

VESTIBULAR EVOKED MYOGENIC POTENTIALS (VEMP)



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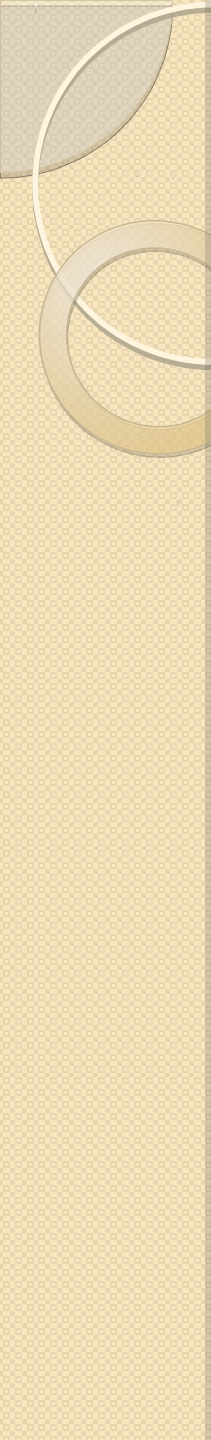
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Anatomy and Physiology of Vestibular system

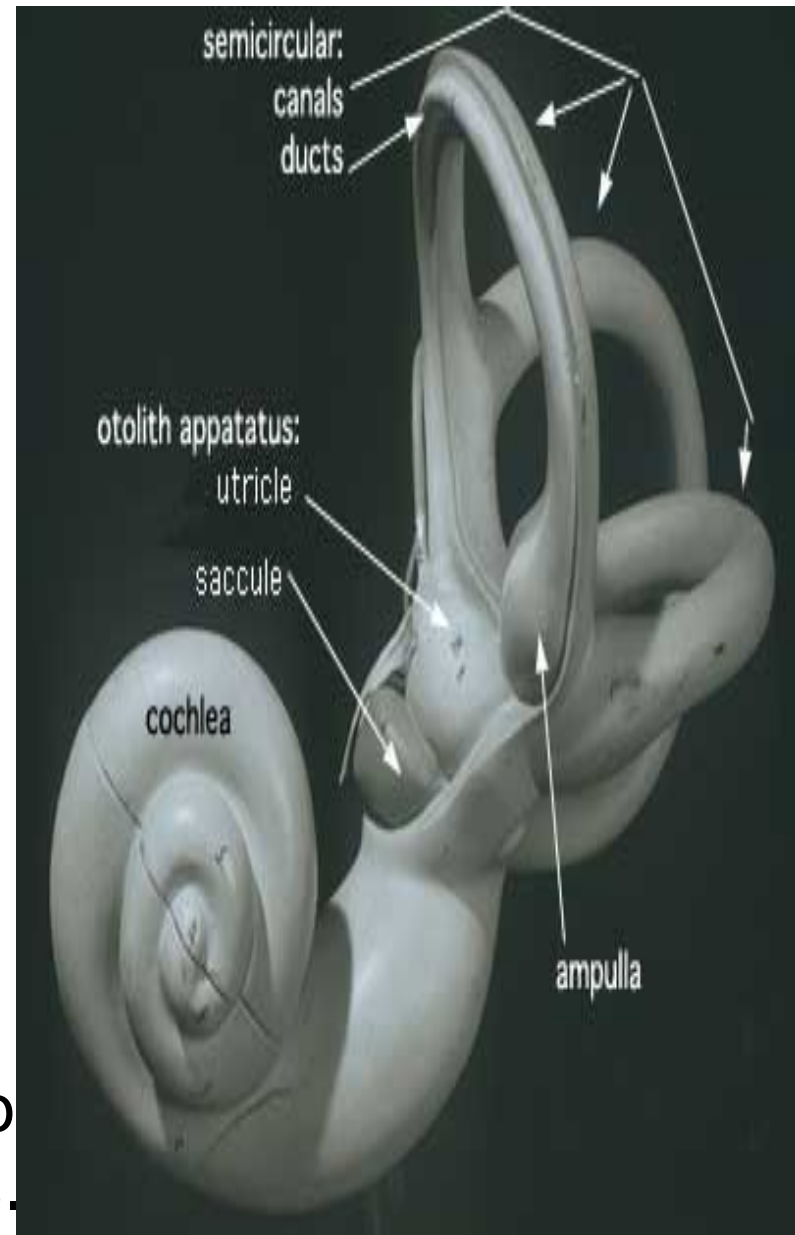
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- The vestibular system is the system of the balance.
 - It also involved in the function of maintaining visual fixation during head movement and in maintaining posture and muscle control.

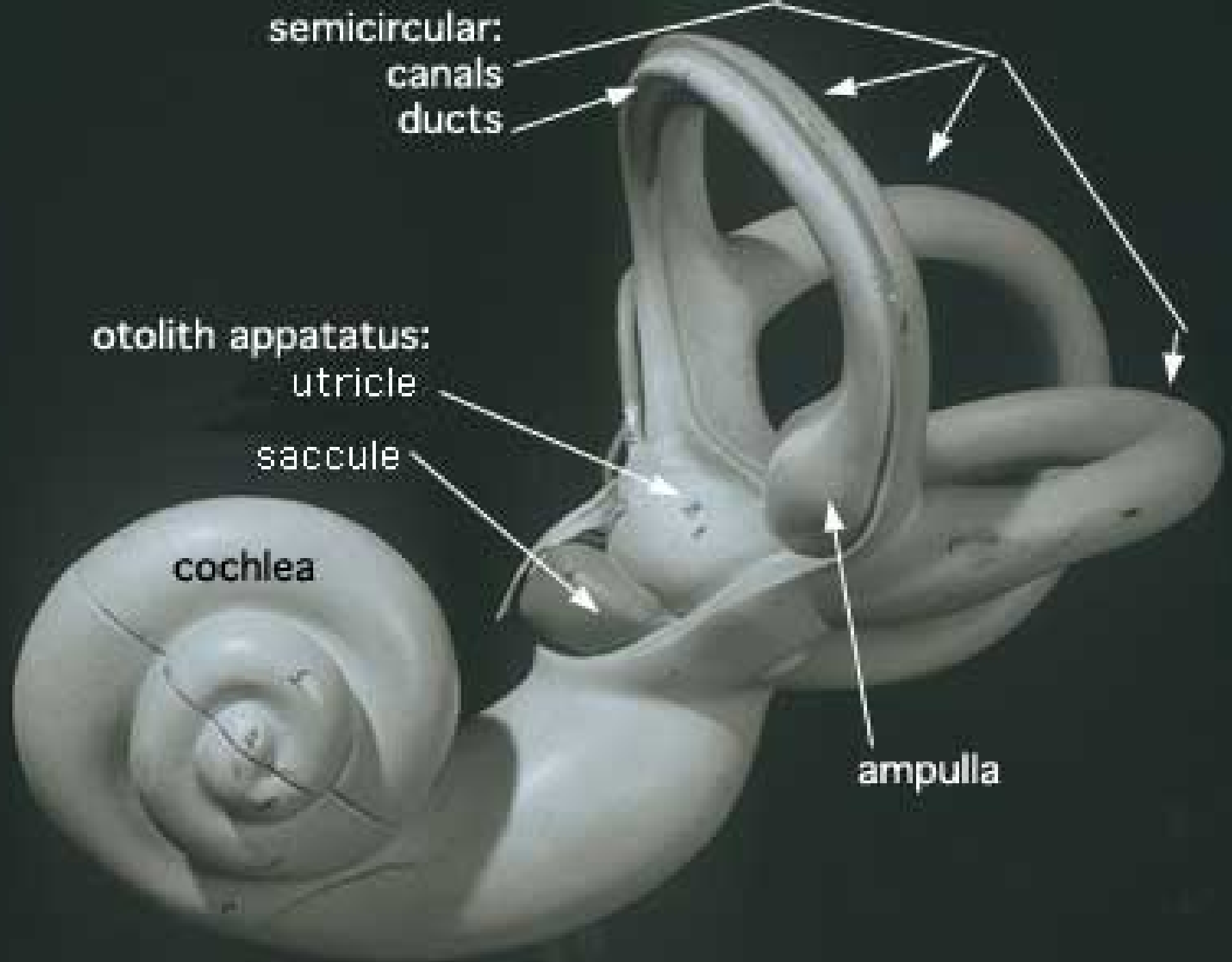
- ▶ Each side of this bilateral system consists of two types of sensors:

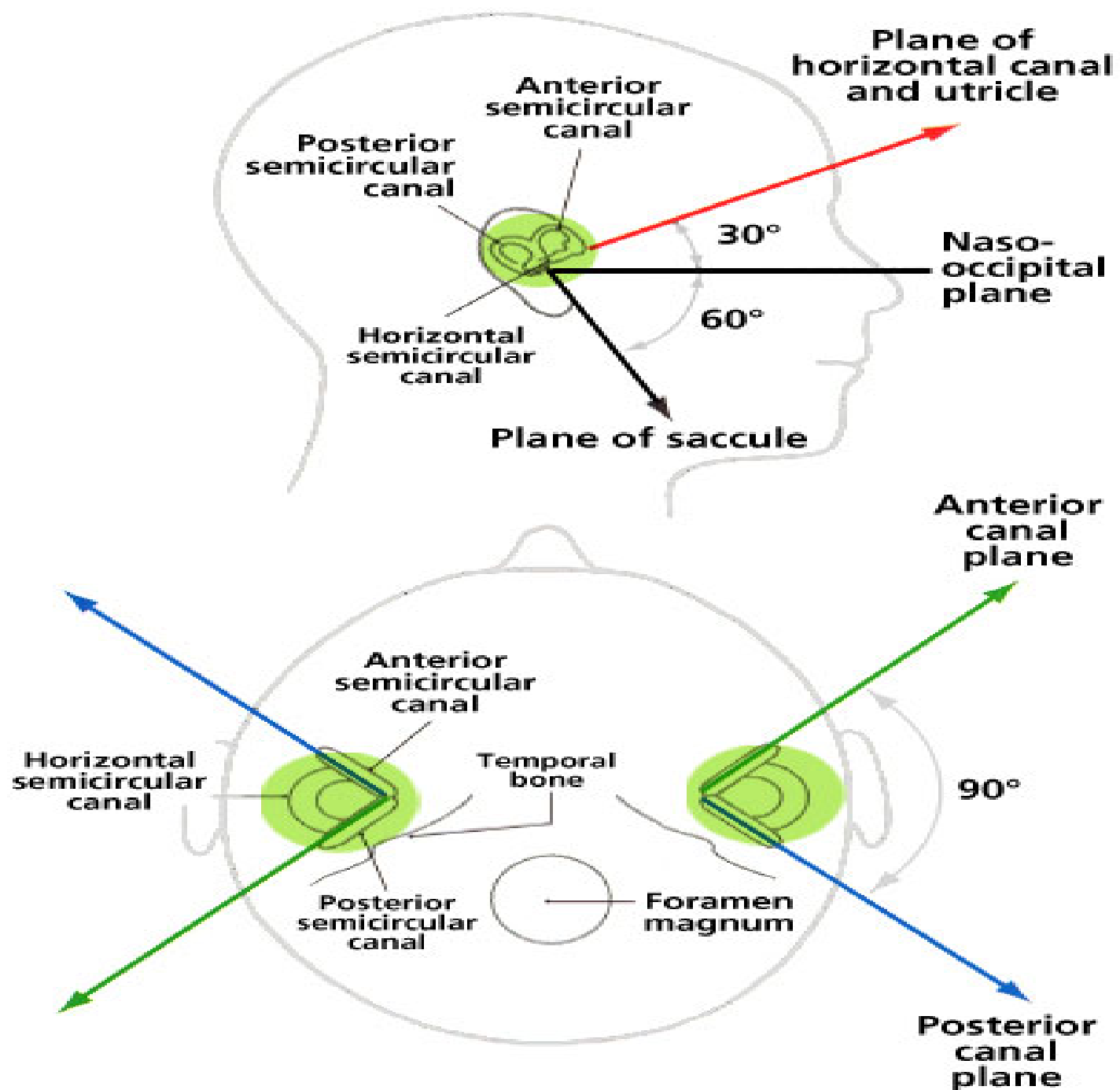
2 Otolith organs (Saccule and Utricle) which senses linear movement and gravity

3 Semi Circular Canals (SCC)

arranged at right angle to each other sensing Rotational movement in three planes.







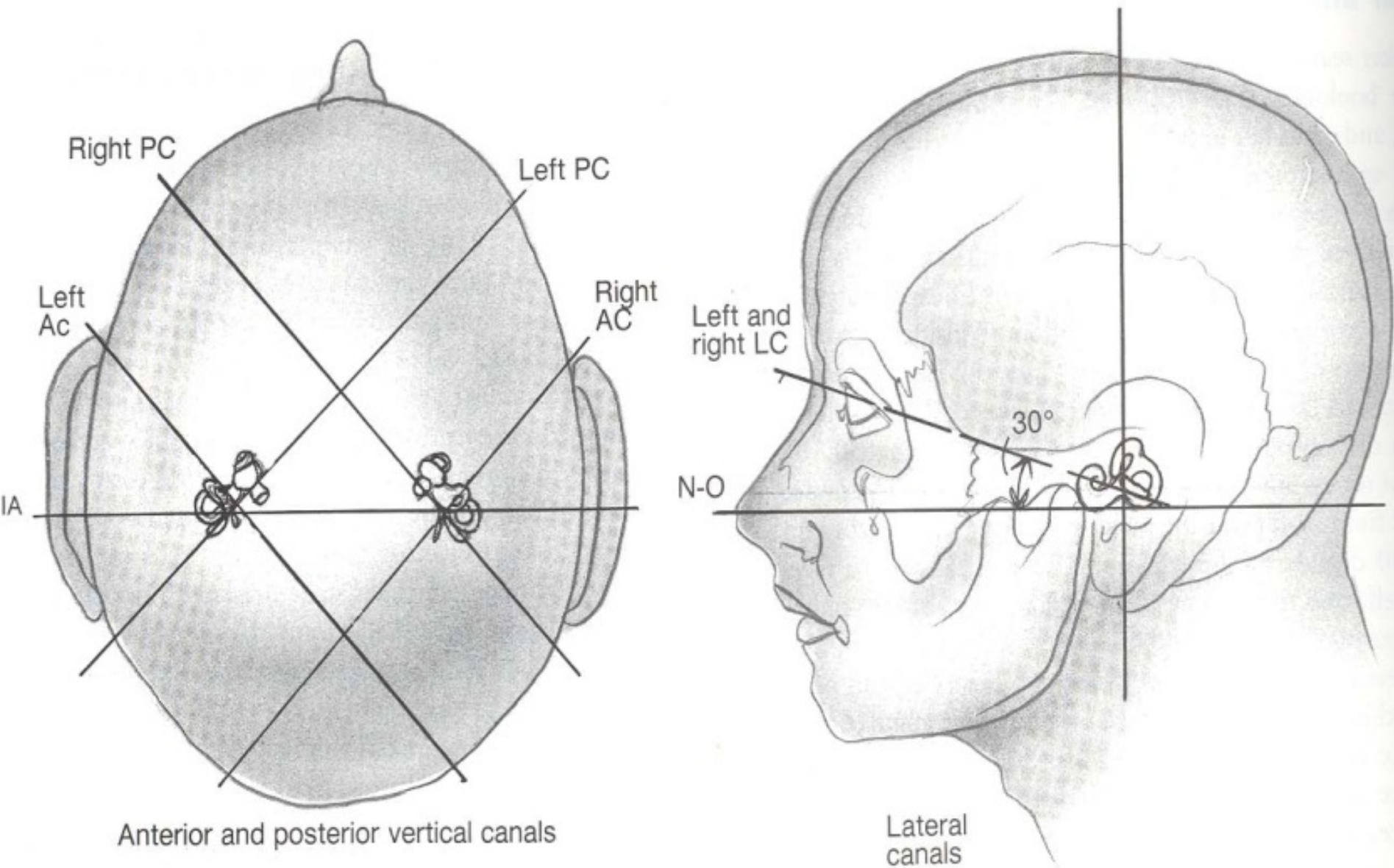
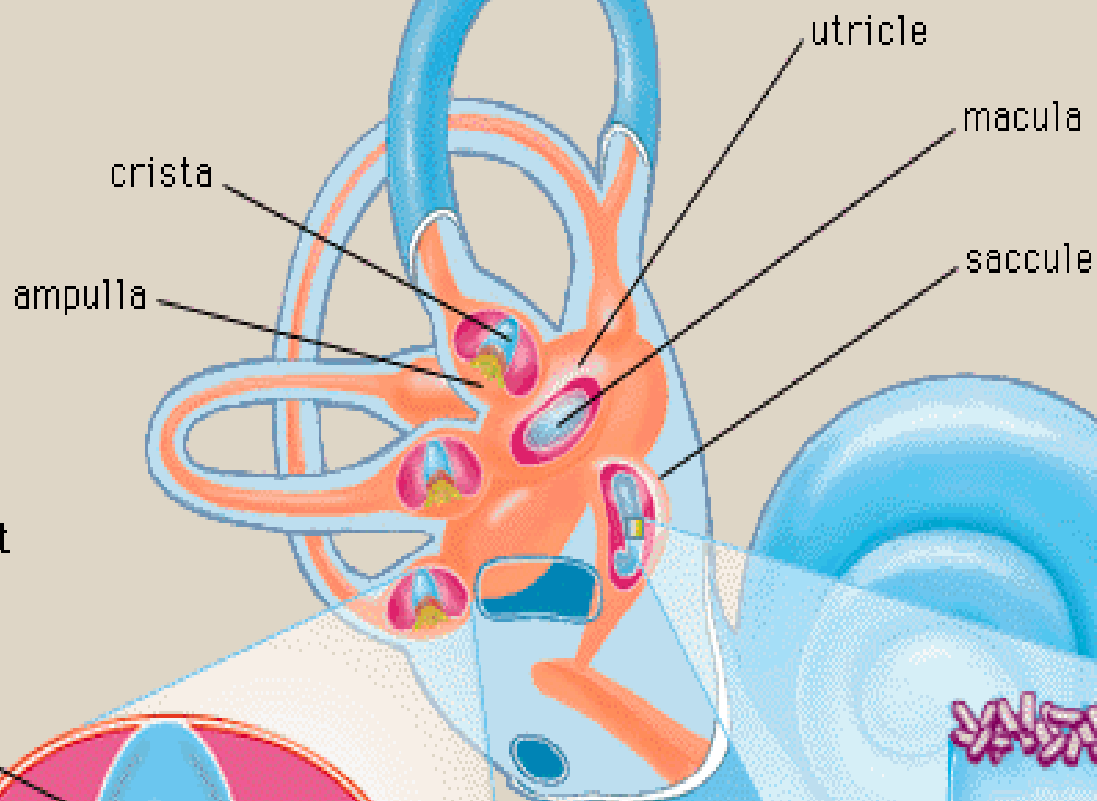
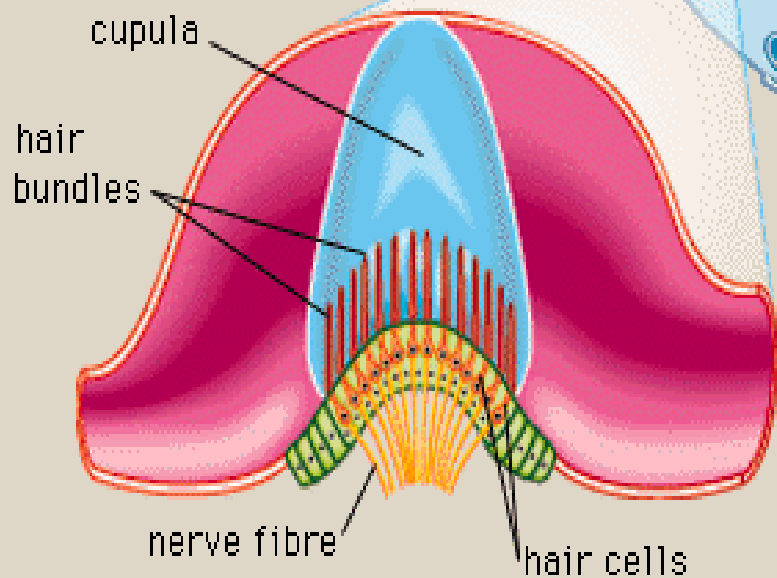


FIGURE 130.2. A: Membranous labyrinth of the right ear. **B:** Planes of the semicircular canals. The size of the canals is exaggerated. AC, Anterior vertical semicircular canal; IA, interaural; LC, lateral semicircular canal; N-O, nasal-occipital axis; PC, posterior vertical semicircular canal; RC, rostral-caudal axis.

