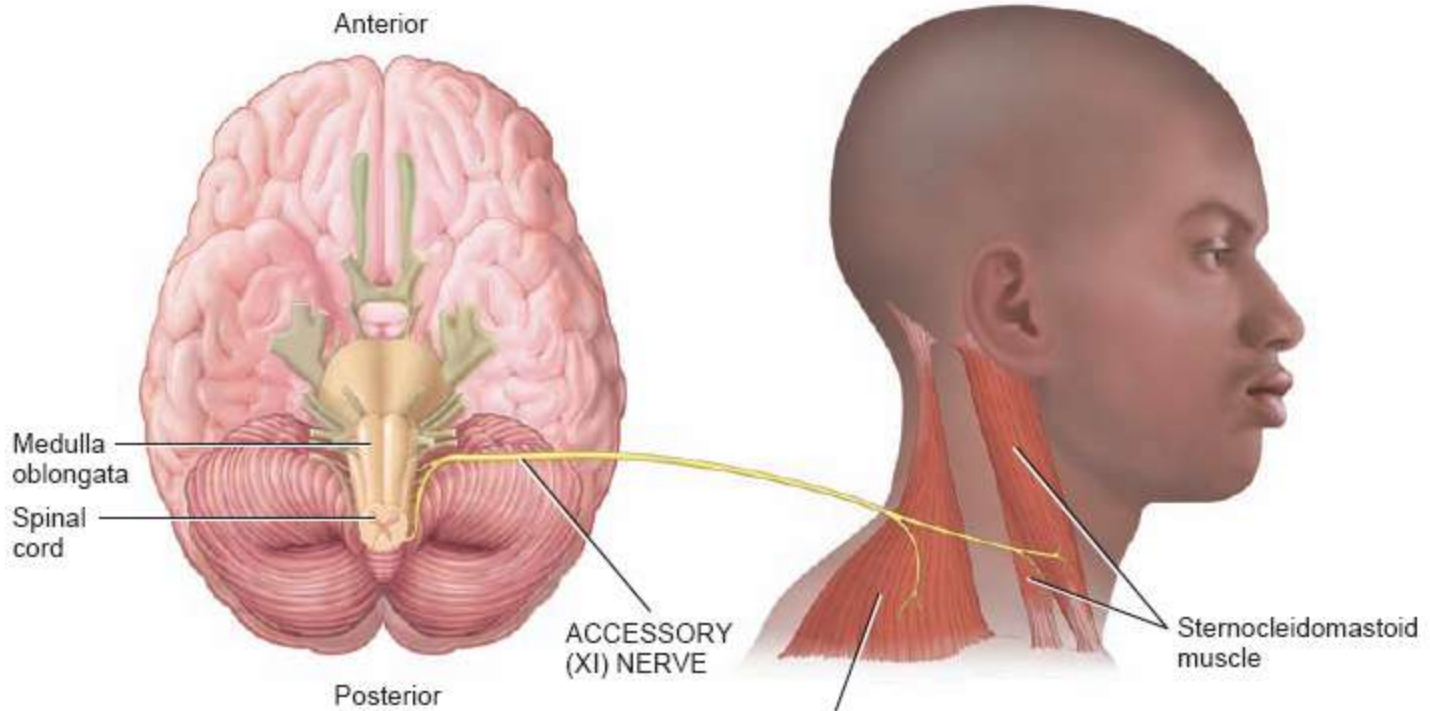
Excite extensor muscle motor neurons

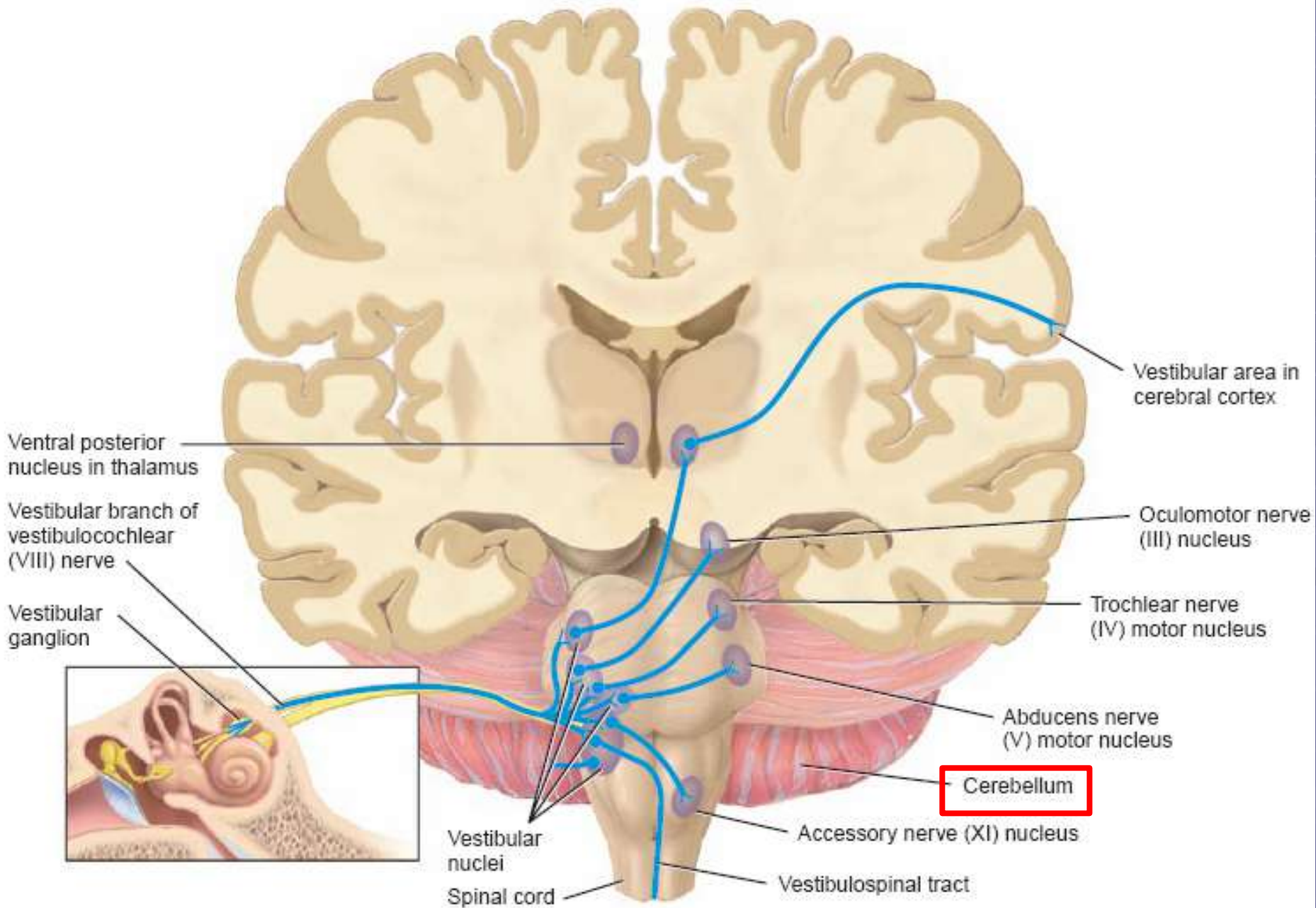
Stabilize body's center of gravity & preserves upright posture