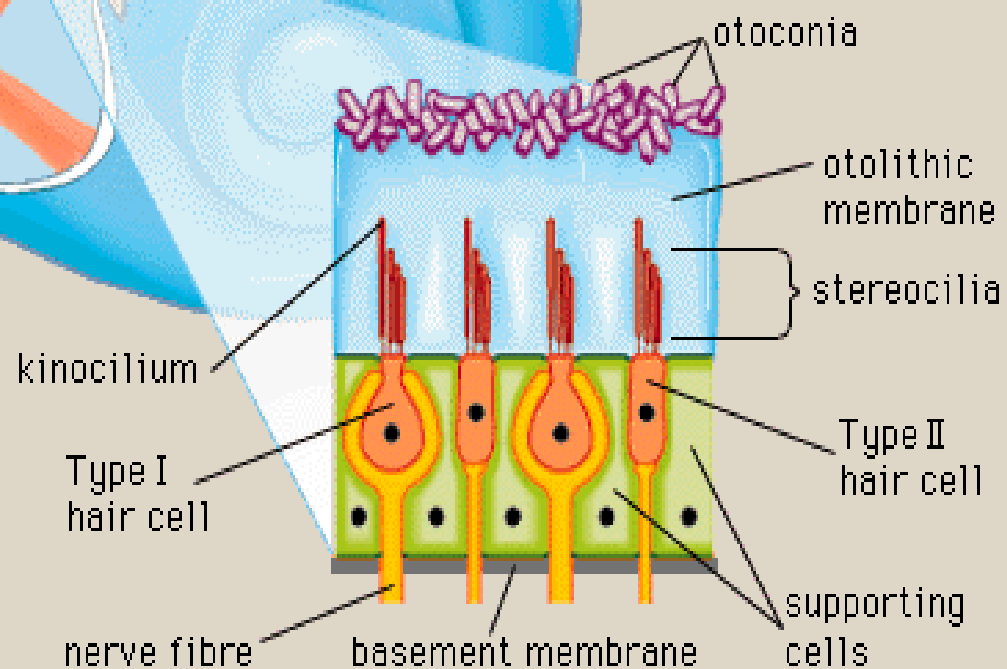
vestibular system

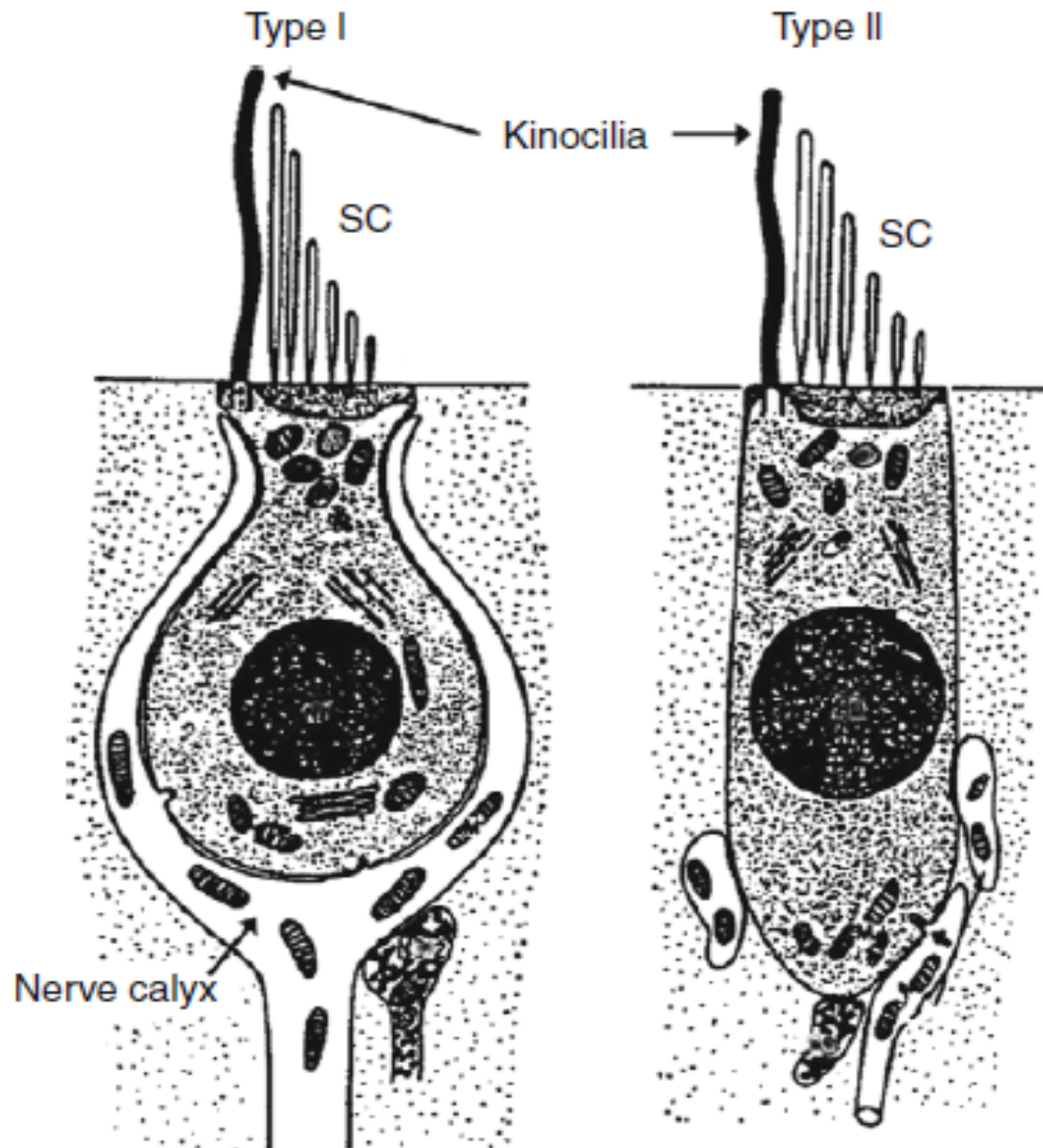


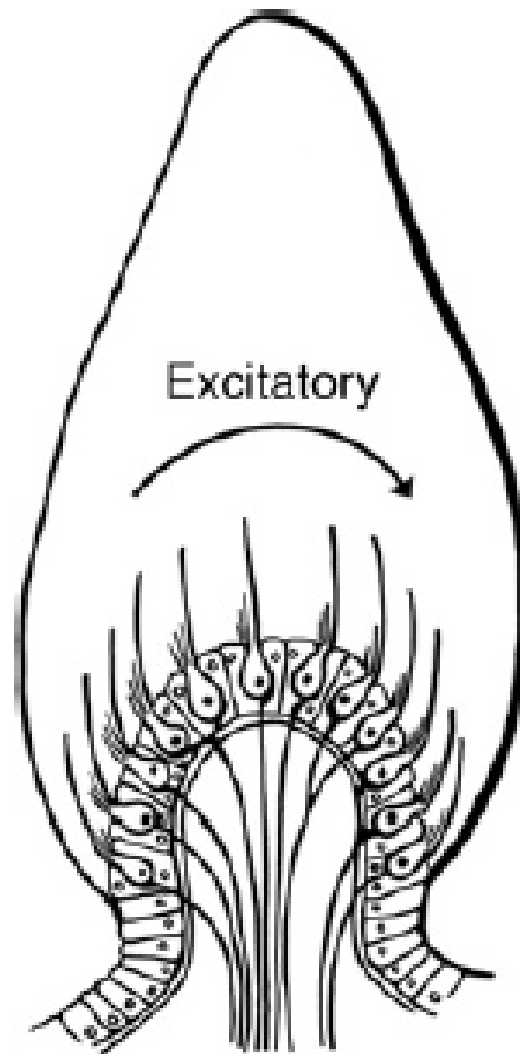
enlargement of crista



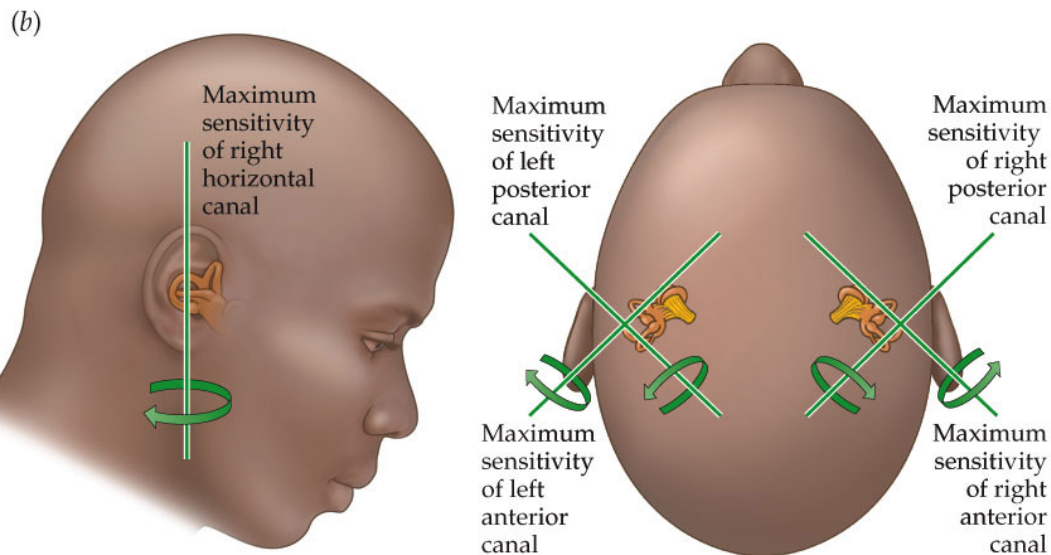
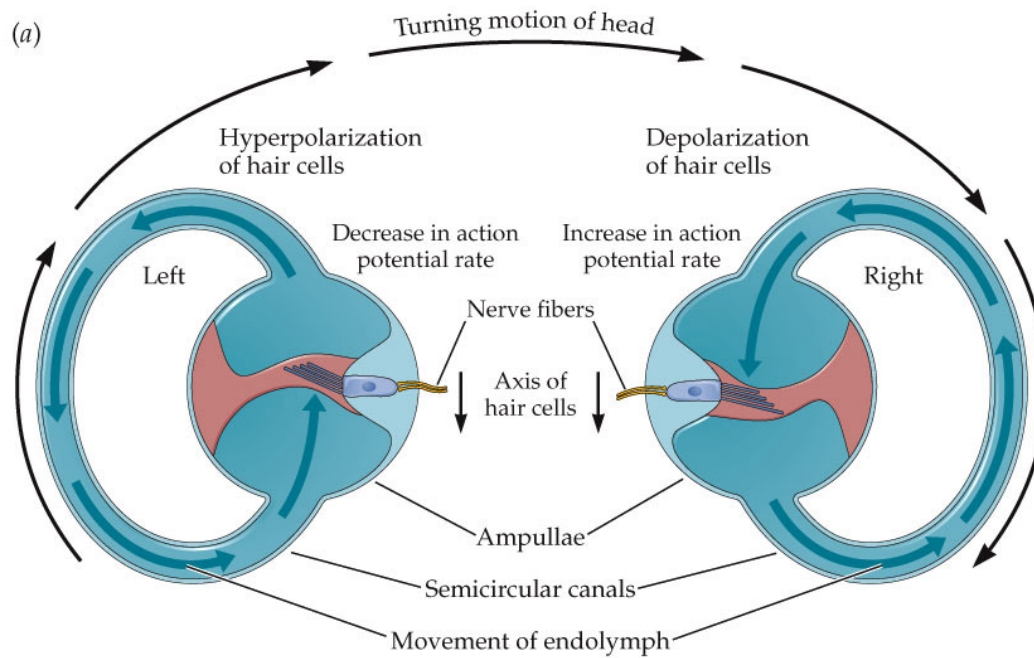
enlargement of macula







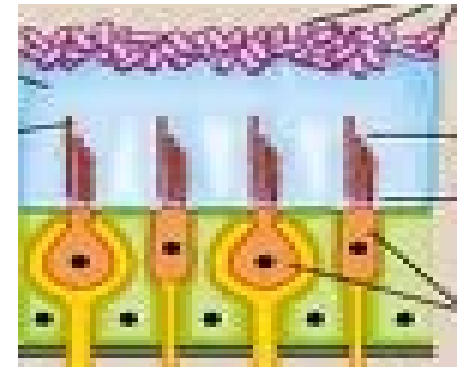
Crista ampullaris



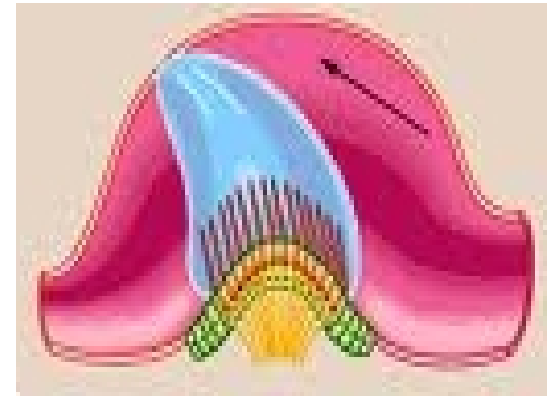
SENSATION & PERCEPTION 2e, Figure 15.10

Recognition of head movement

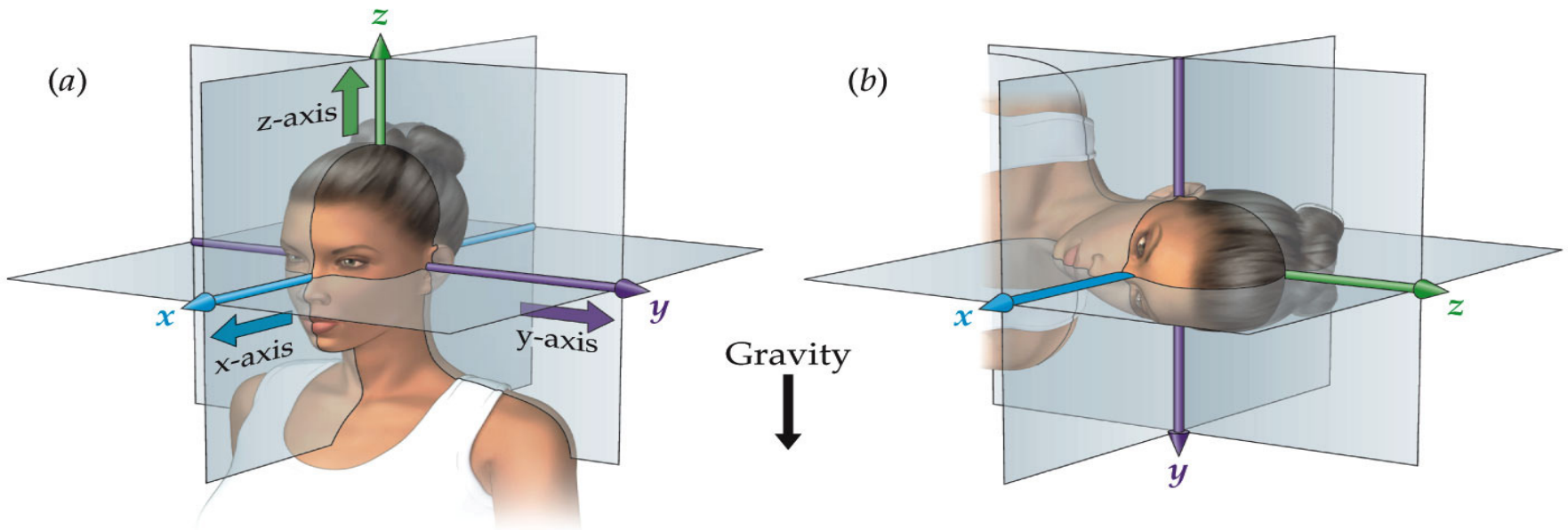
Linear acceleration
and head tilt



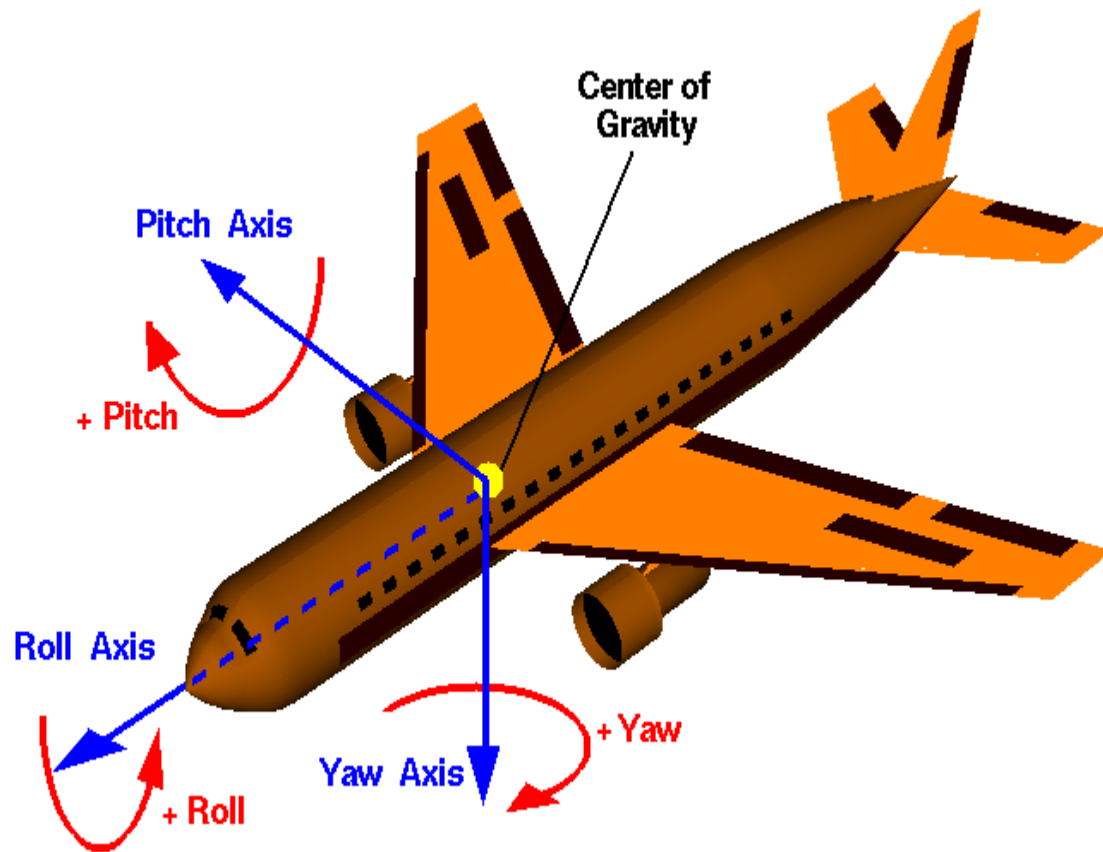
Angular acceleration

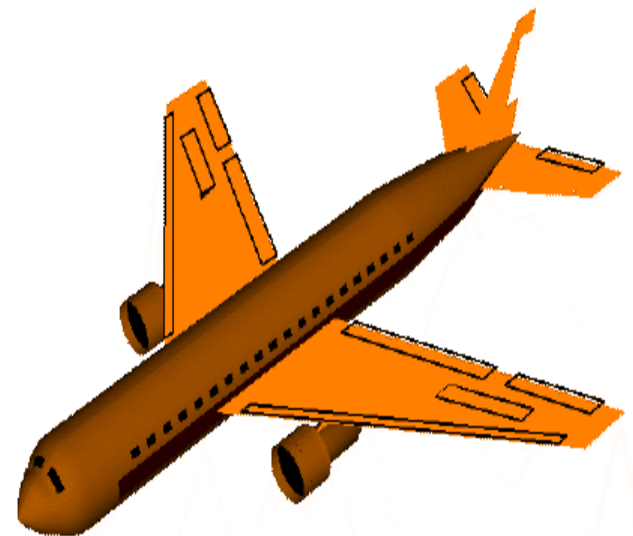
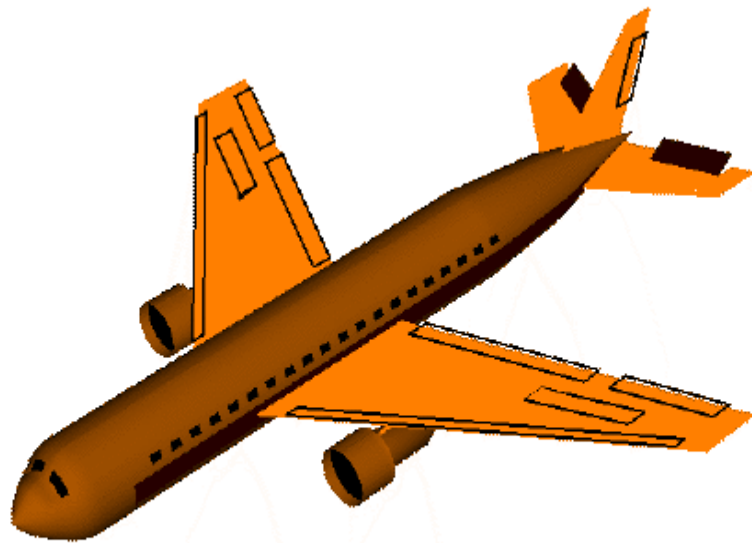
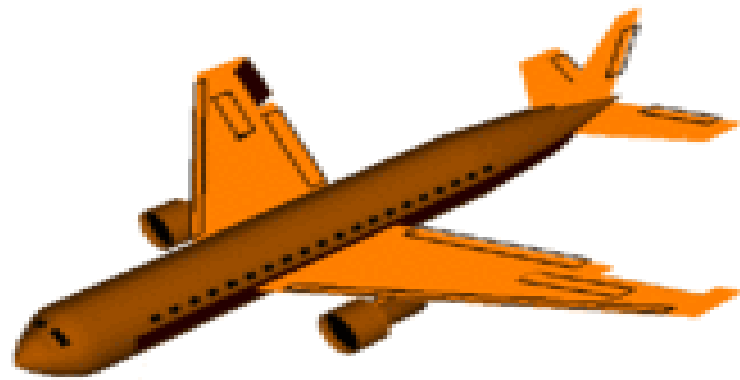