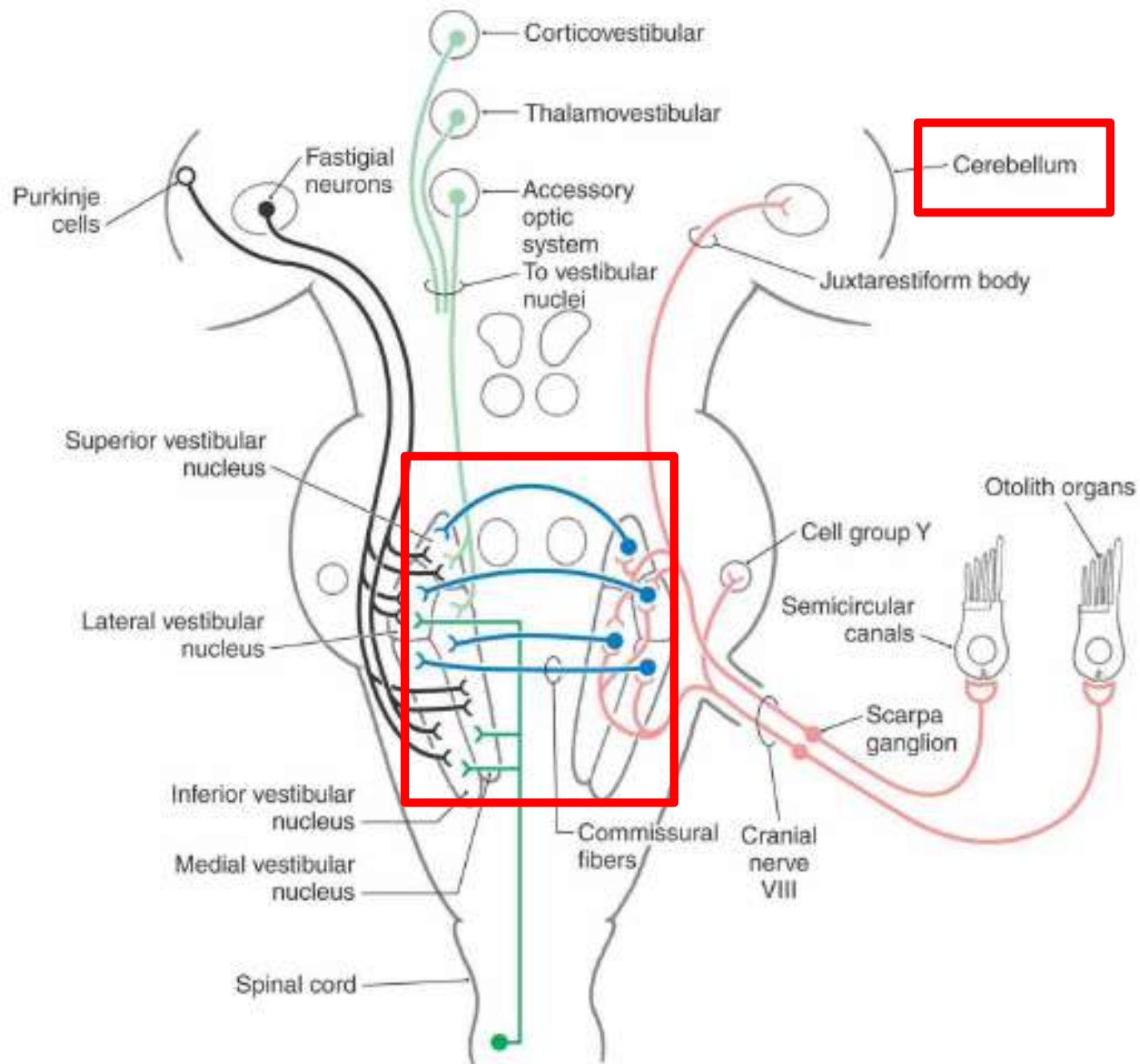


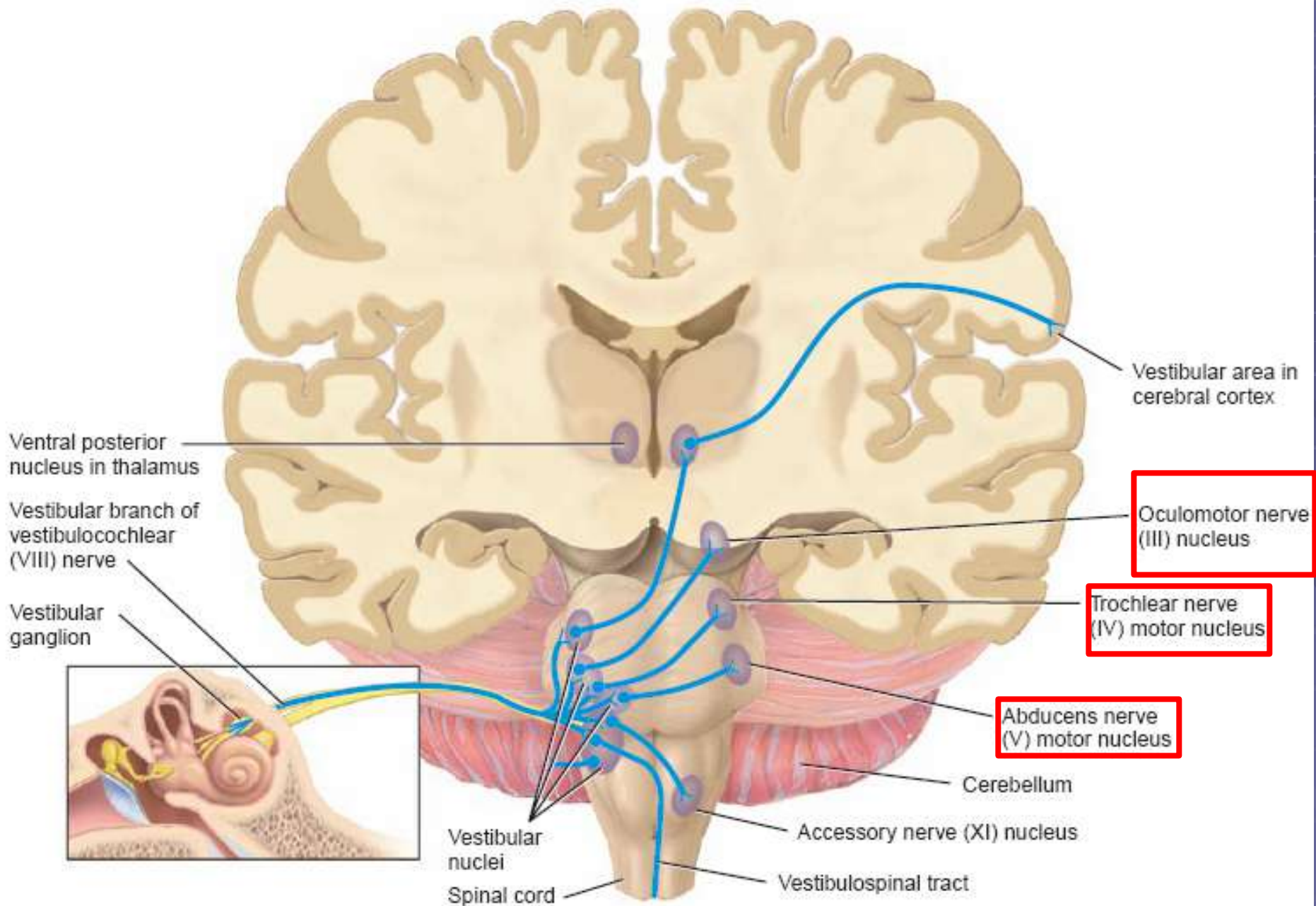


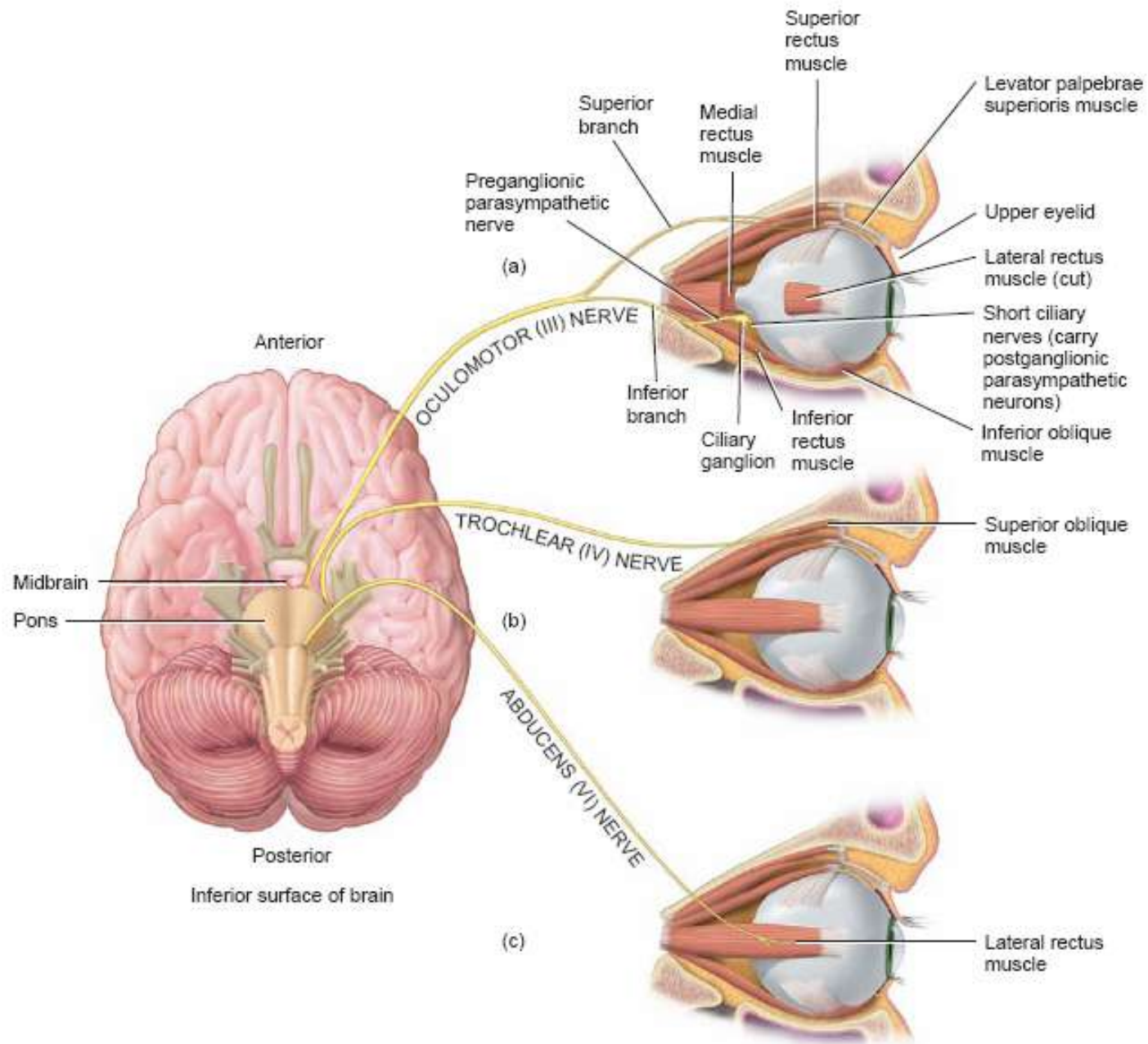


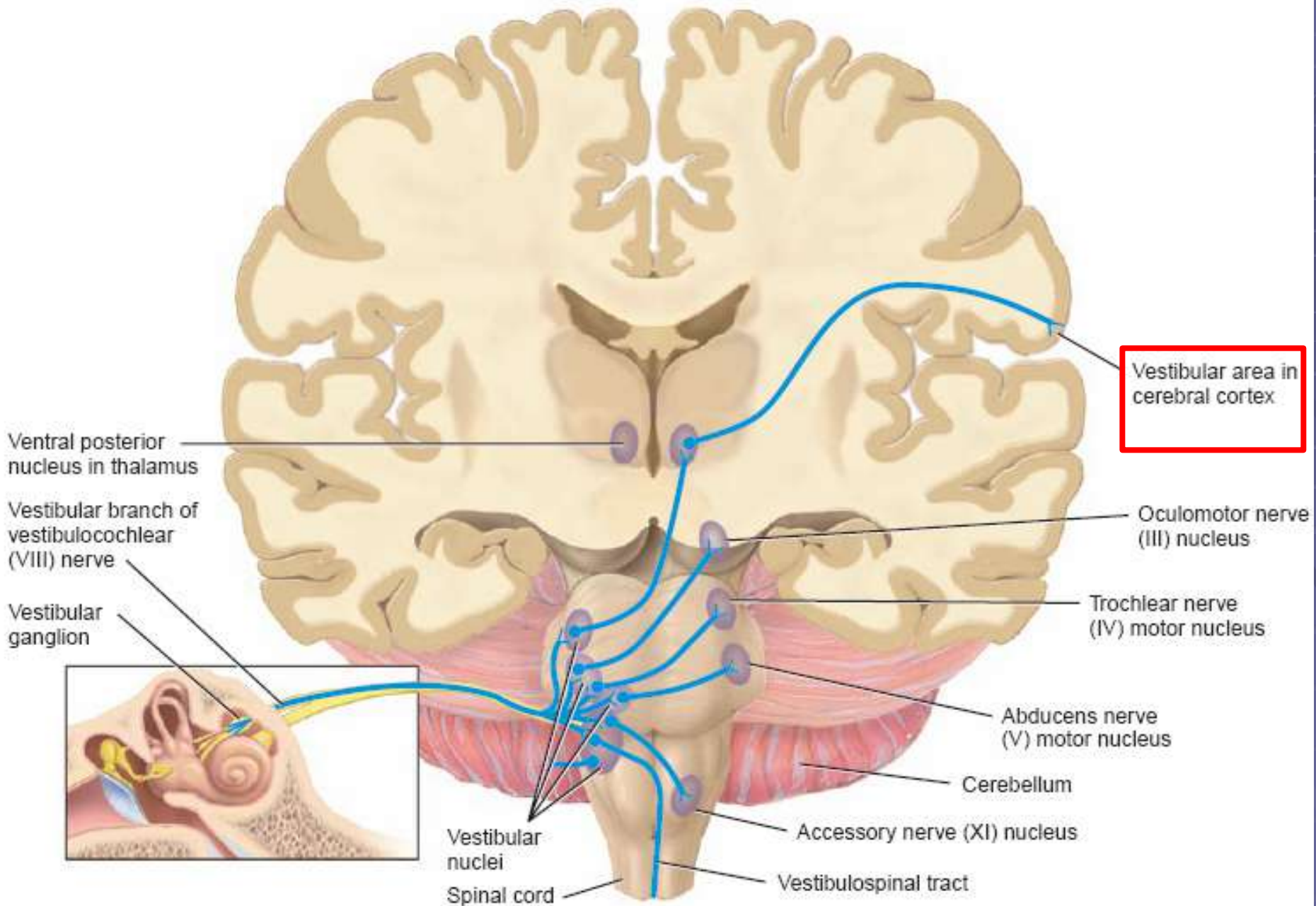