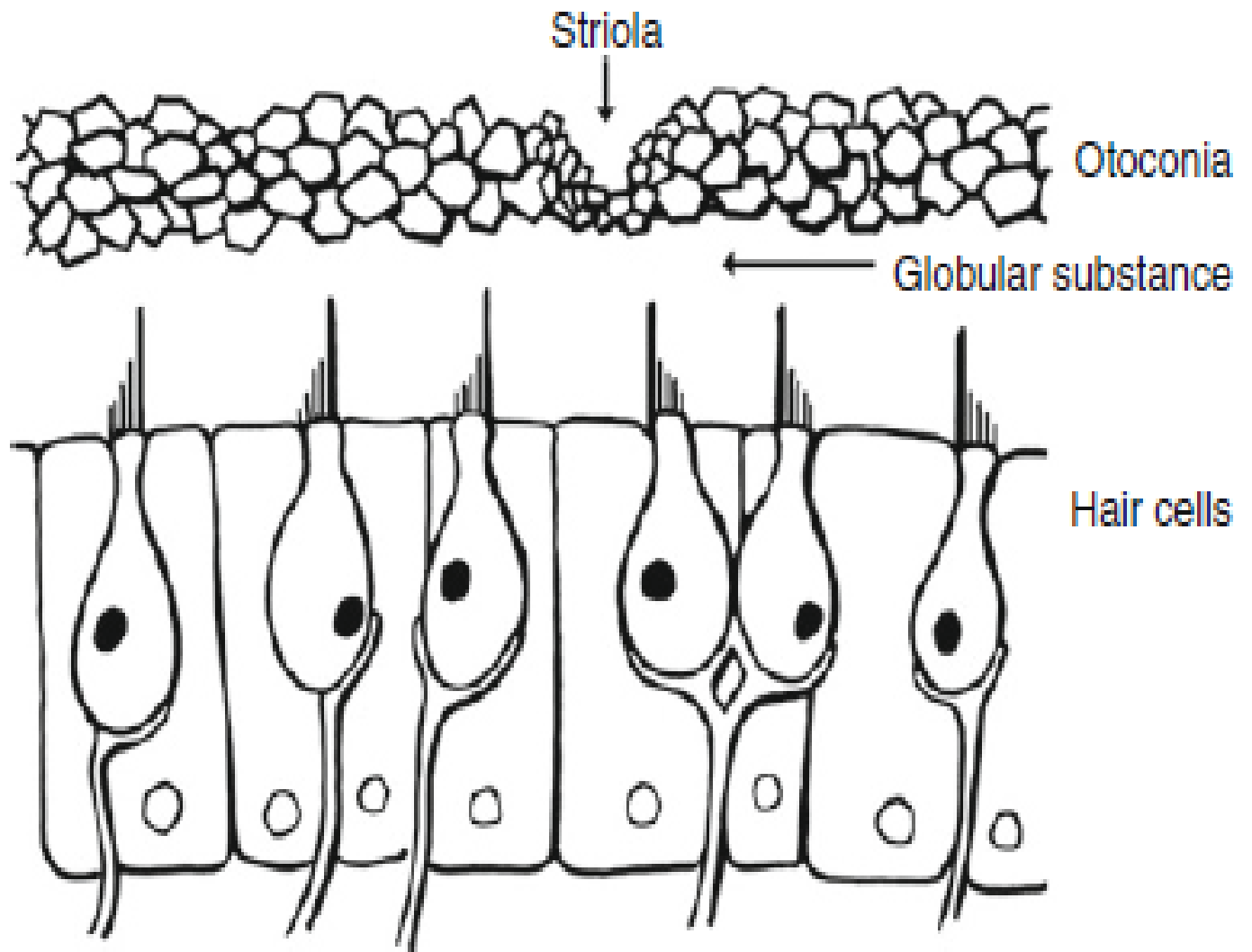
Angular acceleration

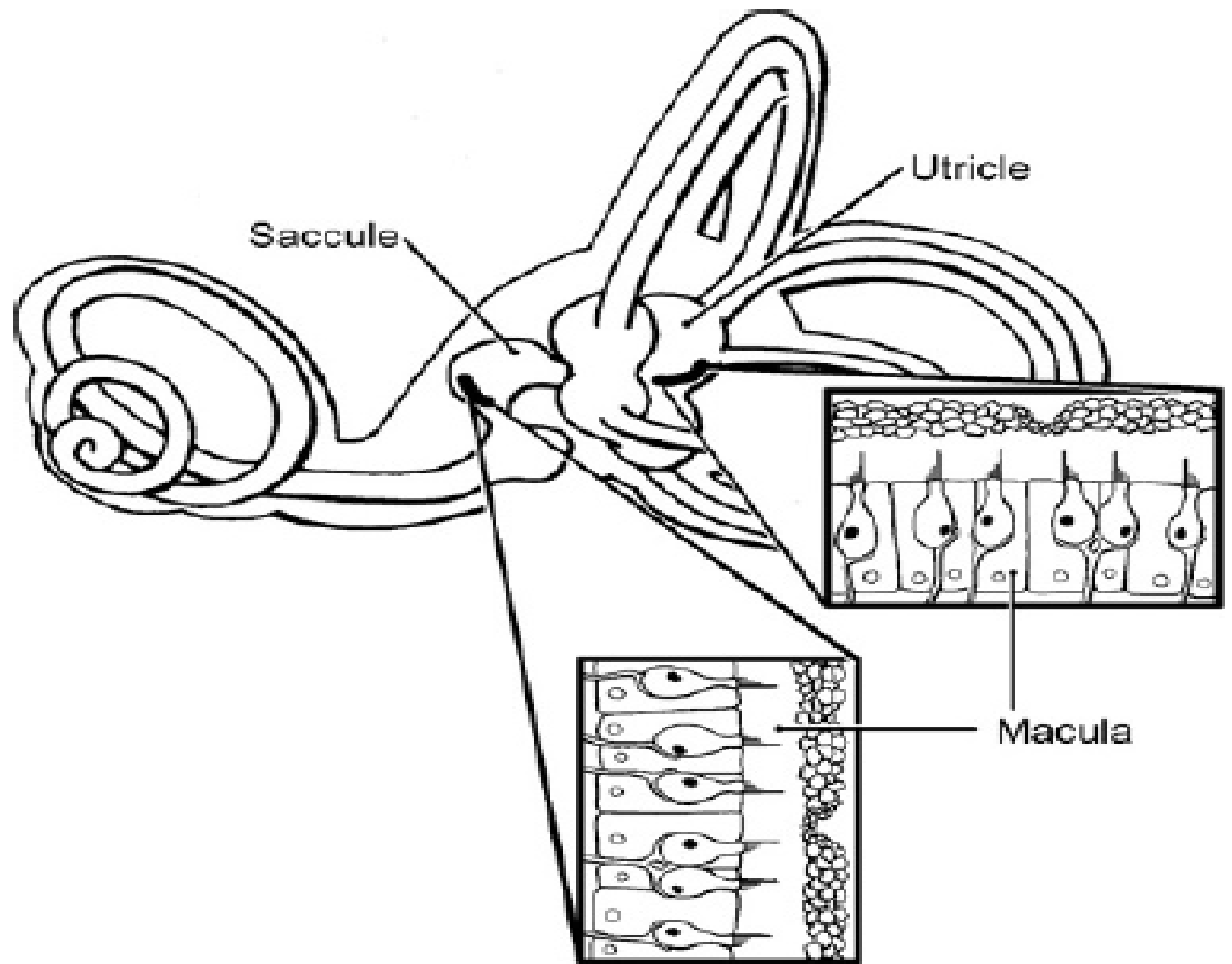


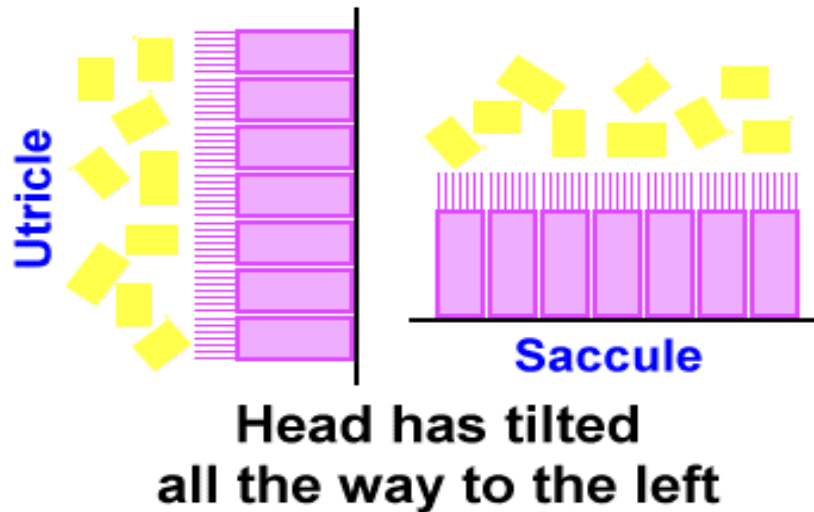
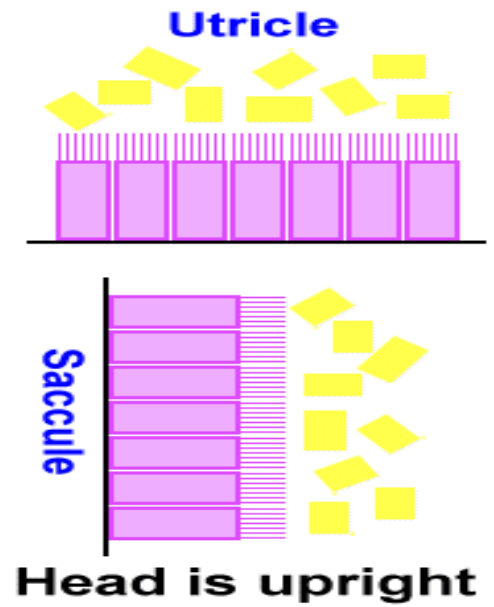
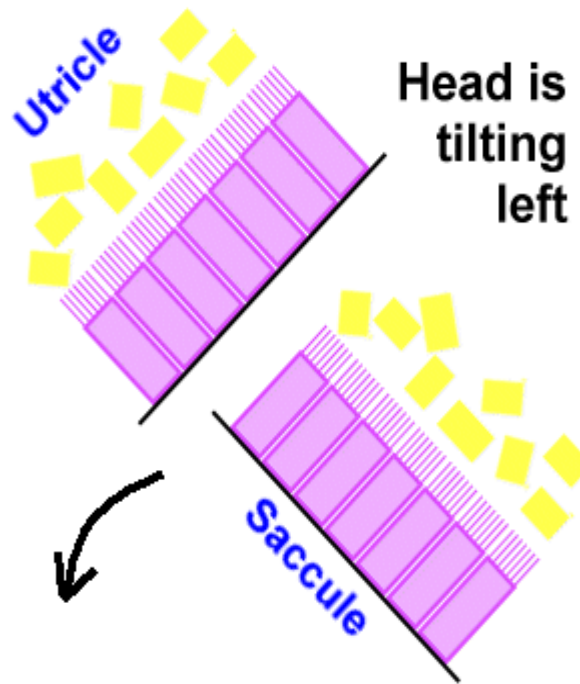
Three directions for sense of rotation

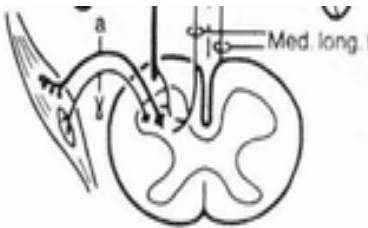
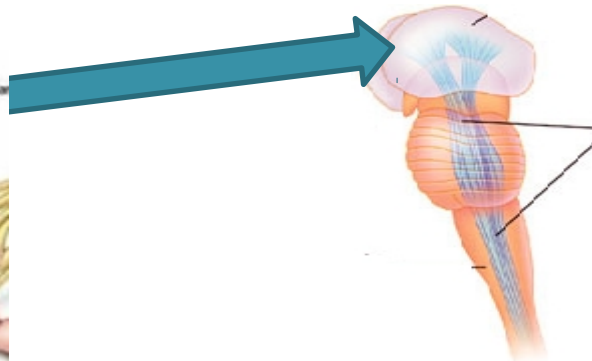
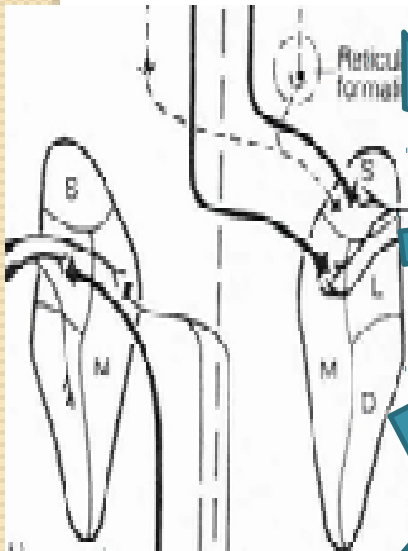
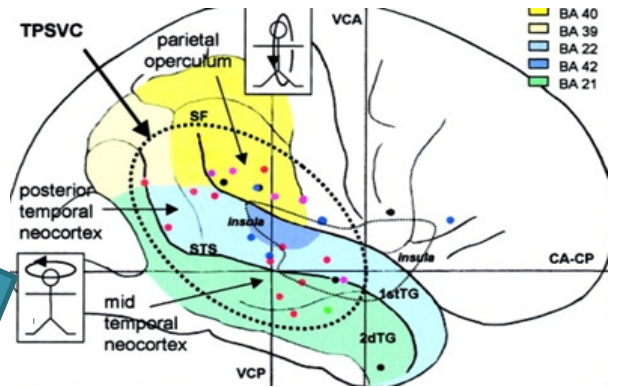
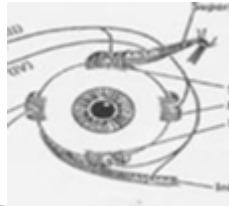


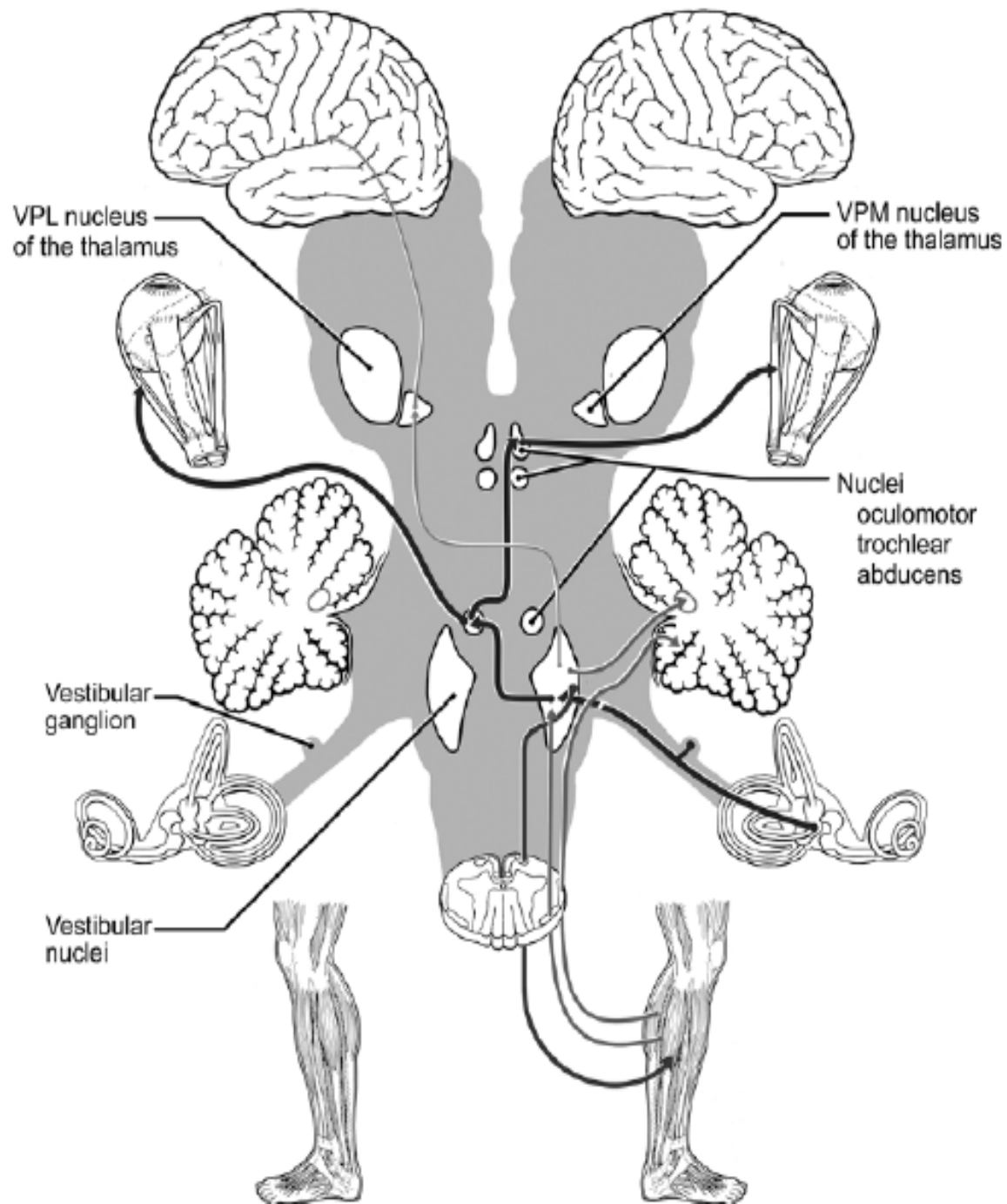


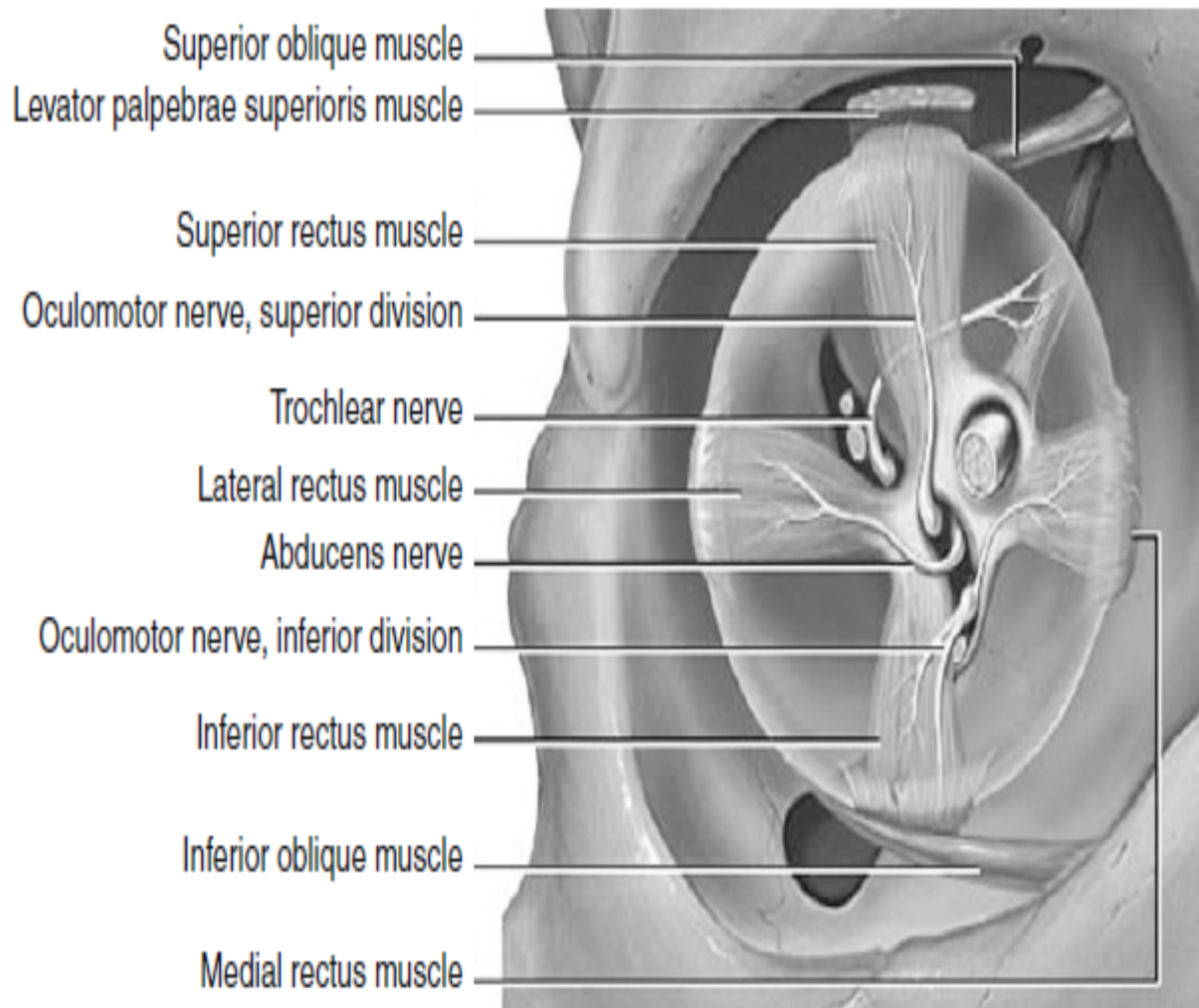


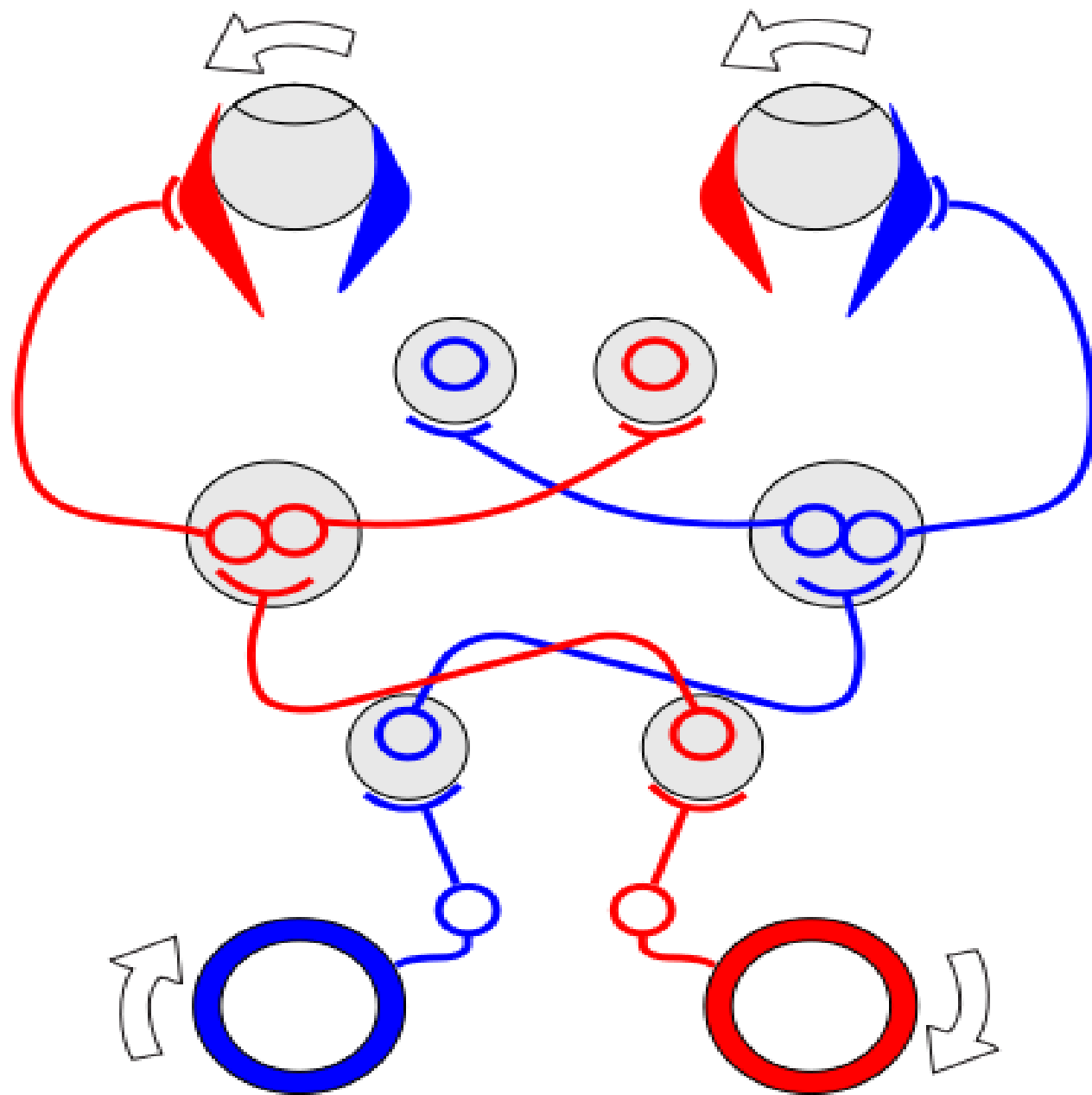






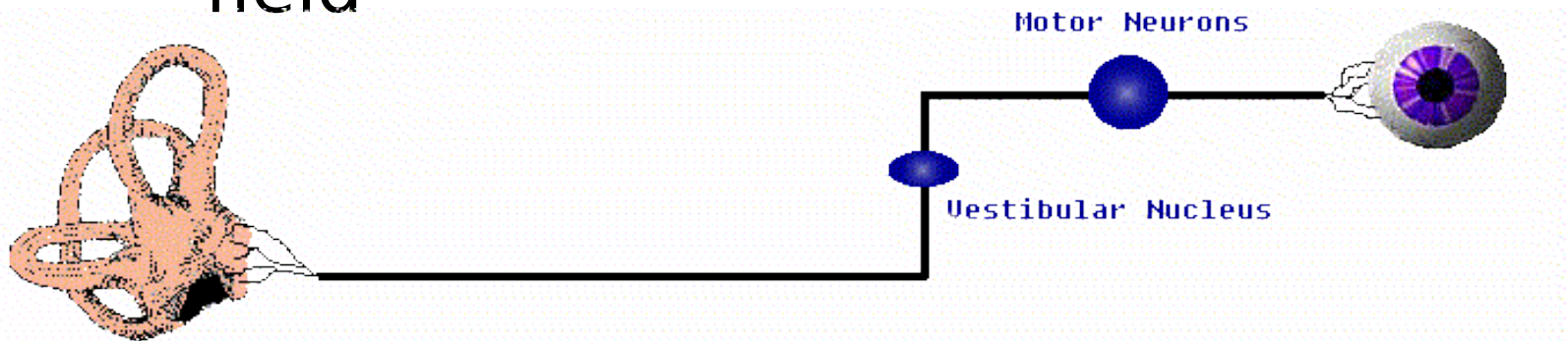






Vestibular Reflexes

- Vestibulo-ocular
 - Helps maintain stability of visual field



The cerebellum is responsible for timing, fine-tuning and coordinating the motor system.