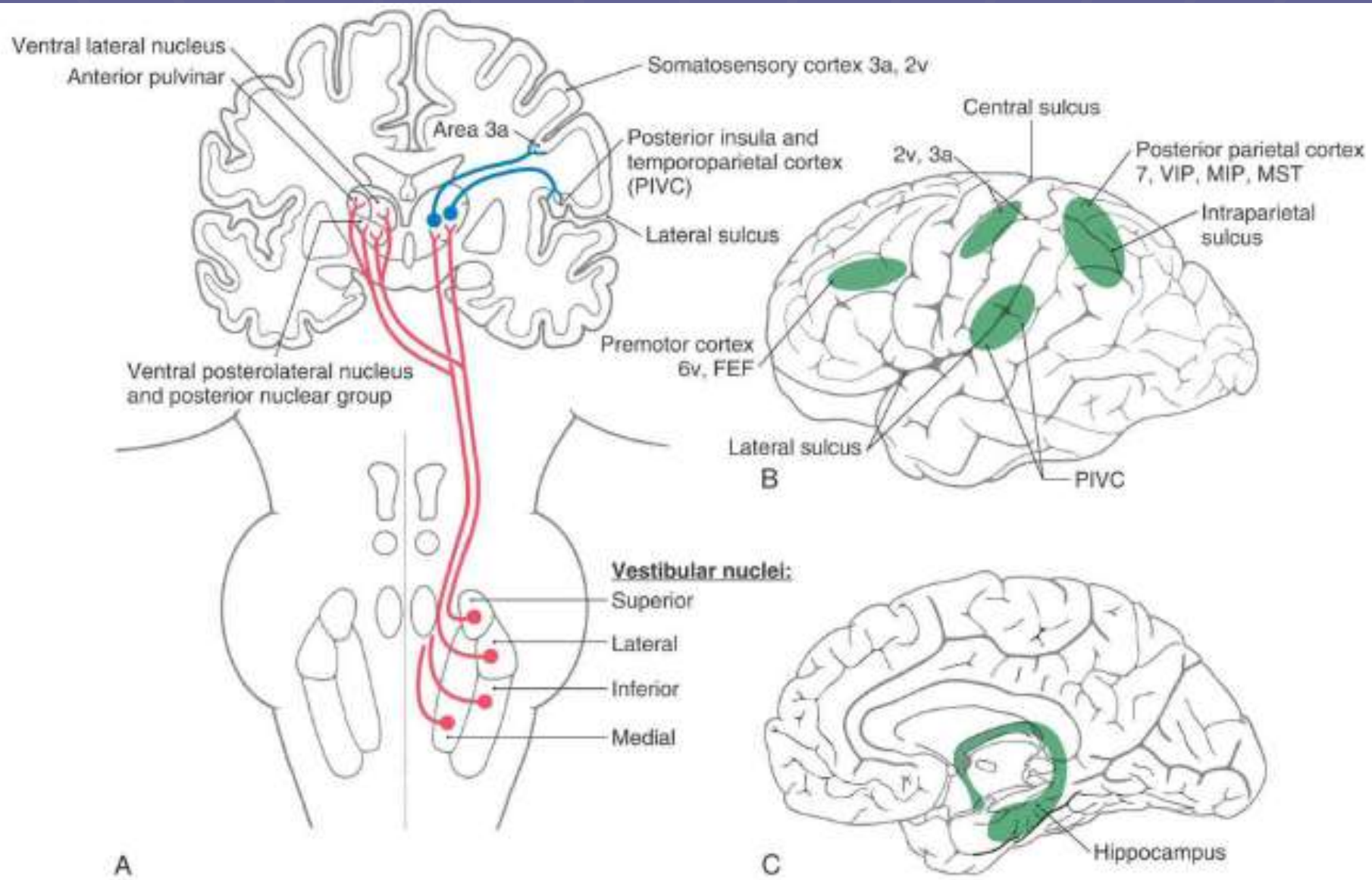








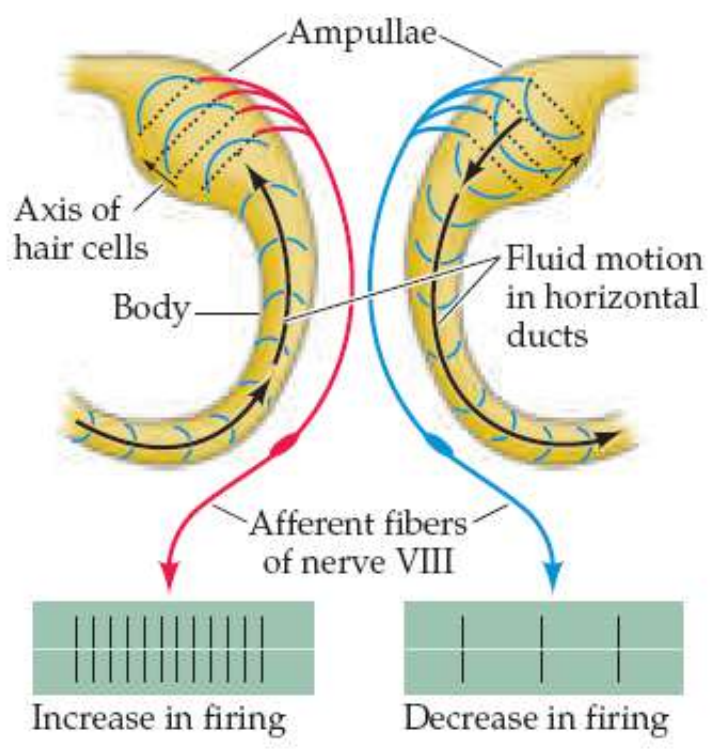
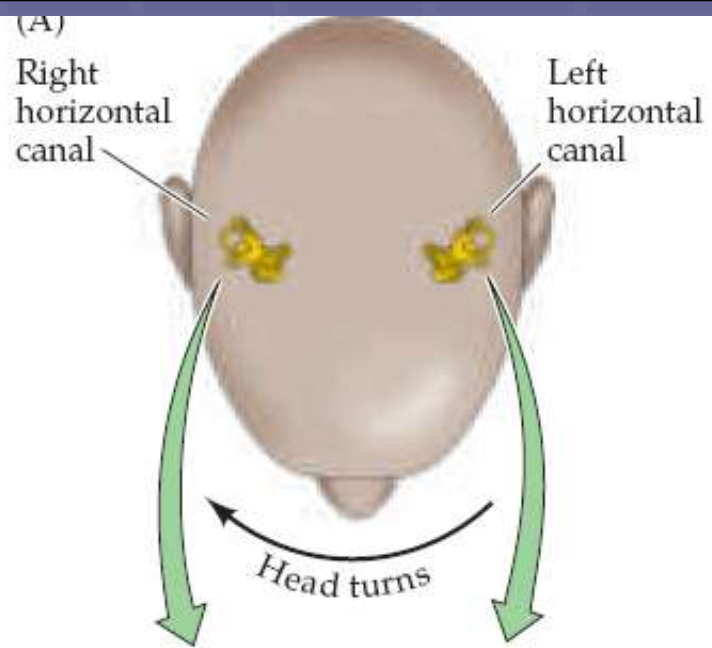