Central oculomotor System

- Optokinetic
- Saccade
- Smooth pursuit

Agenda of Presentation

- *Anatomy and physiology*
- *Historical background*
- *Clinical test protocol*
- *Waveform analysis*
- *Analysis strategies*
- *Factors influencing analysis*

VEMP Background-1

(Important contributors)

- Tullio phenomenon described in 1929
- Von Békésy recorded vestibular response to high intensity (> 125 dB) sounds
- Reginald Bickford and colleagues reported vestibular evoked myogenic responses recorded from posterior neck muscles (remember AMLR responses history and PAM response)

VEMP Background-2 (Important contributors)

- Concept of acoustic stimulation of the saccule was introduced as early as 1971(Townsend & Cody, 1971).
- Animal experiments confirmed stimulation of otolith organs by loud organs sounds (Young et al, 1977,Didier et al, 1987)
- Colebatch and colleagues in Sydney Australia reported first clinical and application in early 1990s
- In 1995, Robertson & Ireland coined the term “VEMP”

VEMP Background-3

Evidence for Vestibular origin

- Animal research confirms that saccule responds to acoustic stimulation
- VEMP can be recorded from persons with intact vestibular function and also profound hearing impairment (deafness)
- VEMP disappears when vestibular nerves are severed (e.g., surgically sectioned)

“VEMP reflects a vestibulocollic reflex, that is, a quick reflexive change in muscle tone (flexor or extensor, depending on the muscle group) that occurs to stabilize the head following an unexpected translation (Zapala & Brey , 2004)

VEMPs

Clinical Protocol-1

Stimulus parameters

- Transducer = Insert earphones
- Type = click or tone burst (low frequency is optimal)
- Duration = 0.1 ms click or 2-0-2 cycle tone burst
- Intensity = > 95 dB nHL
- Polarity = rarefaction Polarity
- Rate = 3 to 5/second

VEMPs

Clinical Protocol-2

• Acquisition parameters

- Acquisition parameters
- Analysis time
- Pre-stimulus = 10 to 20 ms
- Post-stimulus = 50 to 100 ms

Electrodes

- Non-inverting = midpoint of sternocleido mastoid muscle
- Inverting = sternoclavicular junction or other sites, e.g., hand
- Ground = forehead

Filter settings

- High pass = 30 Hz
- Low pass = 1500 Hz
- Notch = no
- Sweeps = 45 to 250

VEMPs Analysis Strategies

- The VEMP reflects a transient inhibition of the spontaneous activity in the SCM during stimulation
- Record optimal response from each side
- Calculate
 - P1 latency
 - N1 latency
 - P1 —N1 amplitude
 - Inter-side differences for each response parameter
- ✓ RE: normal values *or*
- ✓ > 3:1 ratio of normal versus involved side
- ✓ Patient response values versus normative data
- ❖ Analysis problems (G. Jacobson, 2002)
 - Difficult to record a response from some patients
 - Patients with limited neck mobility (e.g., elderly)

VEMPs: Inter-side Analysis of Amplitude (Amplitude Ratio)*

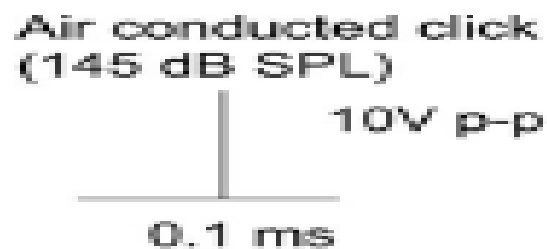
- Amplitude Ratio (%) = $(AU-AA) / (AU+AA)$
U = unaffected, A = affected
- Amplitude Ratio (%) = $(AL-AR) / (AL+AR)$
L= left ear ,R= right ear
- Normal ratio = < 0.35
- Abnormal ratio = > 0.35
- The reflex amplitude scales in proportion to tonic EMG activity and should therefore be normalized to the level of EMG activity
("corrected reflex amplitude" = peak-to-peak amplitude/prestimulus rectified EMG activity).

*for age of less than 60 yrs

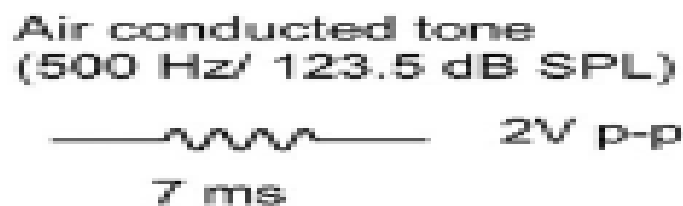
Methods of Recording VEMP

- Air Conducted VEMP
- Bone Conducted VEMP
- Skull Tapps
- Galvanic Stimulus

A



B



C



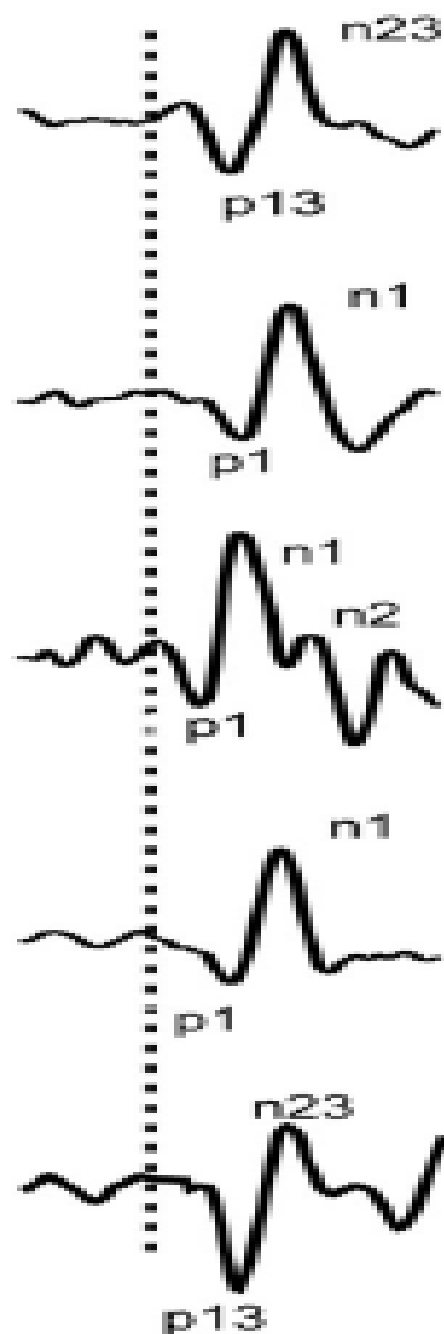
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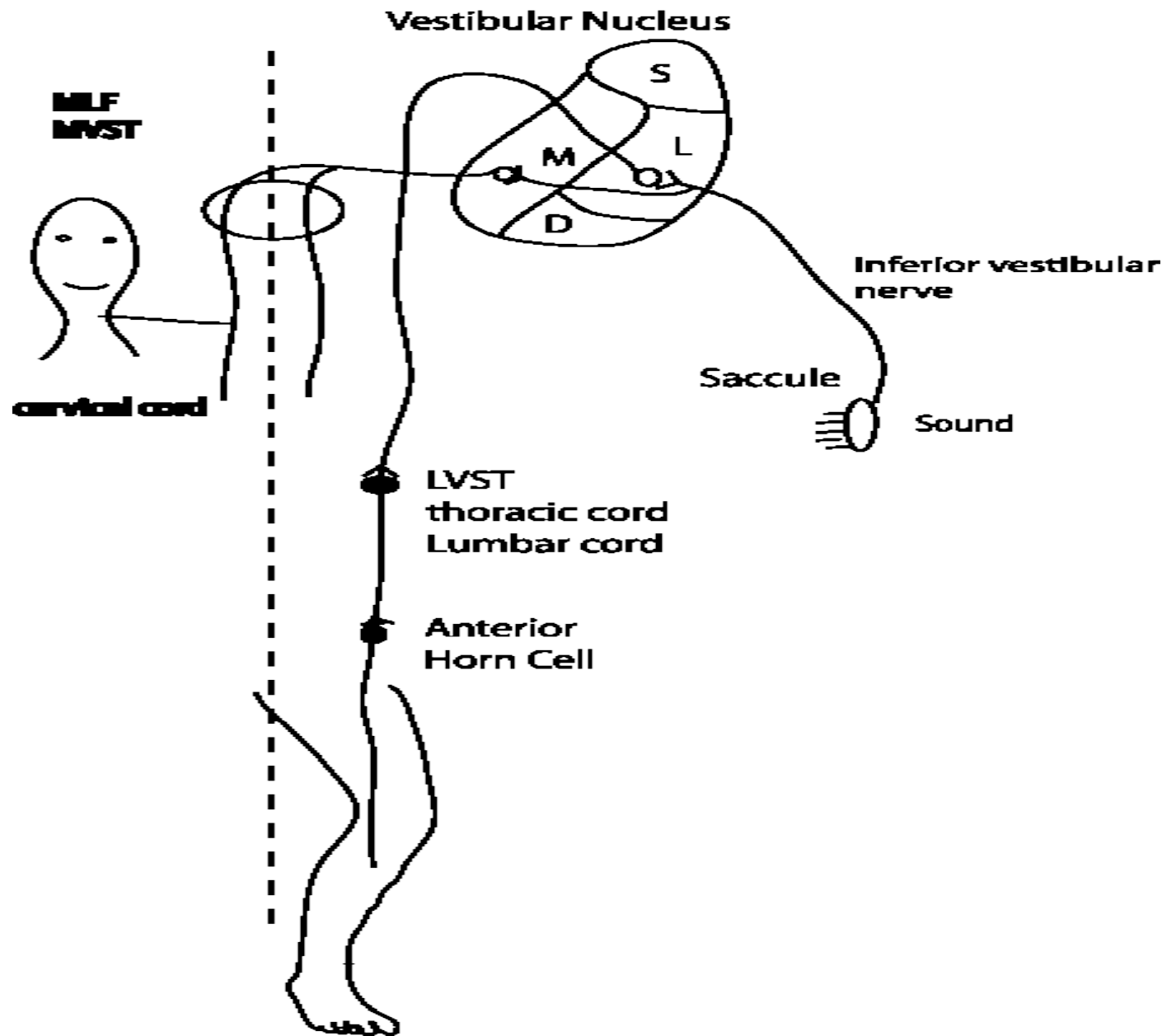
E



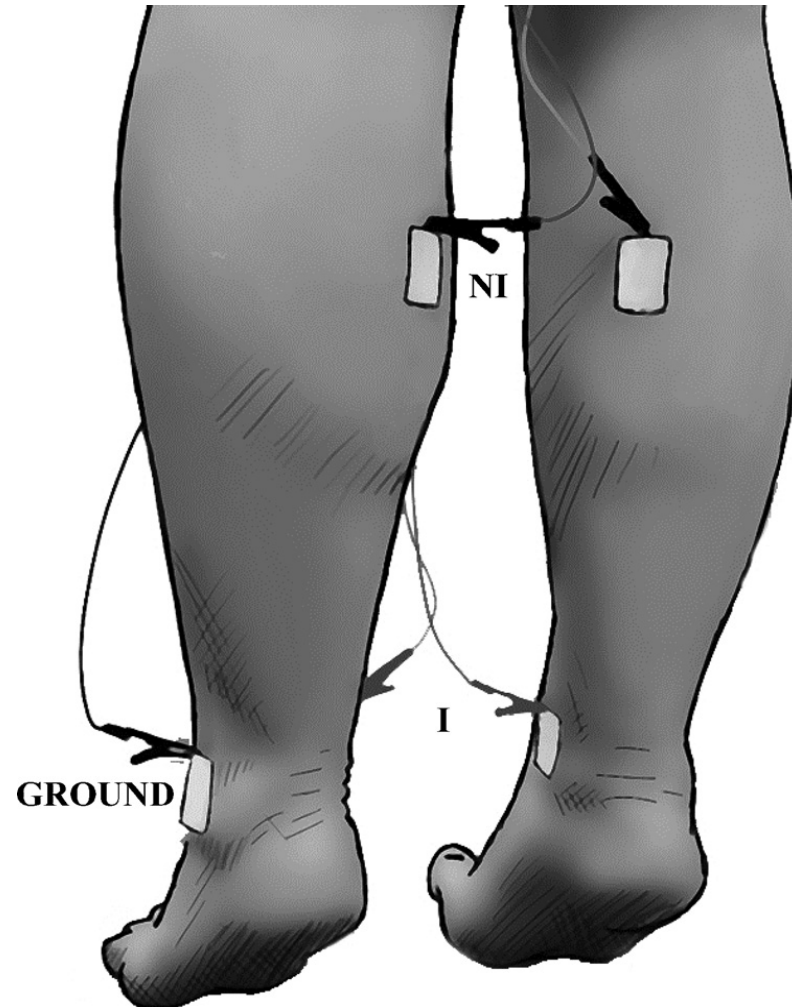
R SCM



VEMP PATHWAY

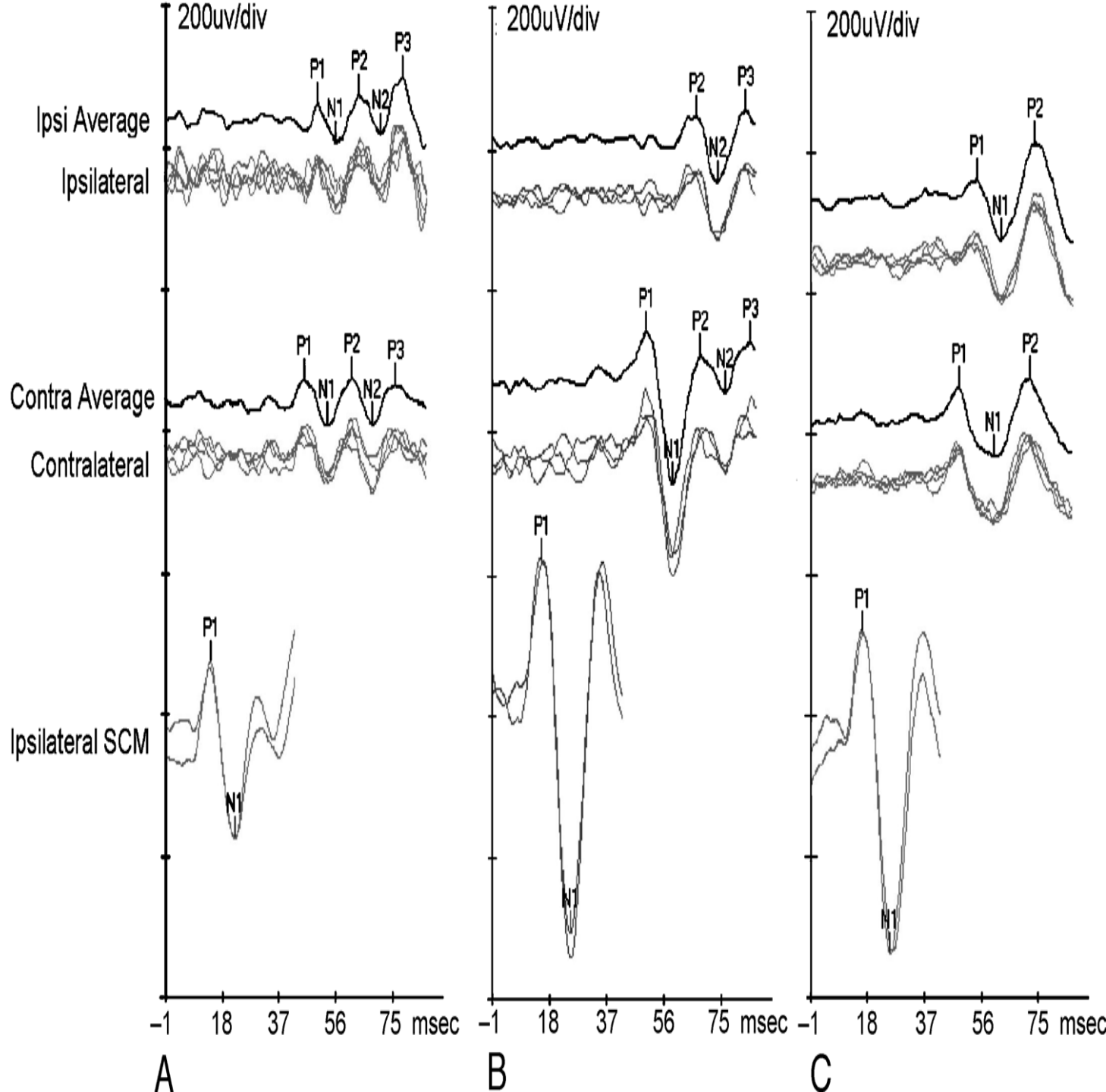


Effect of Electrode placement



GASTROCNEMIUS OF THE LEG

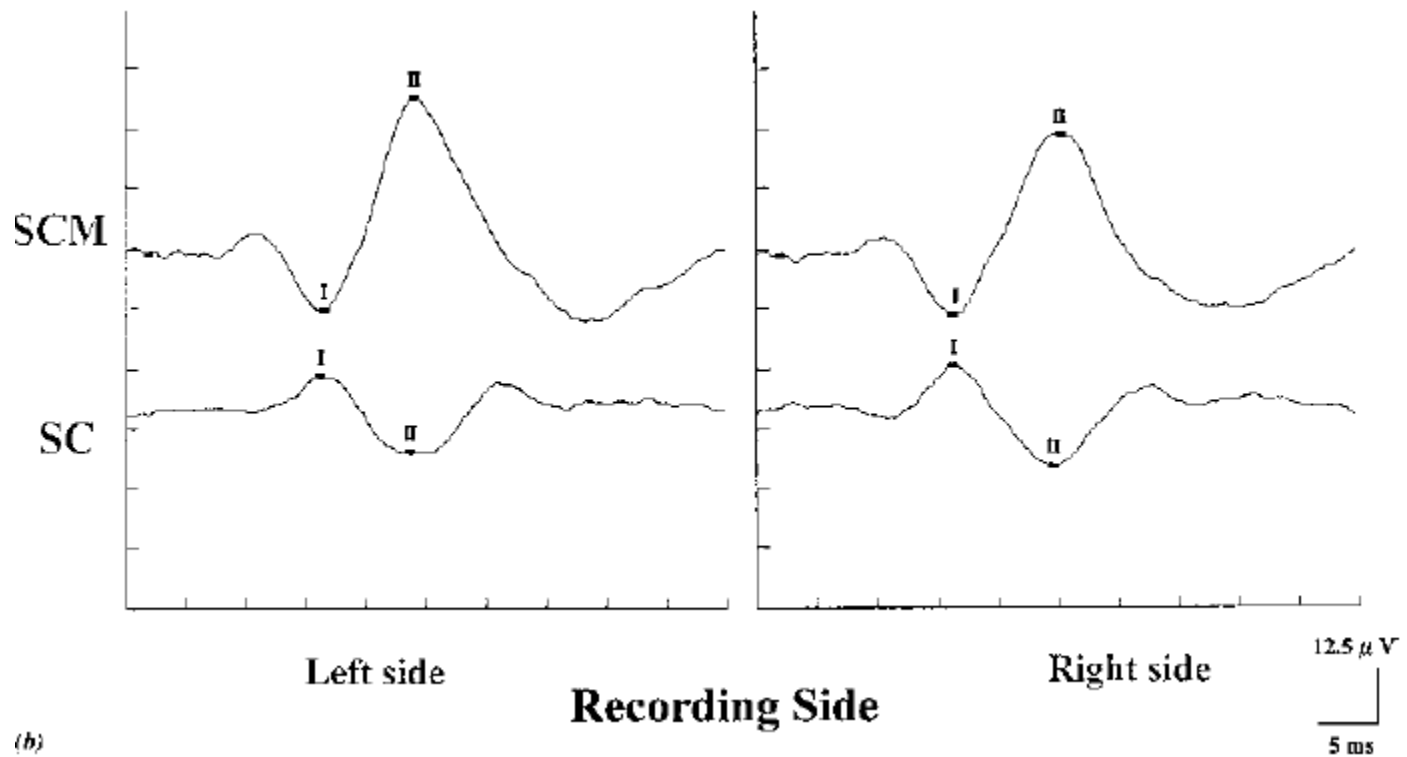
STERNOCLEIDOMASTOID OF THE NECK



Comparison of VEMP from SCM & Splenius muscle (Wu et al. 1999)



Comparison of VEMP from SCM & Splenius muscle

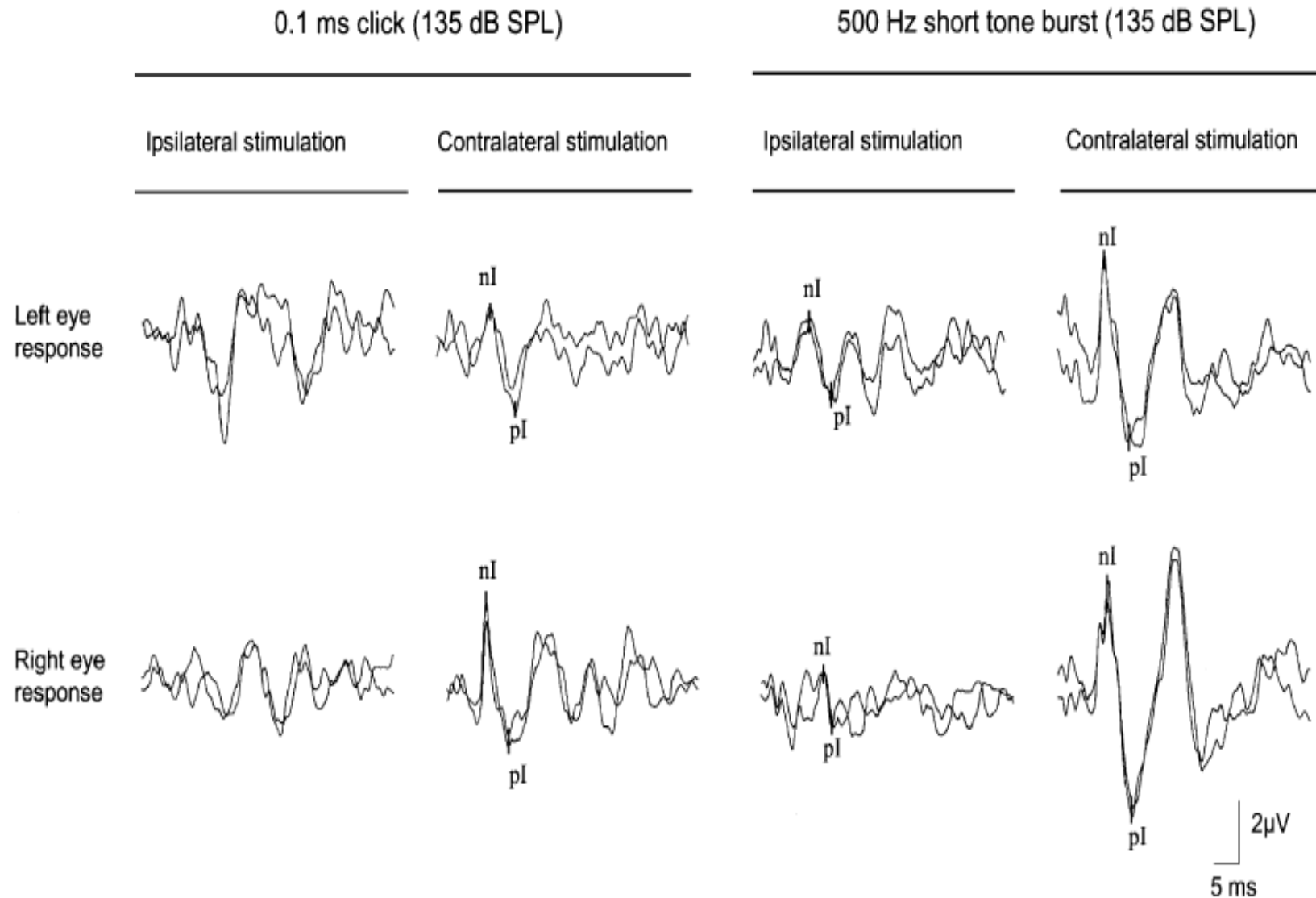


- ❖ Splenius muscle antagonist to SCM
- ❖ Different length of neural pathways or different number of synapses between the excitatory and the inhibitory connections.