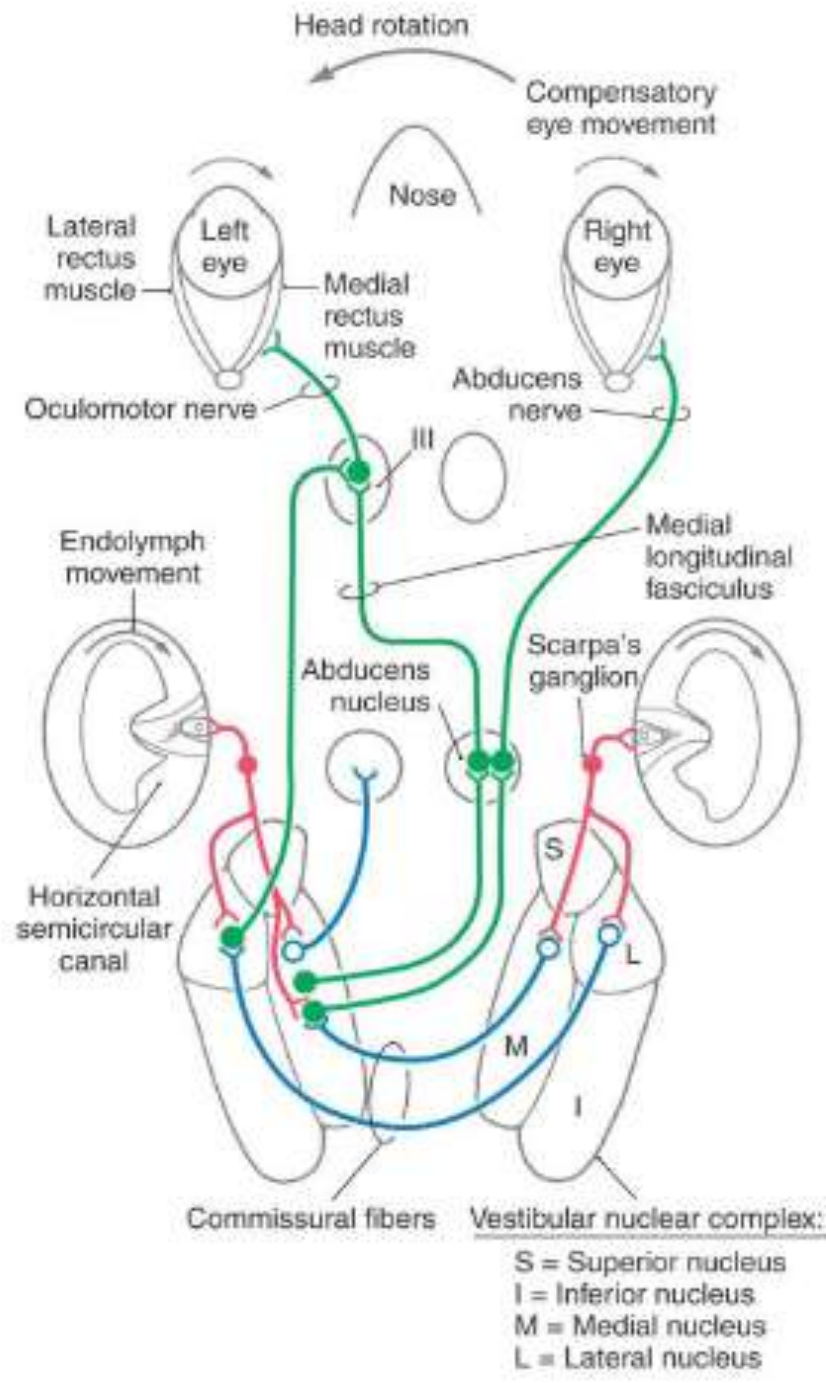


# VESTIBULOOCULAR REFLEX

- **Compensatory for head movements**
  - *Rotational Reflex*
  - *Linear Reflex*







Commissural fibers    Vestibular nuclear complex:  
 S = Superior nucleus  
 I = Inferior nucleus  
 M = Medial nucleus  
 L = Lateral nucleus

# VESTIBULOOCULAR REFLEX

- **Compensatory for head movements**
  - *Rotational Reflex*
  - *Linear Reflex*
- *Nystagmus*

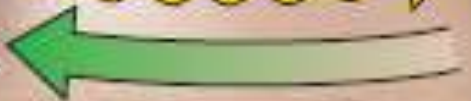
Head rotation



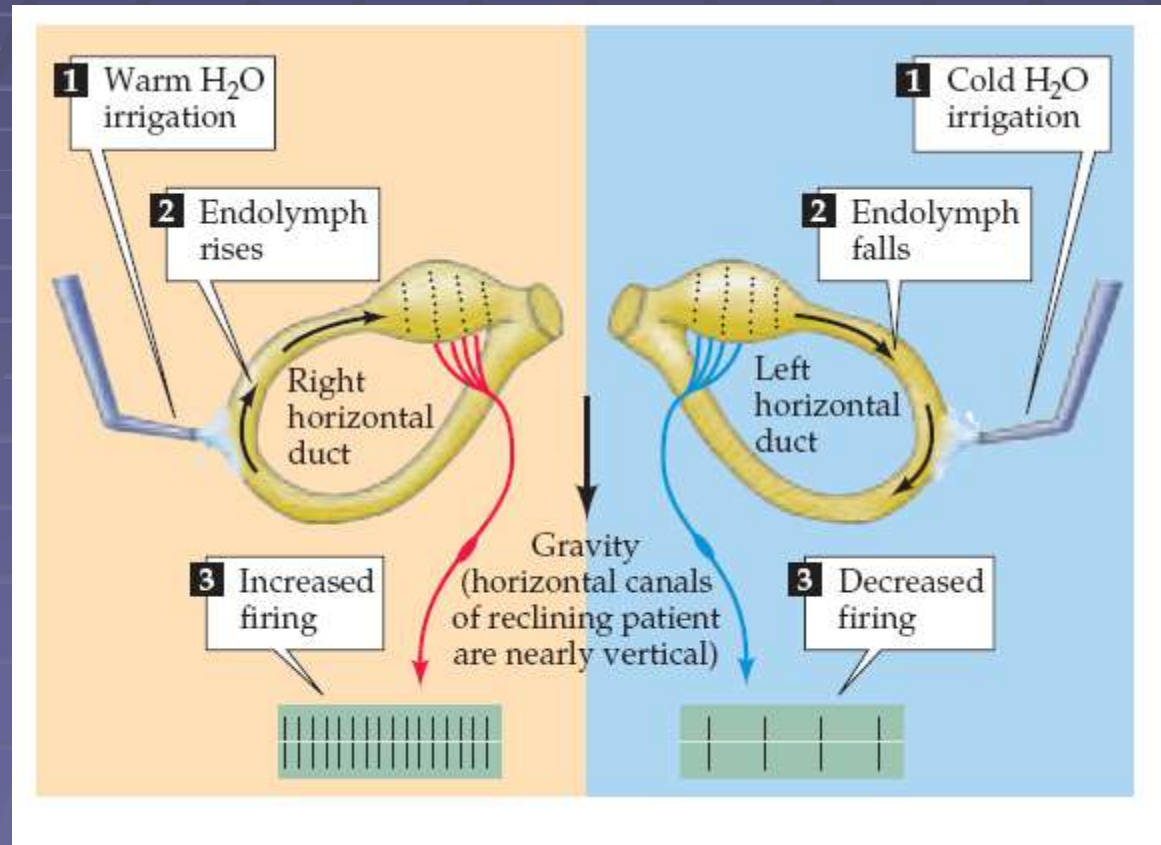
Slow eye movement



Fast eye movement



# Caloric test



Ocular reflexes in conscious patients

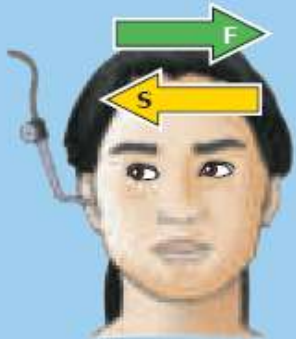
Ocular reflexes in unconscious patients

(1) Normal

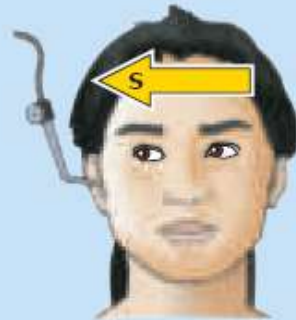
(2) Brainstem intact

(3) MLF lesion (bilateral)