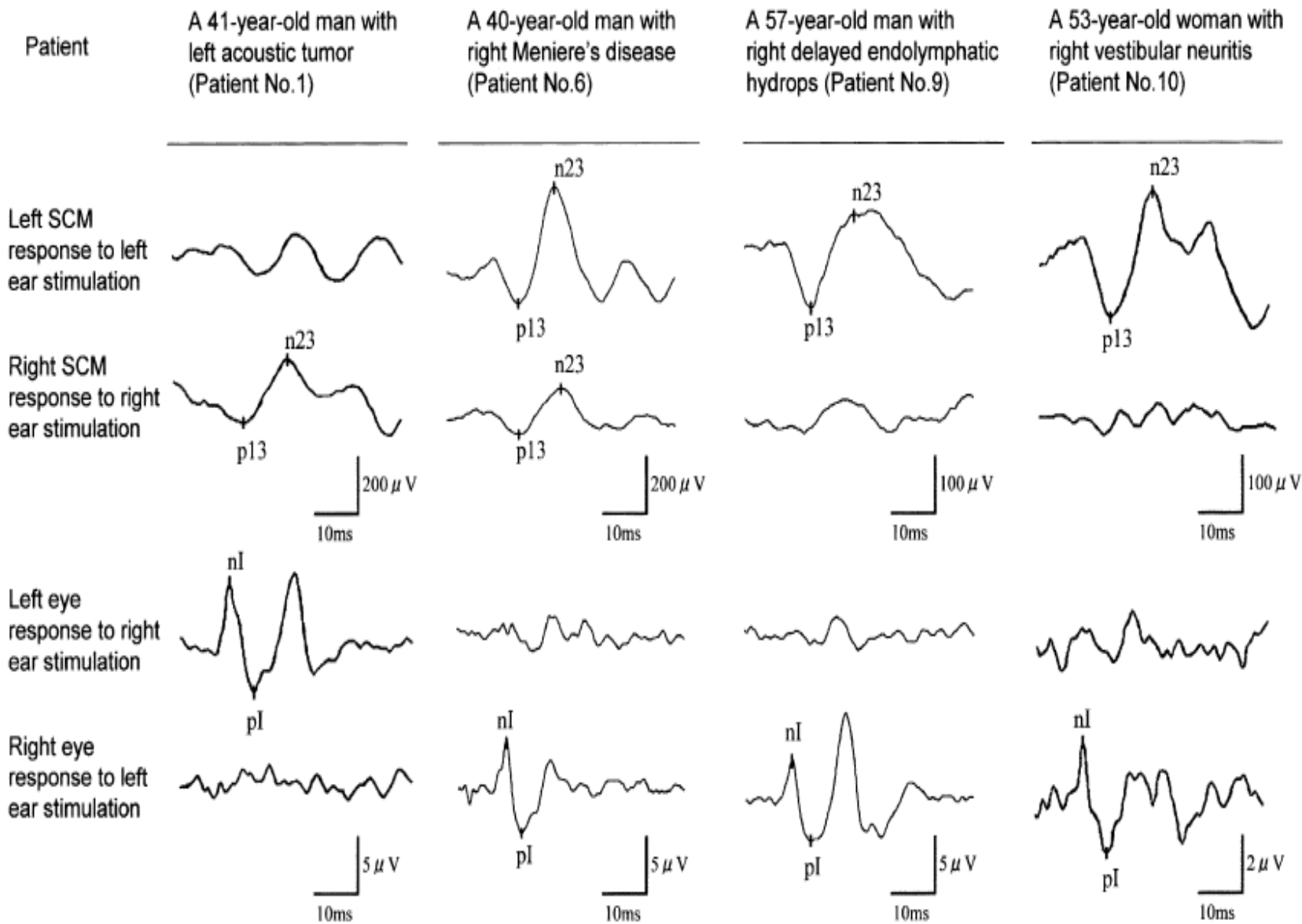
Ocular VEMP

- Can give additional information on Vestibulo-ocular reflexes.
- Recorded From electrodes placed just beneath the eyes.
 - Active electrode: Just inferior to each eye
 - Reference electrode: 1 or 2 cm below the active electrode
 - Ground : Forehead
- Filtered between 5 and 500 Hz.
- Time Window of 50 msec
- total of 100 stimulus

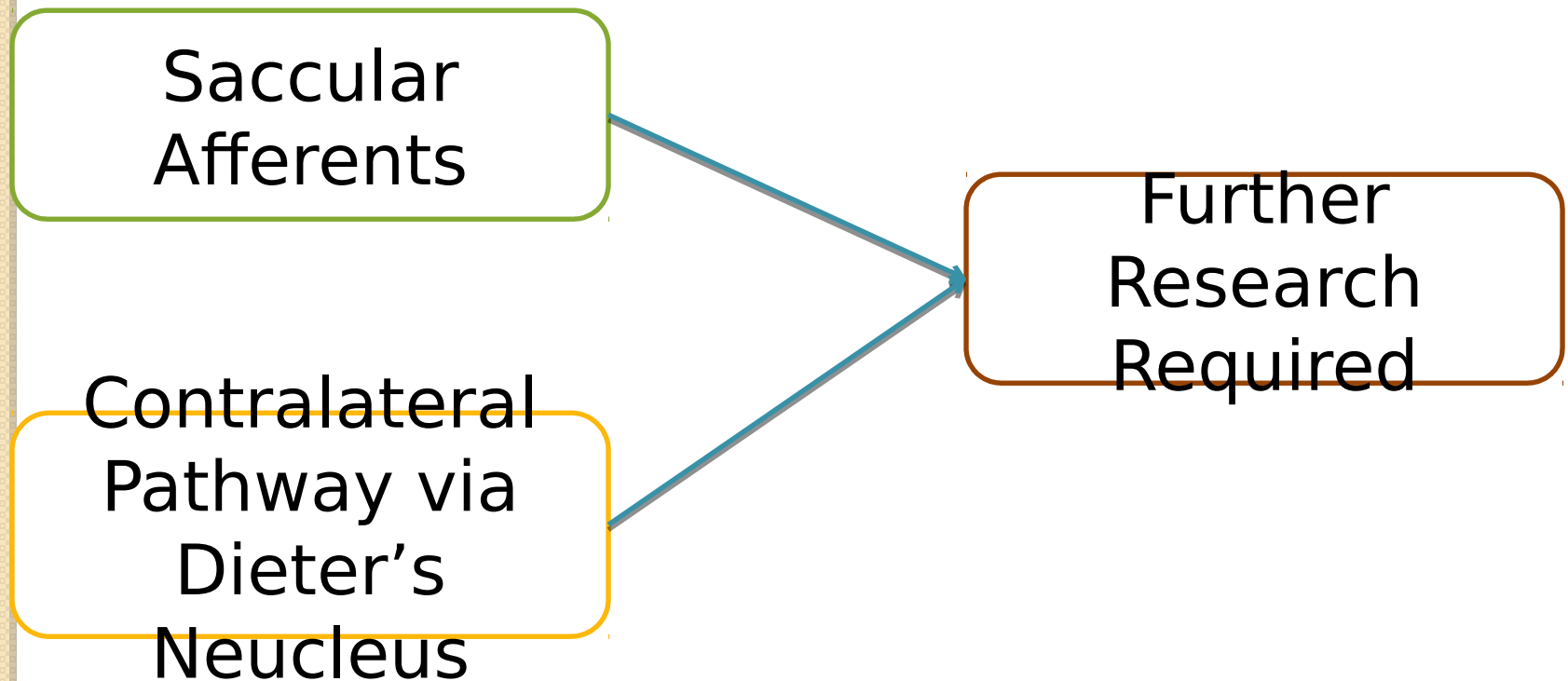
Ocular VEMP



Ocular VEMP contd:



Possible Pathway for Ocular VEMP



Effect of Muscle Contraction

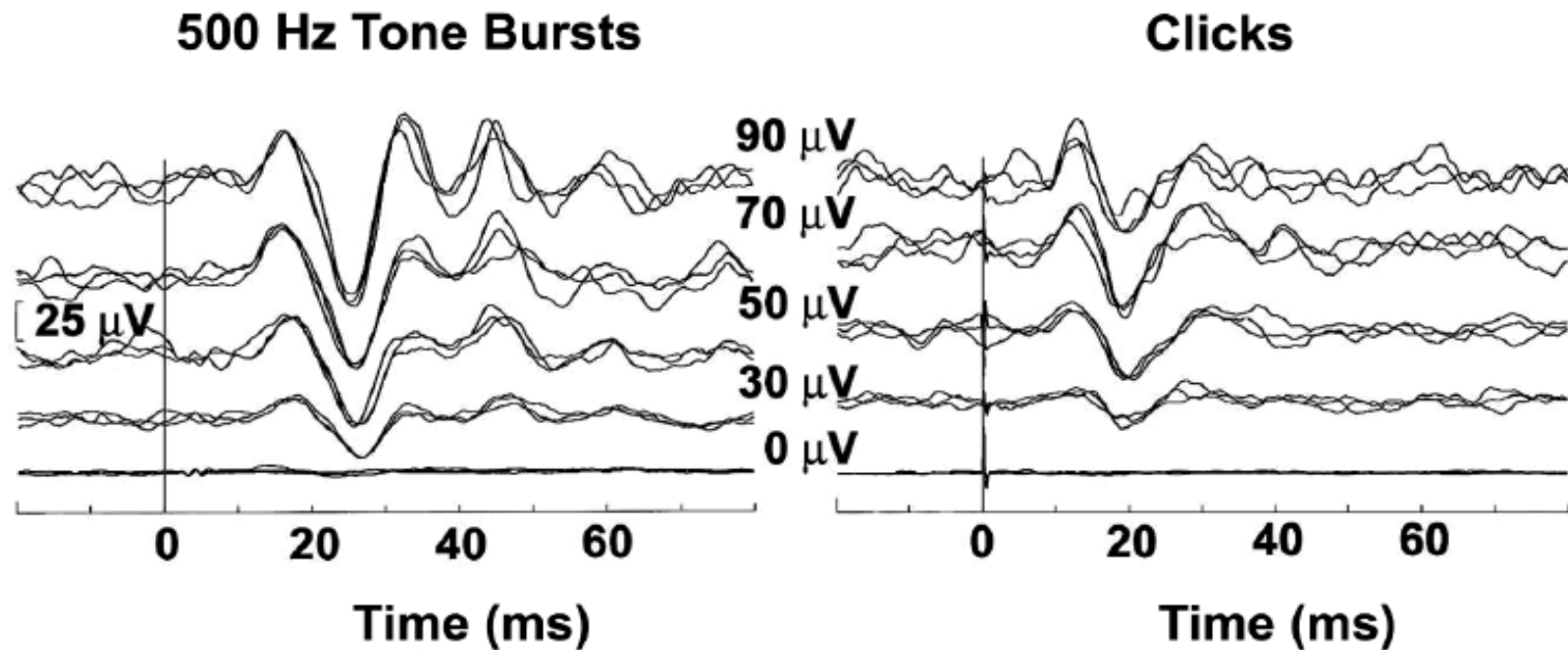
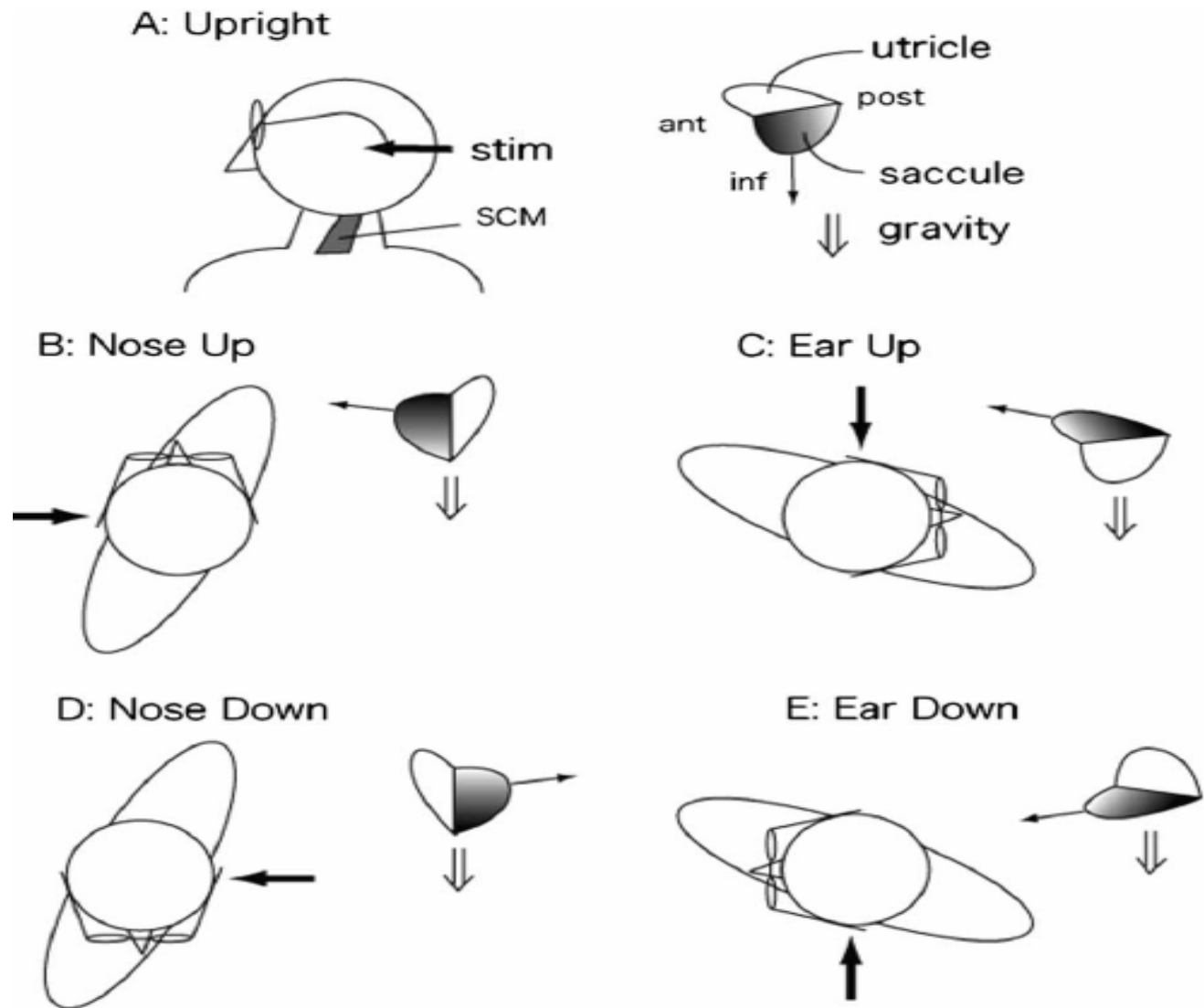


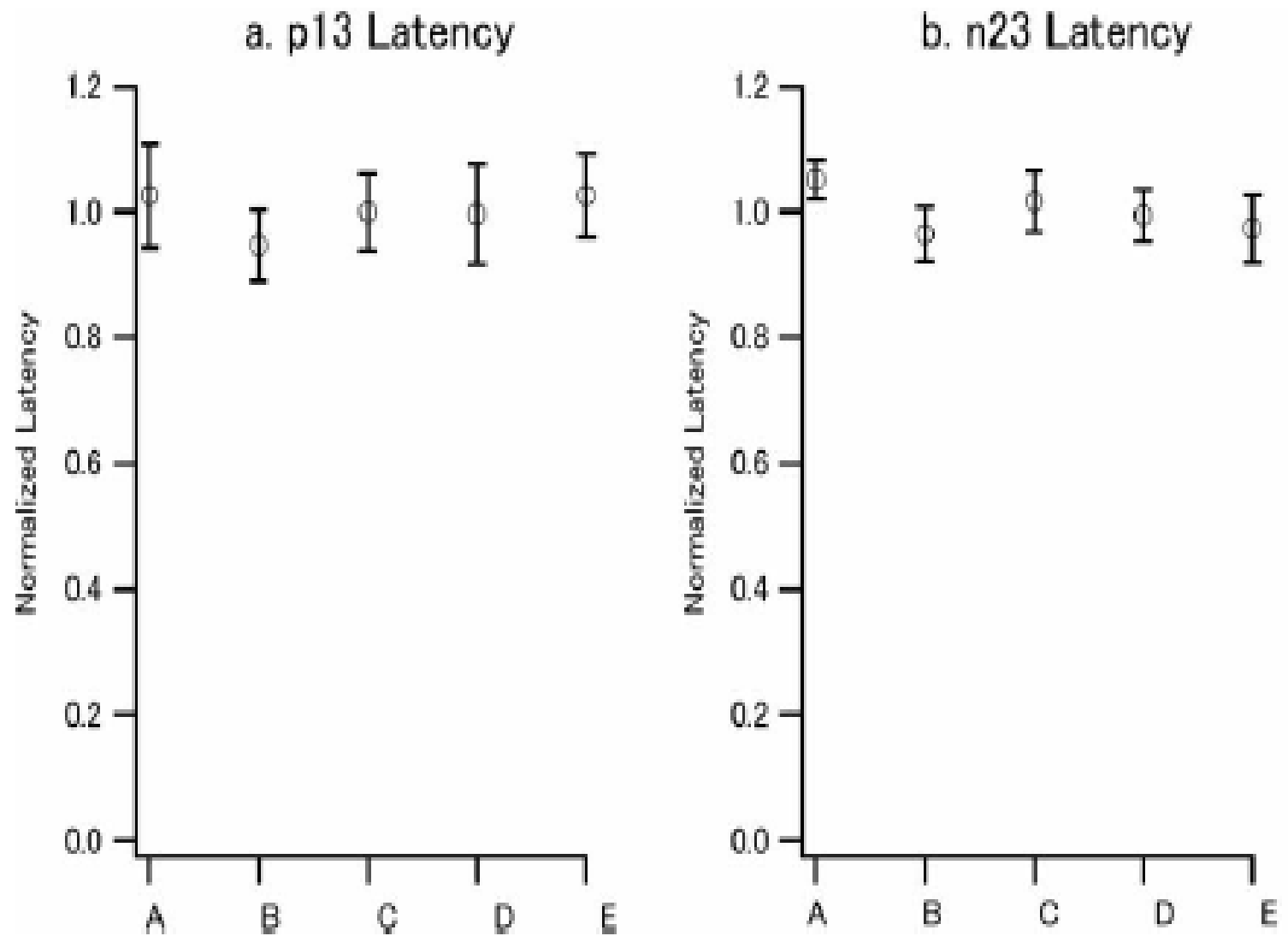
Figure 1.

VEMPs obtained from single subject at each EMG target level and for 500 Hz tone bursts (left) and clicks (right). Target EMG levels are indicated in center of figure.