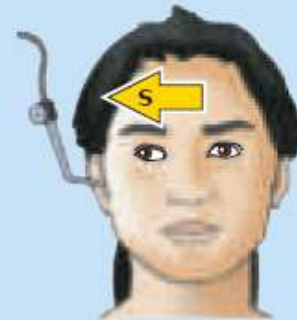
(4) Low brainstem lesion



Cold H<sub>2</sub>O



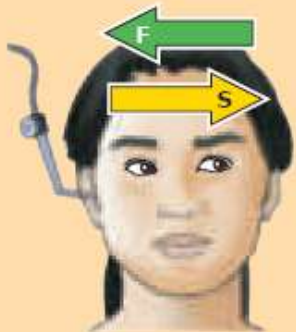
Cold H<sub>2</sub>O



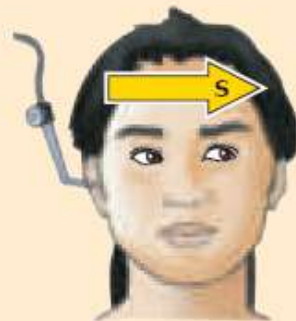
Cold H<sub>2</sub>O



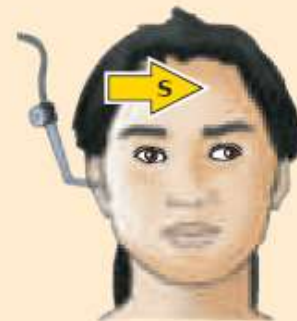
Cold H<sub>2</sub>O



Warm H<sub>2</sub>O



Warm H<sub>2</sub>O



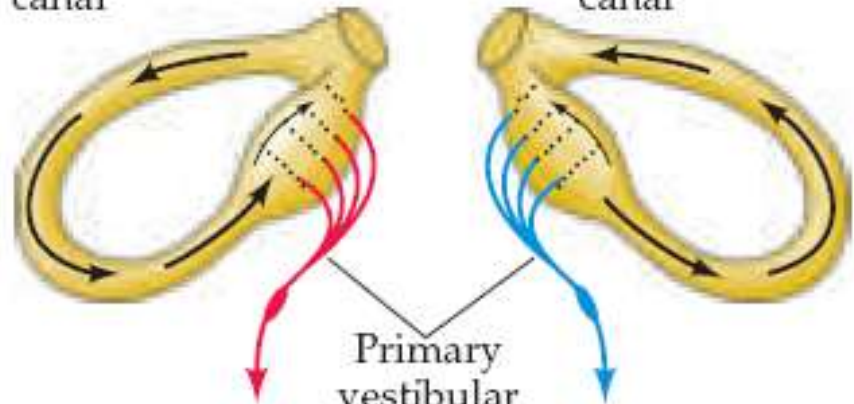
Warm H<sub>2</sub>O



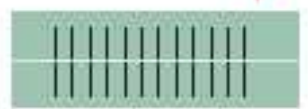
Warm H<sub>2</sub>O

Right horizontal canal

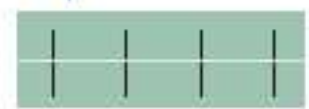
Left horizontal canal



Primary vestibular afferents

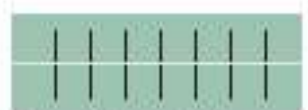


Increased firing



Decreased firing

(2) Spontaneous nystagmus



Baseline firing



No firing

# Ménière Disease

Disease results from a disruption of normal endolymph volume

Symptoms include: Severe vertigo  
Positional nystagmus (nystagmus when head in a particular position)  
Nausea

Affected individuals can also experience-unpredictable attacks of auditory & vestibular symptoms:  
Vomiting  
Tinnitus (ringing in ears)  
Inability to make head movements  
Inability to stand passively  
Low frequency hearing loss

Treatment: administration of a diuretic (hydrochlorothiazide) & a salt restricted diet

Persistent condition: shunt implantation into swollen endolymphatic sac, or  
delivery of a vestibulotoxic agents (gentamicin) into perilymph.



## **DIZZINESS AND VERTIGO**

**Dizziness** is a **nonspecific** term that generally means a spatial disorientation that may or may not involve feelings of movement. Dizziness may be accompanied by nausea or postural instability. A large number of factors may produce a dizzy sensation, and many are not exclusively vestibular in origin.

**Vertigo** is a specific perception of body motion, often spinning or turning, experienced when no real motion is taking place. Vertigo may be perceived as **subjective** vertigo or as **objective** vertigo. In subjective vertigo, the patient experiences the sensation of spinning while things in the environment are not moving; in objective vertigo, the sensation is one of objects spinning while the patient is not moving. As children, we all learn to produce vertigo by whirling in place as fast as possible and then abruptly stopping. For a few moments, the world seems to be spinning in the opposite direction. Examination of the eyes during this phase will reveal a nystagmus that beats in the direction opposite to the original direction of rotation. **Vertigo can also be elicited optokinetically if the visual surroundings are revolved while the body remains stationary.** Many modern amusement games take advantage of this phenomenon to produce the sensation of motion.

### *Benign Paroxysmal Positional Vertigo*

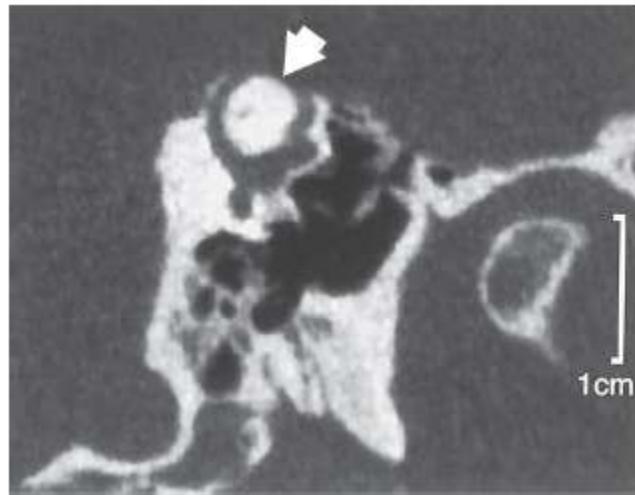
One of the most common vestibular disorders observed clinically is **benign paroxysmal positional vertigo**. This condition is characterized by brief episodes of vertigo that coincide with particular changes in body position. Typically, episodes may be triggered by turning over in bed, getting up in the morning, bending over, or rising from a bent position. The pathophysiologic mechanism of benign positional vertigo is not clearly understood, but posterior canal abnormalities are implicated. One possible explanation is that otoconial crystals from the utricle separate from the otolith membrane and become lodged in the cupula of the posterior canal (a condition called **cupulolithiasis**). The resulting increased density of the cupula produces abnormal cupula deflections when the head changes position relative to gravity.