Effect of different Head Positions on VEMP from SCM(Ito et al.2007)



Head Positions Contd:



Effect of Mode of SCM excitation (Vijay & Basavaraj,2008)

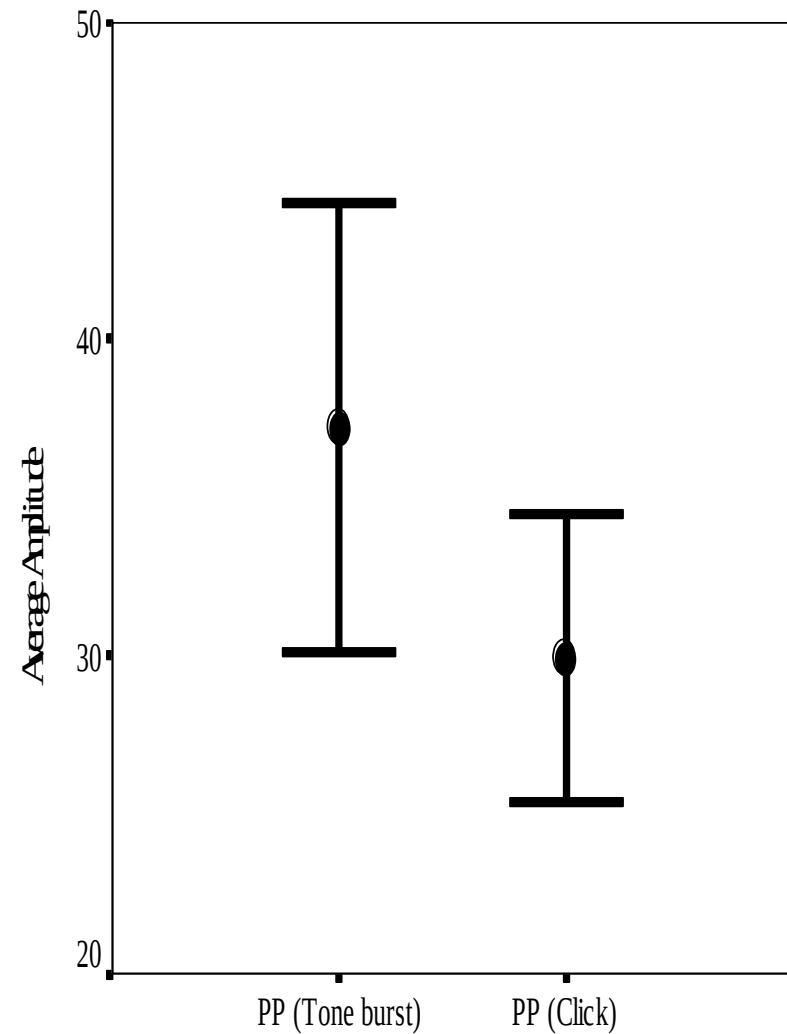
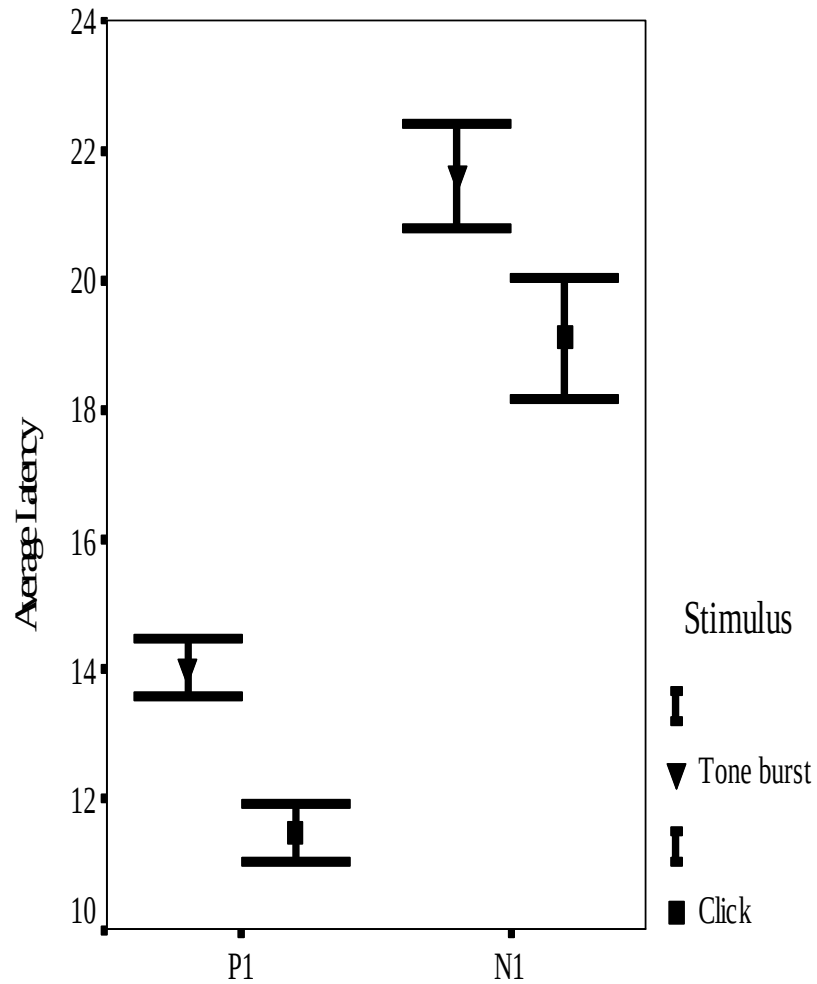
- ❖ Three Body Positions
- ❖ No difference In terms of latency of P13 & N23.
- ❖ No difference in amplitude of P13-N23.
- ❖ No difference b/w males and females.

Click v/s Tone Burst

- Controversy over the use of Click or Tone burst
- Kumar et al(2003) & Cheng et al (2003) reported Click as a better stimulus as the response rate was higher with click stimulus.
- Akin et al(2003) reported Tone burst as a better stimulus.
- Picciotti et al (2007) reports no difference in terms of latency but amplitude more with Tone Burst

Click v/s Tone Burst

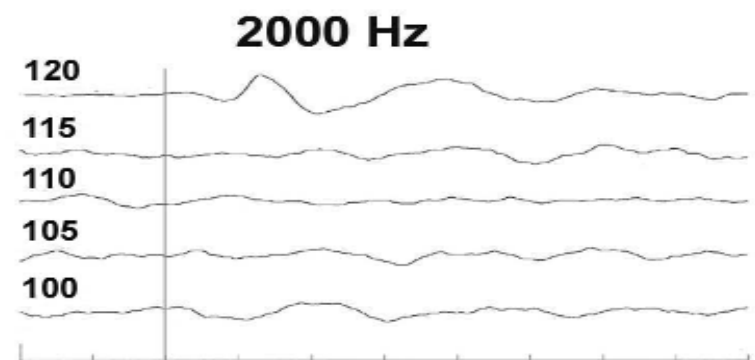
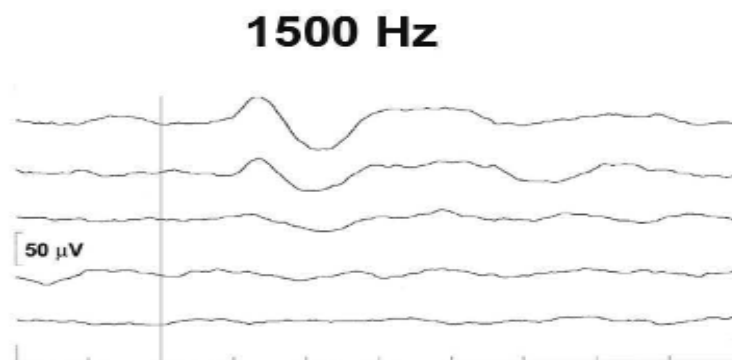
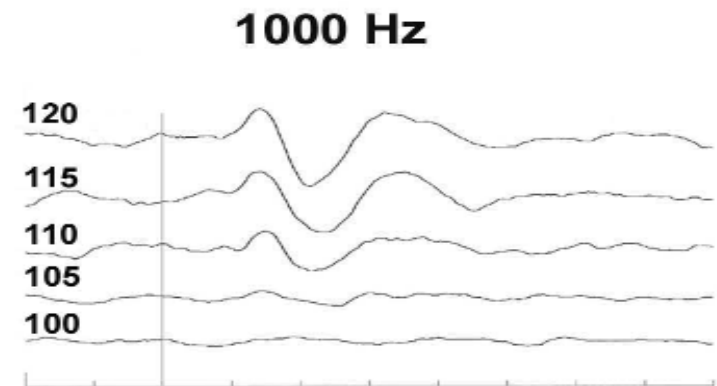
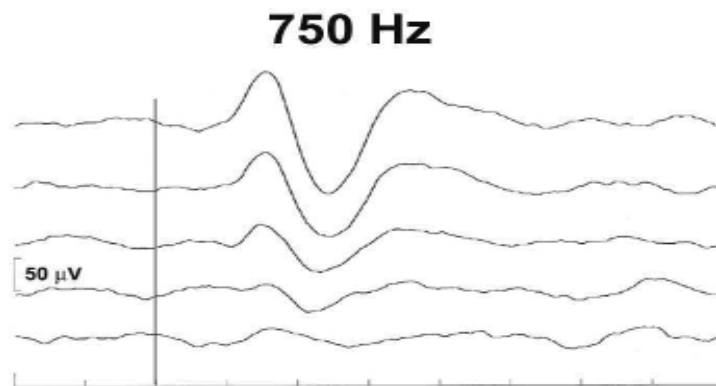
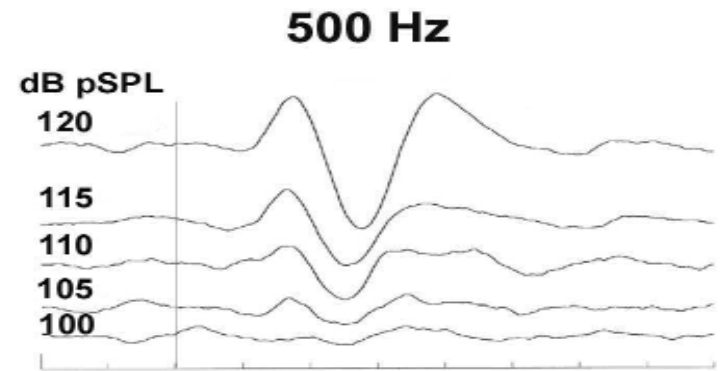
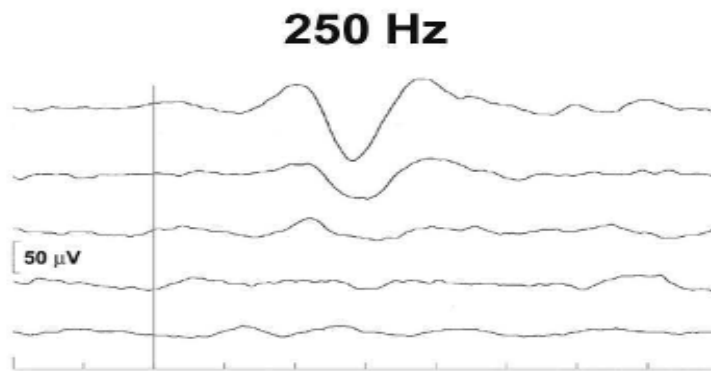
(Kumar, Sinha & Barman, 2005)



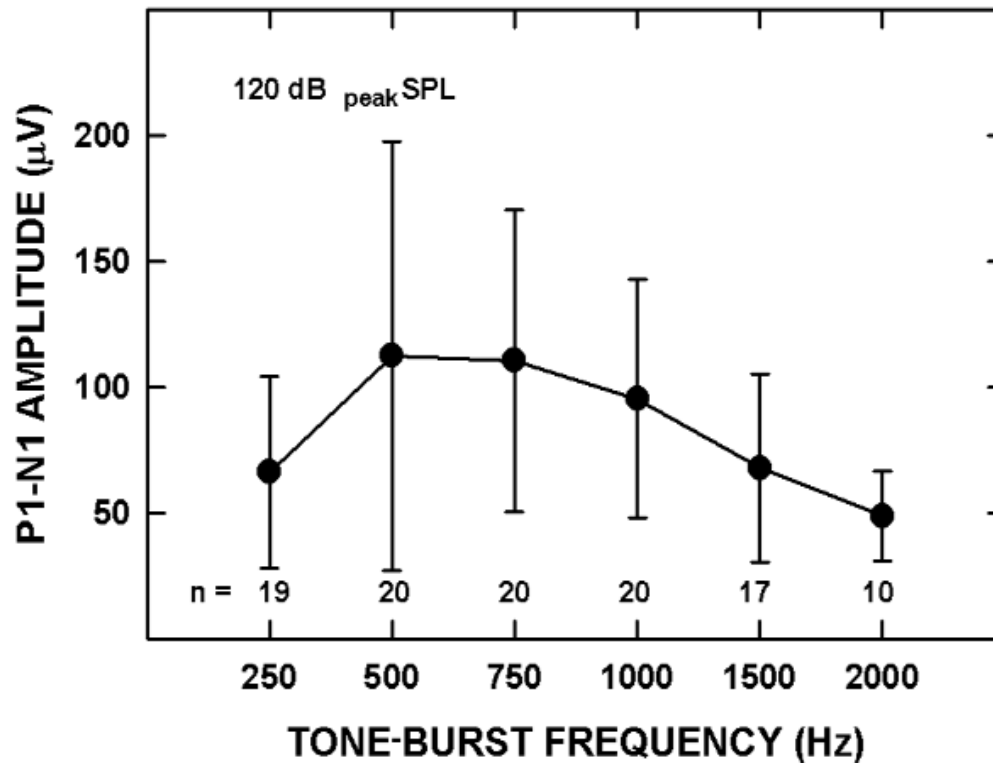
The effects of Logon versus Click on vestibular evoked myogenic potentials (Trivelli et al,2008)

	P13 Latency	N23 Latency	P13-N23 amplitude
Air conducted Click	11.45	21.12	96.80
Air Conducted Logon	15.58	26.12	129.27
Bone Conducted Click	12.93	21.46	45.05
Bone conducted Logon	16.34	25.89	83.14

Effect of Tone Burst Frequency (Akin et al. 2003)

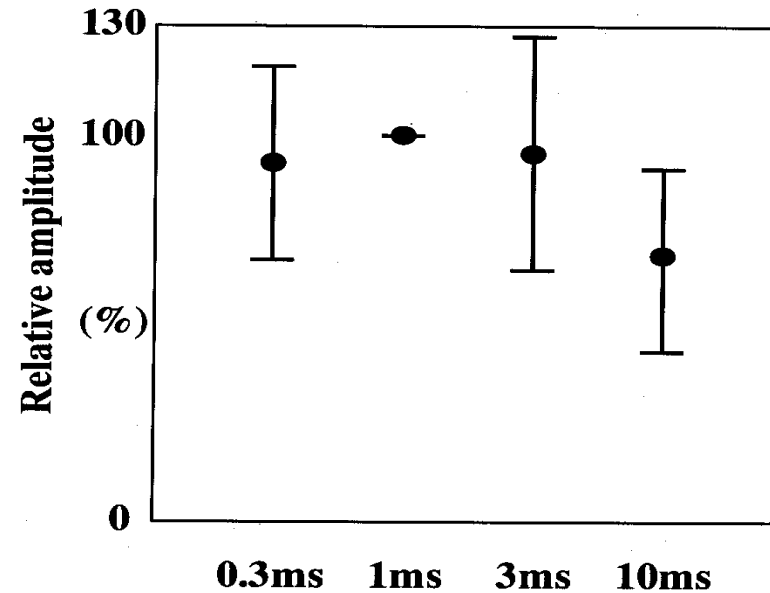
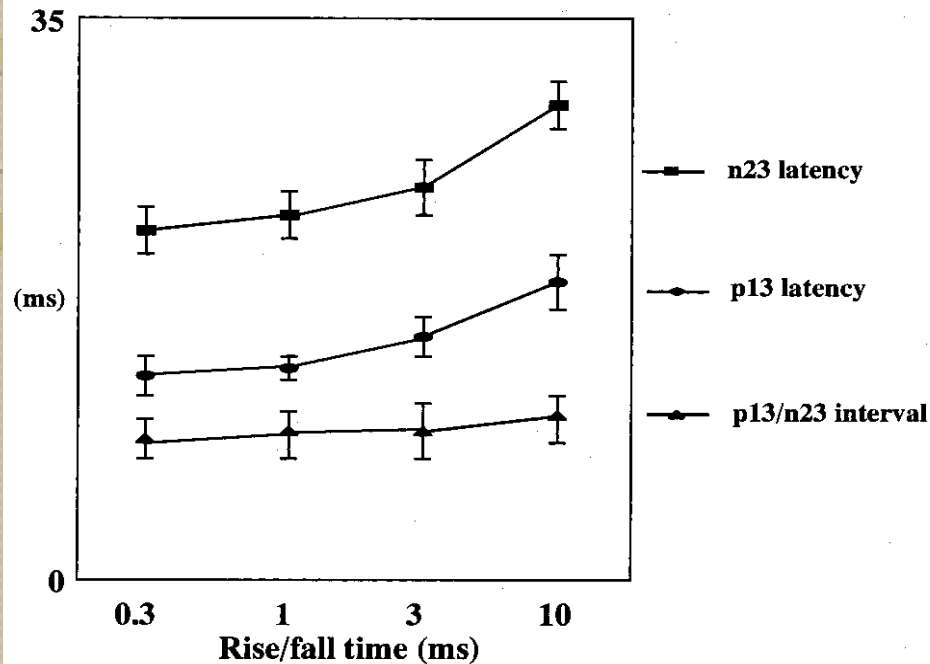


Amplitude of VEMP with different Tone Burst Frequencies (Akin et al. 2003)



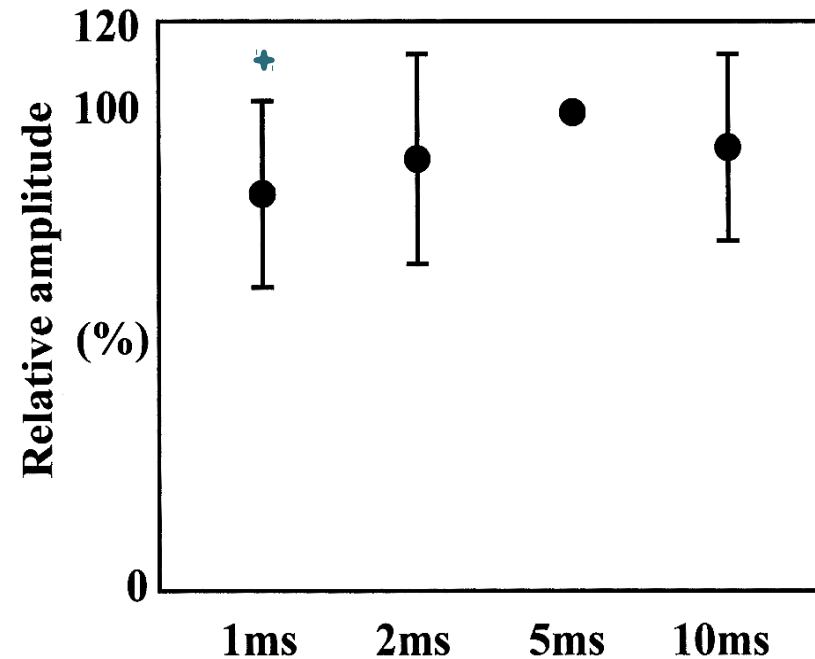
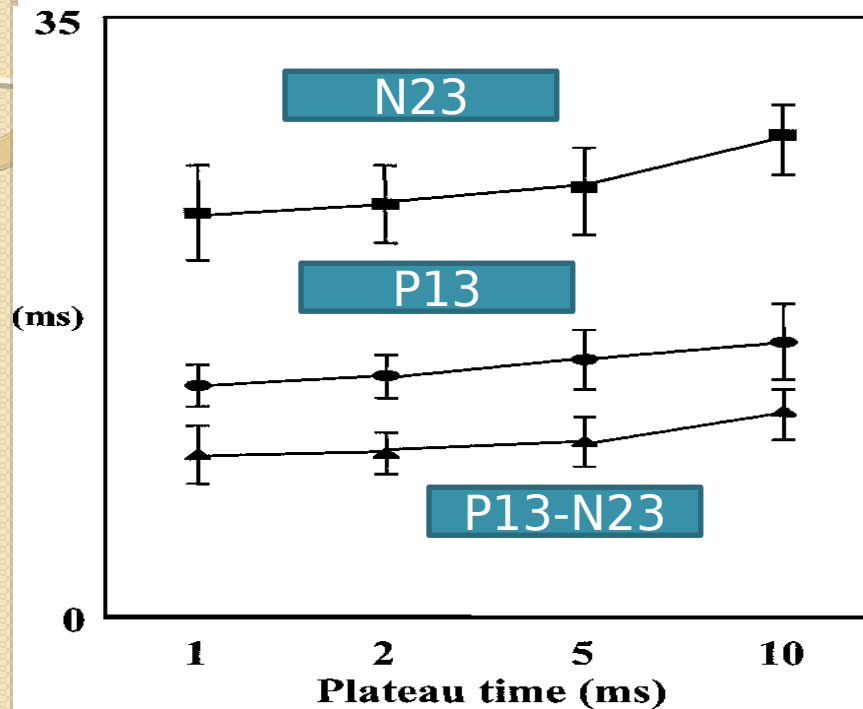
- Utricle band-pass tuning with best frequency between 400 and 800 Hz (Todd, 2009)
- Saccule responds to a well-defined frequency tuning 300 to 350 Hz (Todd, 2000).

Rise time/Fall time of Toneburst



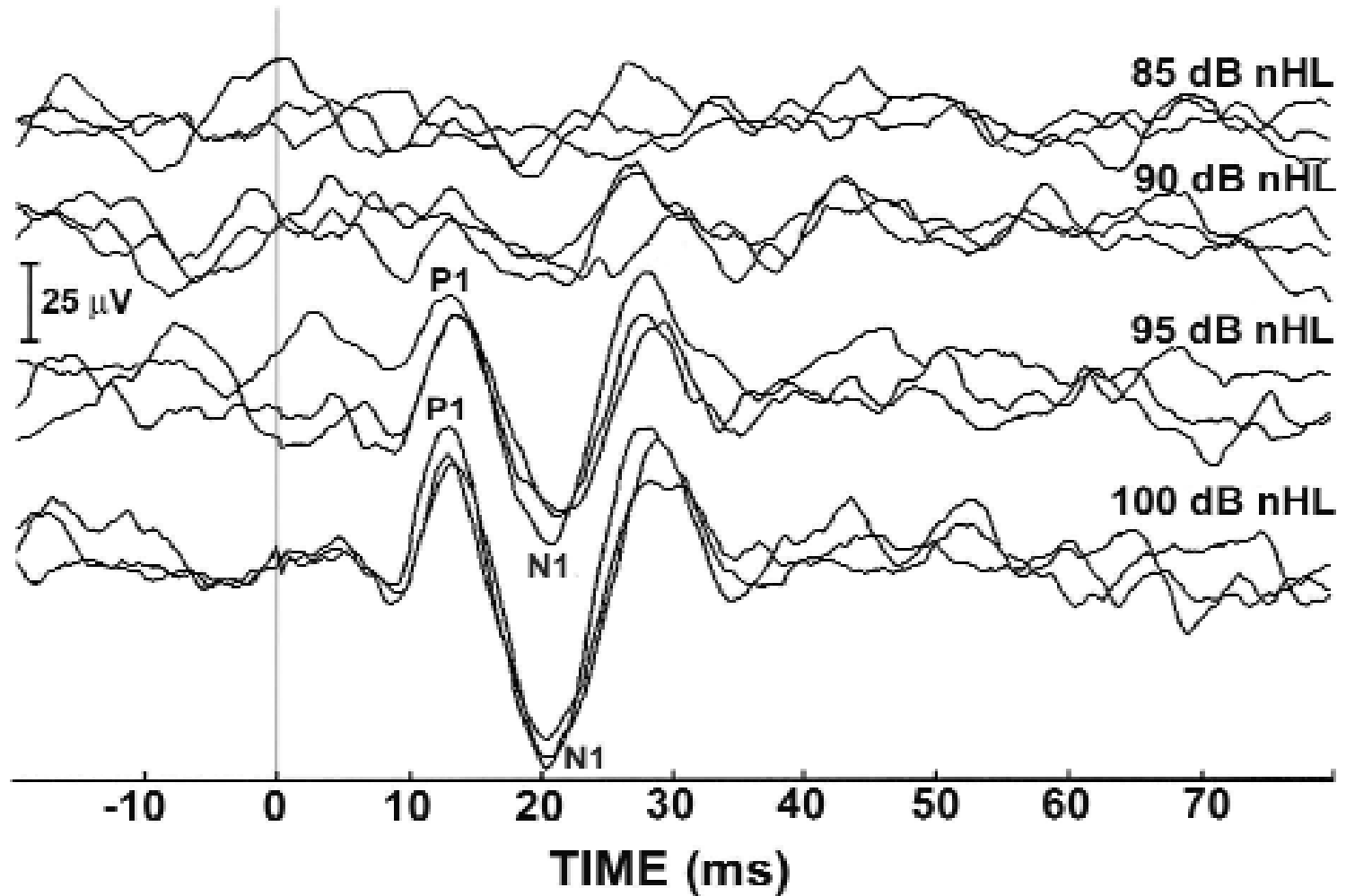
- ❖ Time to maximal stimuli was lengthened
 - ❖ 1 msec less variations
- ❖ Stapedial reflexes which has a latency of 4.5 to 10 msec

Tone Burst Plateau Time

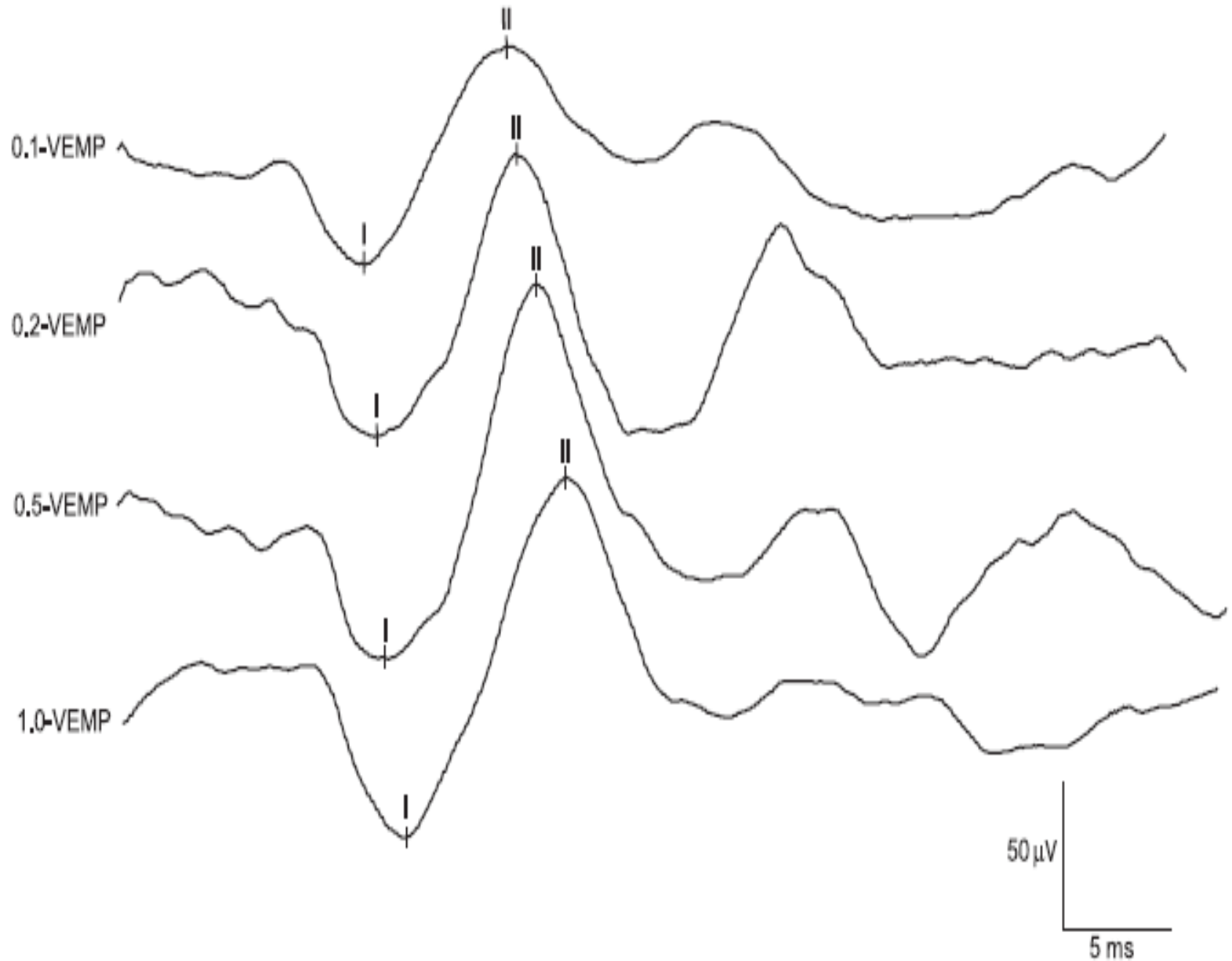


- 2msec plateau caused smallest difference
- Increased duration of the plateau time used to generate the VEMP responses.
- Neurons may have double or triple firing to one tone burst
- 10 msec difference due to stapedial reflexes.

Effect of Click Stimulus level (Akin et al.2003)



Effect of Click duration on VEMP

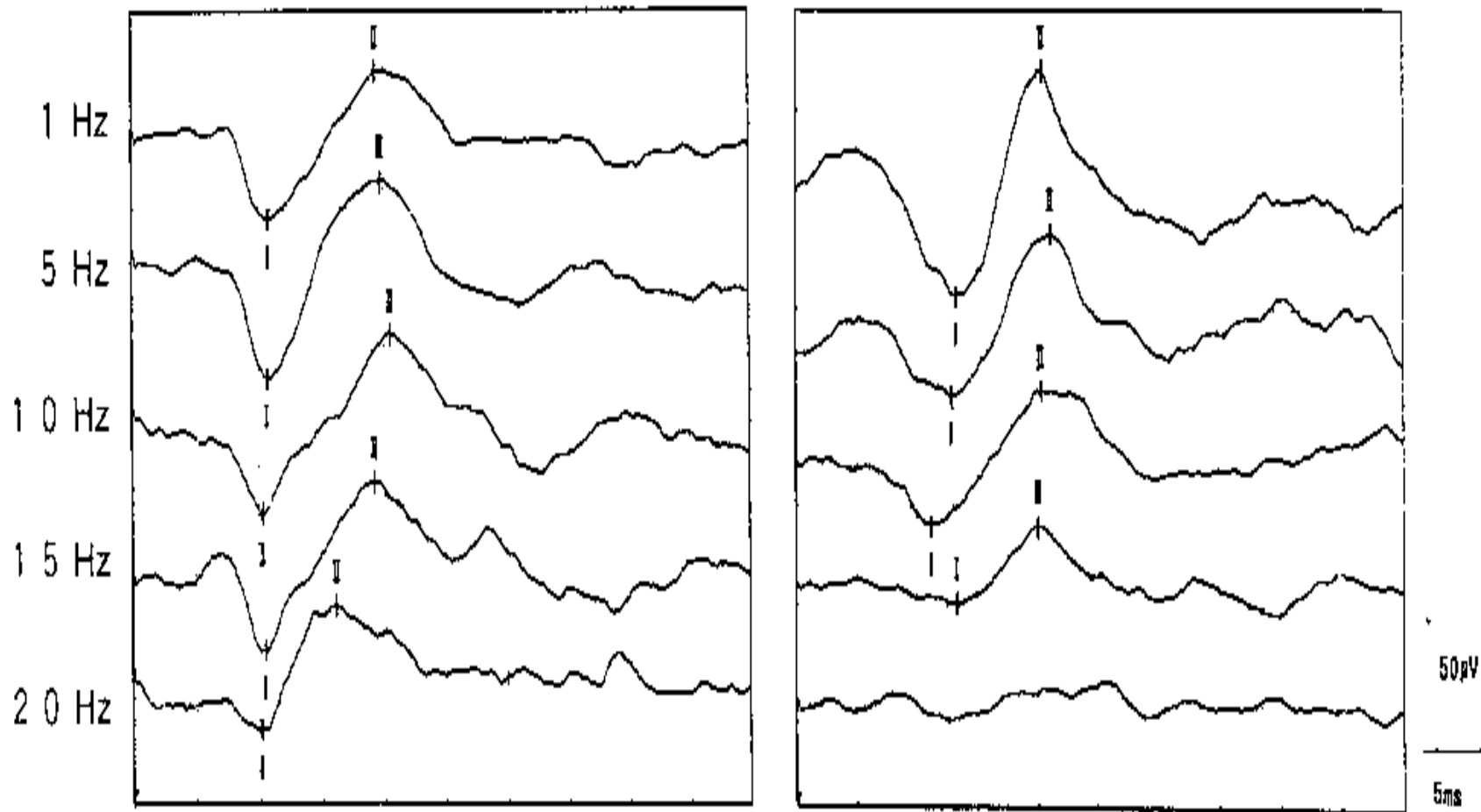


Effect of Click duration

	P13 latency	N23 latency	amplitude	Response rate
0.1 msec click	11.31±1.1 3	18.44±1.33	111.38±47. 56	94%
0.2 msec click	11.52±1.1 2	18.90±1.30	164.75±70. 50	100%
0.5 msec click	11.89±1.1 2	19.20±1.32	193.88±79. 50	100%
1.0 msec click	12.44±1.3 8	19.65±1.66	192.21±65. 64	100%

Data Taken from Huang et al.(2005)

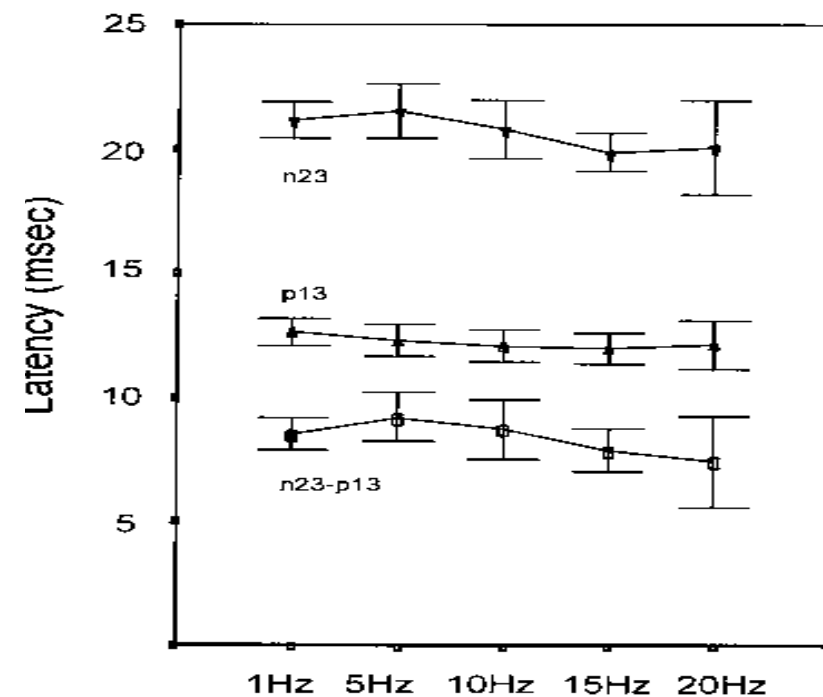
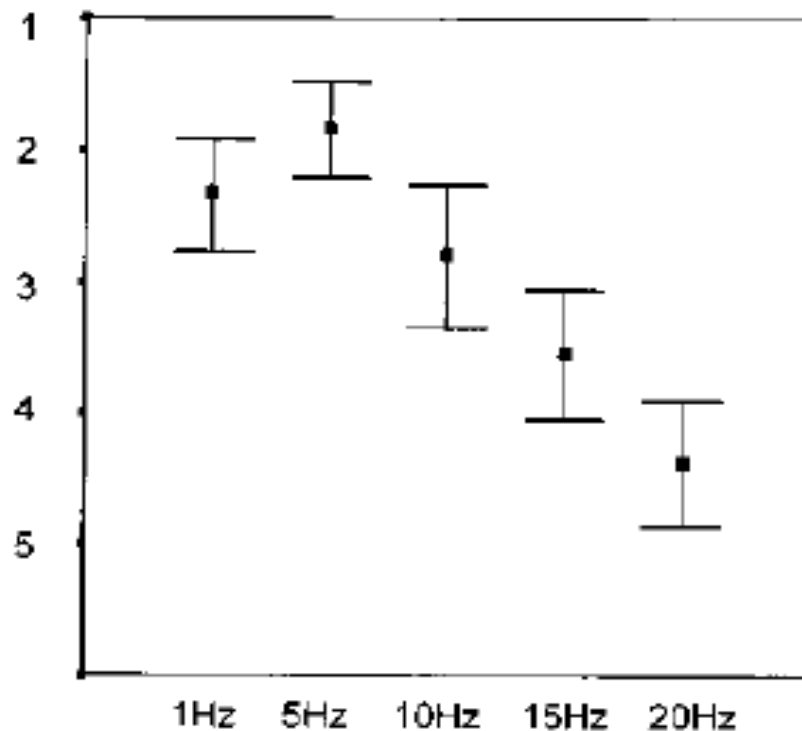
The Effect of Repetition Rate on Vestibular Evoked Myogenic Potential



(a)

(b)

The Effect of Repetition Rate on Vestibular Evoked Myogenic Potential



- ❖ Response rate with 1 Hz, 5 Hz, 10 Hz was 100 % whereas 96% at 15 Hz & 63 % at 20 Hz
- ❖ Adaptation of the vestibular end organs may lead to decrease in amplitude.

Effect of Eye Position (Sandhu et al.2009)

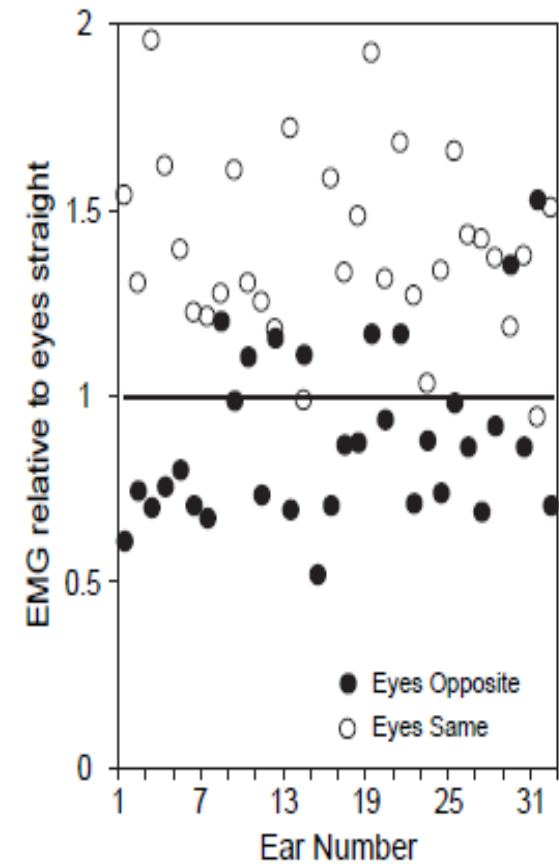
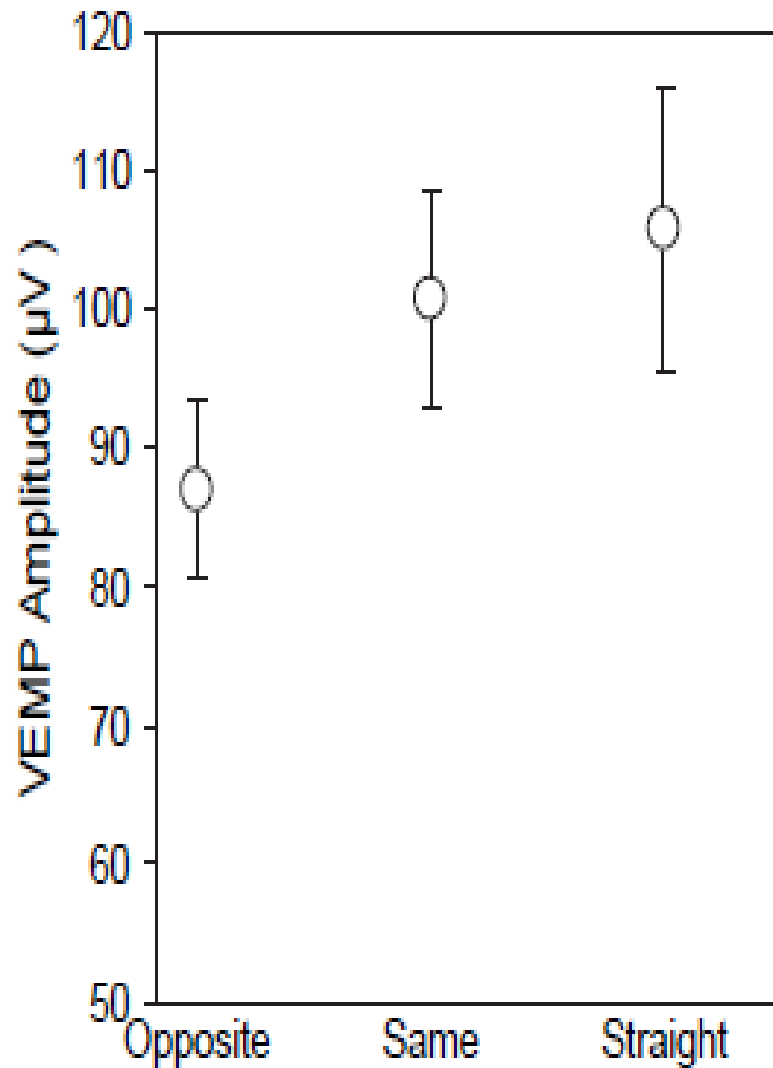
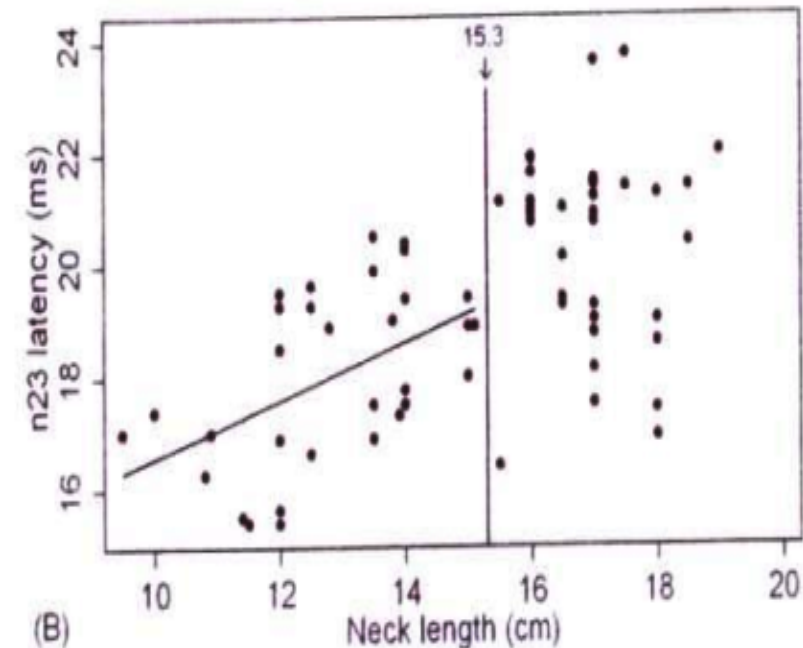
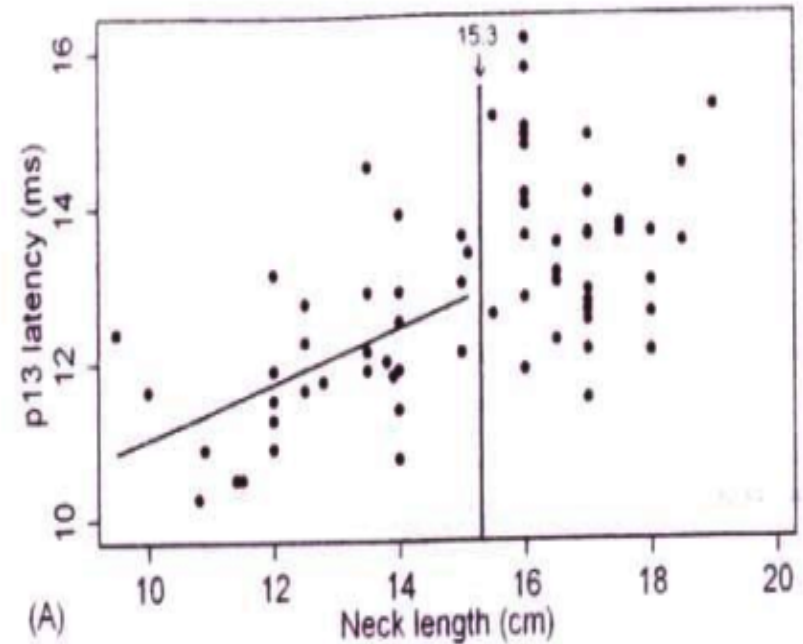


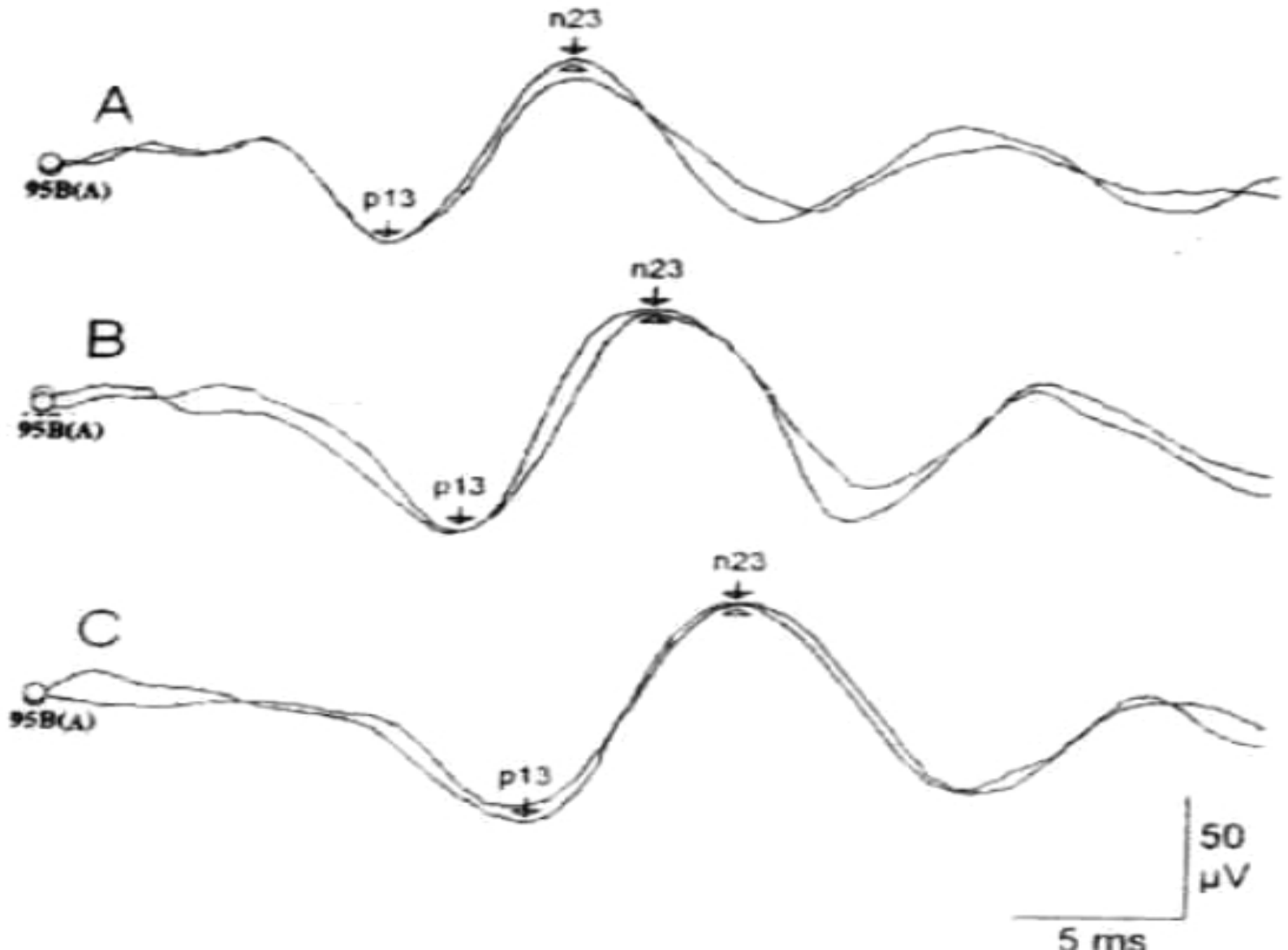
Figure 3. Mean rectified background EMG values for eyes opposite to and in same direction as the direction of head turn relative to eyes straight ahead.