### ***Vestibular Neuritis***

Patients often present with severe vertigo, nausea, and vomiting yet have no accompanying hearing loss or other central nervous system abnormalities. In many of these cases, **vestibular neuritis** is diagnosed and is thought to involve edema of the vestibular nerve (or ganglion). The edema is most commonly believed to be produced through an acute viral infection, such as herpes simplex virus. In fact, some patients report a recent history of upper respiratory tract infection, cold, or influenza. Treatment options include antiemetics, vestibular suppressants, corticosteroids to reduce inflammation, and antiviral agents.

### *Semicircular Canal Dehiscence*

On occasion, a condition may develop in which a portion of the temporal bone overlying either the anterior or the posterior semicircular canal thins so much that an opening (dehiscence) is created next to the dura (Fig. 22-5). In affected patients, the canal dehiscence exposes the normally closed bony labyrinth to the extradural space. Symptoms can include **vertigo** and **oscillopsia** (a sense that objects are moving to and fro, oscillating, in the visual fields) in response to loud sounds (the **Tullio phenomenon**) or in response to maneuvers that change middle ear or intracranial pressure. The eye movements evoked by these stimuli (nystagmus) align with the plane of the dehiscent superior canal. Surgical closure of the defect by bone replacement is often performed.



# Semicircular Canal Dehiscence (opening)

Temporal bone overlying the anterior or the posterior semicircular canal thins, creating an opening/dehiscence next to the dura.



Text Fig. 22-5

CT scan of the temporal bone projected into the plane of the left superior/anterior canal, in a patient with superior canal dehiscence syndrome.

The dehiscence exposes the bony labyrinth to the extradural space.

Symptoms: vertigo and oscillopsia in response to loud sounds (Tullio Phenomenon), or in response to maneuvers that change middle ear or intracranial pressure.

Nystagmus evoked by these stimuli aligns with the plane of the dehiscent superior canal.

Treatment: Surgical closure of the defect by bone replacement.

Dizziness: non-specific term.  
generally means **spatial disorientation**.  
may or may not involve feelings of movement.  
may be accompanied by nausea or postural instability.  
may be caused by factors other than vestibular dysfunction.

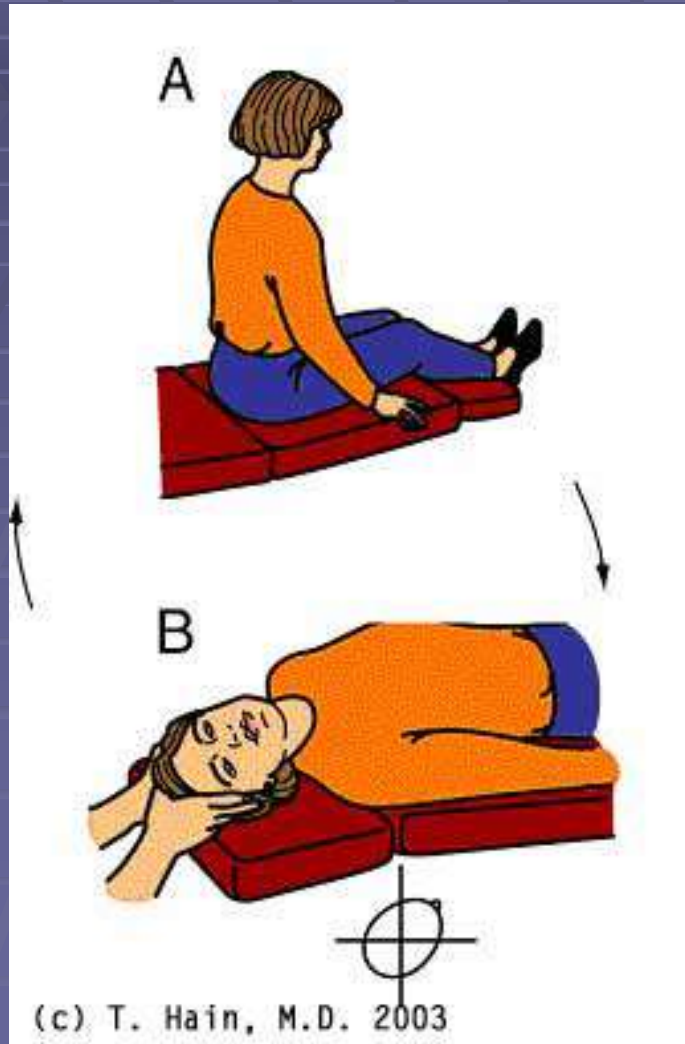
Vertigo: specific term.  
perception of body motion.  
spinning or turning sensation when no real motion is taking place.

Benign Paroxysmal Positional Vertigo  
common clinical disorder.  
condition characterized by **brief episodes of vertigo that coincide with particular changes in body position**.  
pathophysiology poorly understood.  
posterior canal abnormalities are implicated.  
otoconia crystals in the utricle may separate from the otolith membrane and become lodged in the cupula, causing abnormal cupula deflections.

Vestibular Neuritis:  
severe vertigo, nausea, vomiting  
no hearing loss or other CNS abnormalities  
**possible edema of the vestibular nerve/ganglion**.  
thought to be produced by acute viral infection.  
treated with antiemetics, vestibular suppressants, corticosteroids, & antiviral agents.

# Dix-Hallpike test

The definitive diagnostic test for benign paroxysmal positional vertigo



- Patient from sitting to supine position.
- Head turned  $45^{\circ}$  to one side and extended  $20^{\circ}$  backward.
- Observe eyes for nystagmus (30 sec.).
- Bring back to a sitting position.
- Small delay, test other side.
- A positive test consists of a burst of nystagmus.
- Posterior canal BPPV (more common) – eyes jump upward.