Effect of age or Structural variations(Wang et.al.2008)

	P13 latency(msec)	N23 latency(ms ec)
Age (yrs)		
5-12	10.1~13.7	14.8~20.8
13-18	11.5~13.9	15.2~22.8
26-38	11.9~15.9	18.7~23.1
Neck Length(cm)		
<15.3	10.0~14.0	15.0~21.0
>15.3	11.4~15.8	17.0~23.8
Body height(cm)		
<150	10.1~13.7	14.9~20.9
>150	11.3~15.7	16.9~23.7
Body weight(kg)		
<43.5	9.9~13.9	14.9~20.1
>43.5	11.2~15.6	17.1~23.5

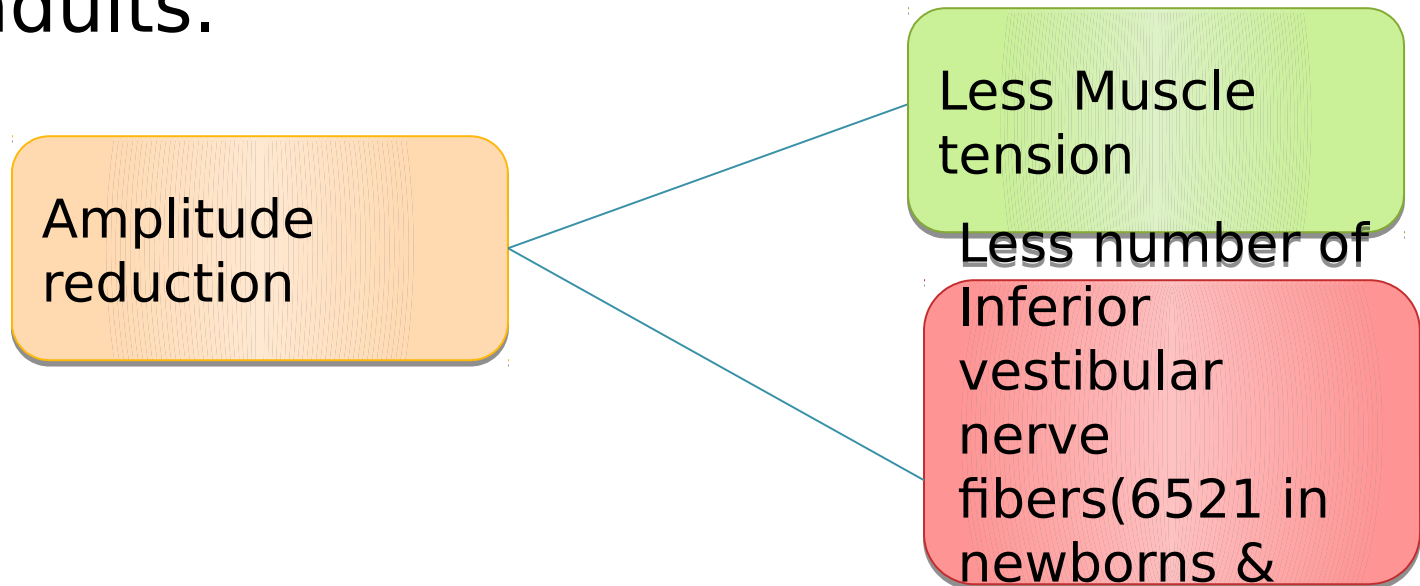


Effect of age or Structural variations (Wang et.al.2008)



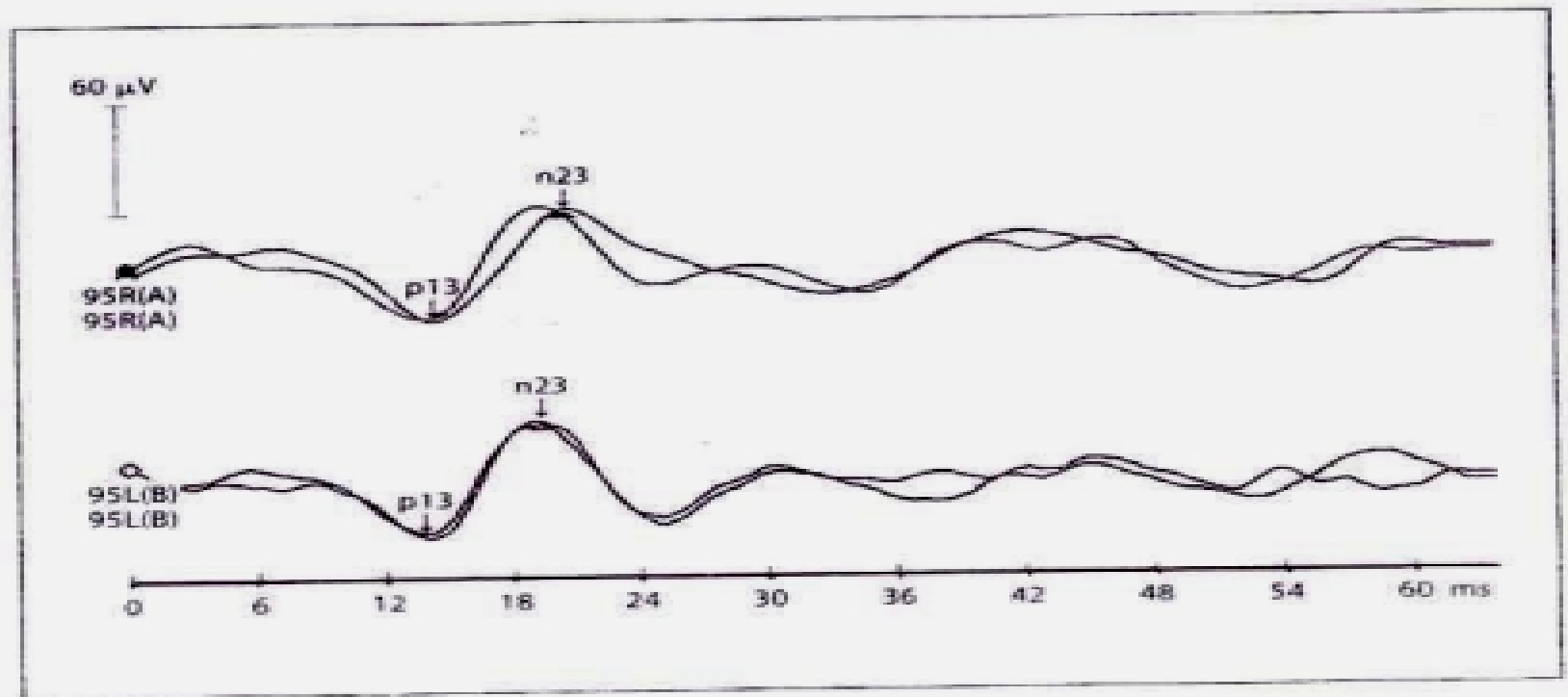
VEMPs in Newborns

- In newborns latency is delayed and amplitude is reduced compared to the adults.



Vestibulo collic reflex normalizes by 2 months of age and matures further till 2 yrs of life(Fife et al.2000). Investigating VEMPs in Newborn may help to study the sacculocollic reflex at birth

VEMPs in Newborns contd:



	Response Rate	P13 latency	N23 latency	amplitude (μv)	P13-N23 interval
Newborns	30(75%)	17.7 ± 4.1	23.9±4.5	27.5	6.3± 1.5
Adults	34(85%)	14.5±1.3	22.3±	59.1	7.8± 1.3

With courtesy from Chen et al. 2007

Development of VEMPs in Preterm Baby

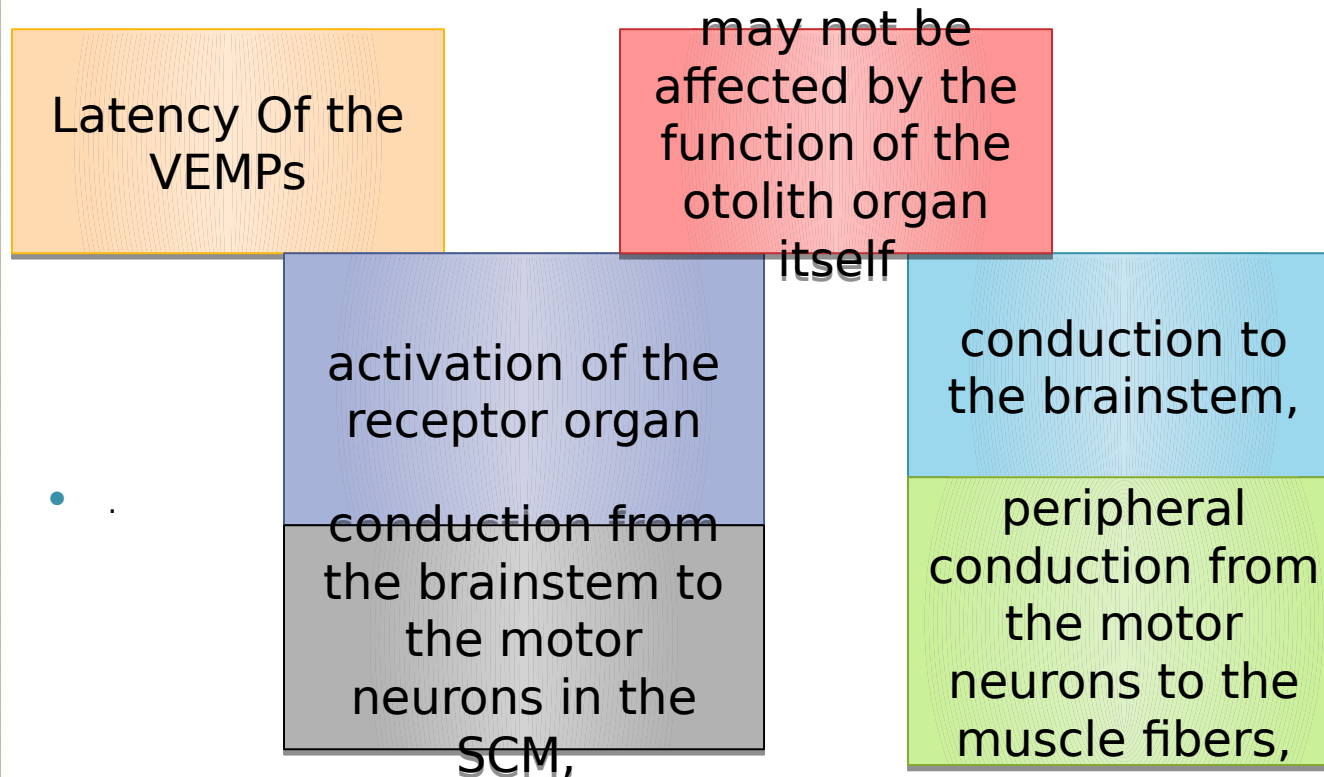
- Vestibular nerve myelination begins at the 20th Fetal week and myelinated at birth(Barkovich,2005).
- Vestibular pathway may be completely responsive but end organs may not be

	Preterm	Fullterm
Response rate	26 %	72%
P13 latency	14.2± 0.4	13.3±0.4
N23 latency	20.1±1.3	18.3±0.6
P13-N23 amplitude (μv)	29.8±12.9	27.5±13.7

Data with courtesy from Wang et al. 2008.

Effect of Aging on VEMPs

- Reduction in the amplitude as well as latency
- Decreases in the number of vestibular hair cells, Scarpa's ganglion cells, and cells of the vestibular brainstem during the aging process.



Effect of Aging on VEMPs

Table II. Parameters of VEMP: latencies, amplitude, and VEMP asymmetry in subjects sorted by age group.

Age group* (number of ears)	Latency (ms)		Interlatency (ms)	Amplitude (mV)	Vemp asymmetry
	p13	n23			
I (26)	12.6±1.9	18.5±1.4	5.9±1.6	24.1±6.2	-0.2±7.1
II (34)	13.1±1.6	18.8±1.8	5.6±1.4	19.4±8.6	5.1±10.9
III (28)	13.1±1.6	18.3±1.6	5.2±1.0	16.3±5.5	4.6±6.8
IV (26)	12.9±2.1	18.2±2.1	5.3±1.2	15.1±6.1	-2.1±13.8
V (38)	14.1±2.4	19.8±2.5	5.7±1.3	14.8±6.9	-3.4±9.3
VI (28)	15.8±2.7	22.0±2.7	6.2±1.6	13.8±5.1	-4.1±13.2
VII (14)	15.6±2.2	21.5±3.0	5.9±1.7	14.9±5.8	1.4±8.1
Total (197)	13.8±2.4	19.5±2.6	5.7±1.4	17.0±7.3	0.1±10.8

*Group I, 10–19 years ($n=13$); group II, 20–29 years ($n=17$); group III, 30–39 years ($n=14$); group IV, 40–49 years ($n=13$); group V, 50–59 years ($n=19$); group VI, 60–69 years ($n=14$); and group VII, 70–79 years ($n=7$).

Things to remember

- ❖ Get UCL of all the person
- ❖ Once u have got the VEMP at higher intensity you should repeat it at 80 dB.
- ❖ Rule out middle ear pathology
- ❖ Nearly all VEMP problems are caused by operator error
- ❖ Assuring neck muscle activation is the biggest problem
- ❖ Patient not co-operative
- ❖ Sound not getting to the ears



Clinical Applications of Vestibular Evoked Myogenic Potentials (VEMPs)

Agenda of Presentation

- *Meniere's Disease & Related disorder*
- *Vestibular Neuritis*
- *Other peripheral vestibulopathy*
- *Idiopathic sudden sensorineural hearing loss with Vertigo*
- *Superior Canal Dehiscence Syndrome*
- *Migraine associated vertigo and VEMP*
- *Acoustic Neuroma*
- *Noise Induced Hearing Loss*
- *Auditory Neuropathy/Dys-Synchrony*
- *Neuro-otological applications of VEMP during Infancy & Childhood*
- *Aging & VEMP responses*

Meniere's Disease

- ✓ recurrent vertigo attacks
- ✓ Fluctuating Hearing loss
- ✓ Tinnitus
- ✓ Sensation of aural fullness

Findings in Meniere's Disease

- Overall incidence of abnormal VEMP is 58%
- Most of the cases shows absent or decreased VEMP
- Latency remains to be normal
- Elevated thresholds of VEMP
- Shift of frequency tuning of VEMP

Meniere's Disease stage and VEMP

Total no. of patients	Stage of M.D	Normal VEMP	Abnormal/ Absent VEMP
6	I	5	1
12	II	4	8
17	III	7	10
5	IV	2	3

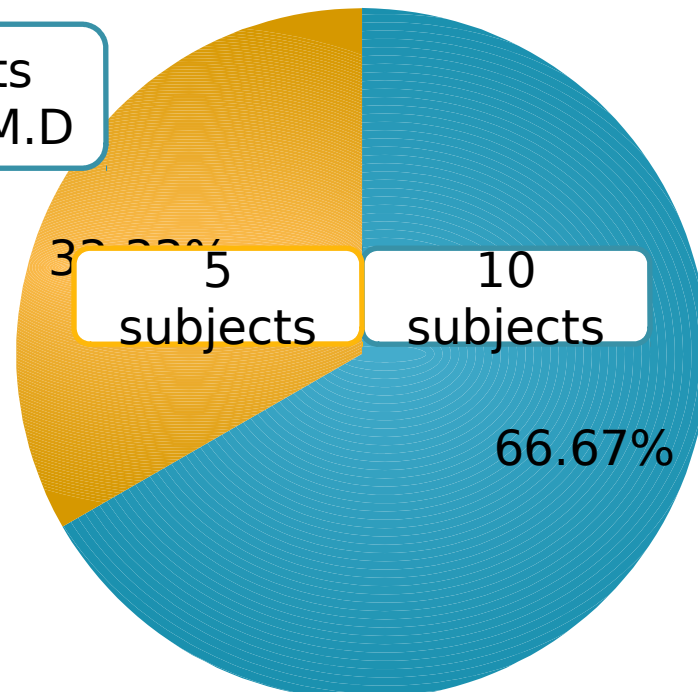
Reference: Young et al(2003)

VEMP and Glycerol test in M.D

Total 17 subjects

■ Abnormal VEMP ■ Positive VEMP

All 5 subjects
were stage II M.D



Murofushi et al. 2001

VEMP in Delayed Endolymphatic Hydrops

- ✓ Characterized by delayed onset of vertigo in subjects with unilateral profound hearing loss
- ✓ Divided in to Ipsilateral and contralateral types

		No. With VEMP responses		
Type	No.	Normal	Decreased	absent
IPSI	12	3	5	4
CONTRA	9	3	0	6

Source: Okhi et al. (2003)

Galvanic VEMP in Endolymphatic Hydrops

Delayed Peaks
of acoustic
VEMP
indicative of
retrolabyrinthine
or central
lesions

Because of
wide variation
in normal
range of
latency makes
it difficult to
separate
labyrinthine
from
retrolabyrinthine

Galvanic
VEMP bypasses
HCs and
directly
stimulates
distal portion of
the vestibular
nerve

Thus, Galvanic VEMP along with
acoustic VEMP can separate
labyrinthine from retrolabyrinthine
lesion

Galavenic VEMP results

Group	Increase d	Normal	Decreas ed	Absent	Total
M.D/ EDH	1	9	0	0	10
CP tumor	0	2	2	14	18
Total	1	11	2	14	28

Reference: Murofishi et al. 2001

Vestibular Neuritis

- ✓ Vestibular loss due to VN may be in the superior vestibular nerve but that the inferior vestibular nerve may be spared (Fetter & Dichgans, 1996).
- ✓ Clinical tests of the peripheral vestibular system have been focused on the lateral semicircular canal and its afferents, the superior vestibular nerve.
- ✓ Issue of the involvement of inferior vestibular loss remains to be clarified.
- ✓ VEMP assess— Inferior Vestibular Nerve

Vestibular Neuritis

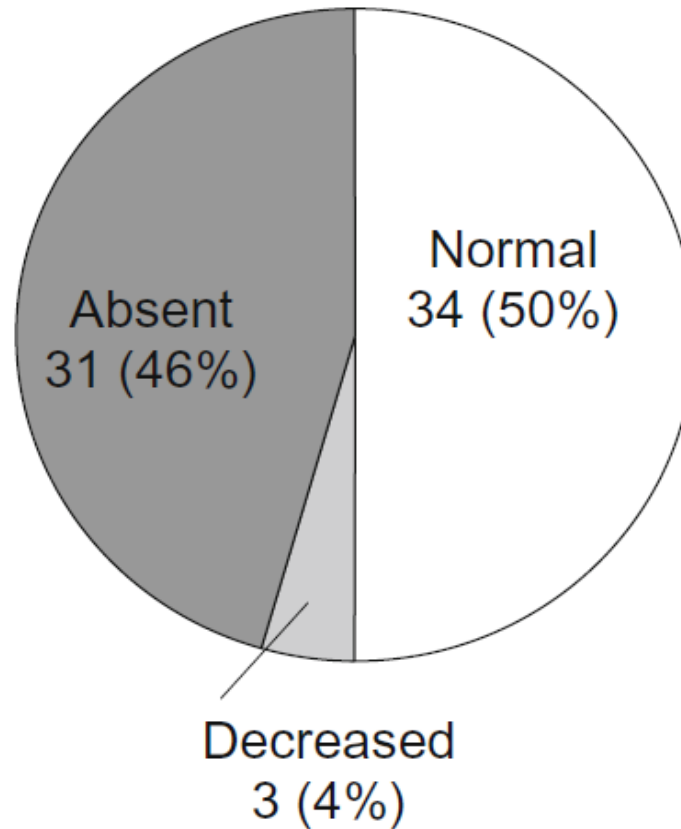
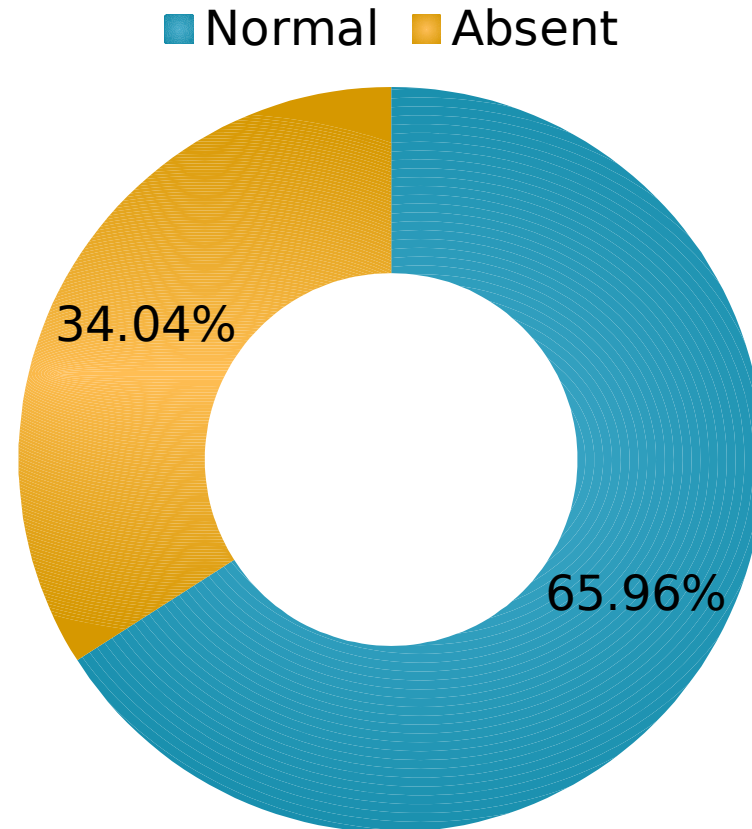


Fig. 1. Vestibular evoked myogenic potential (VEMP) responses in 68 vestibular neuritis (VN) patients

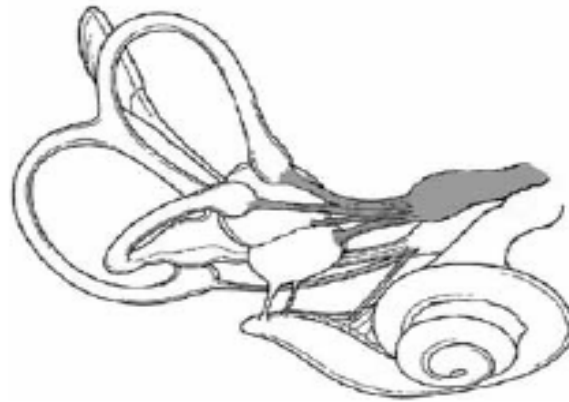
Murofishi et al. (1996)

Total 47 VN patients (Australian Population)



Murofishi et al. (1996)

(a)



(b)

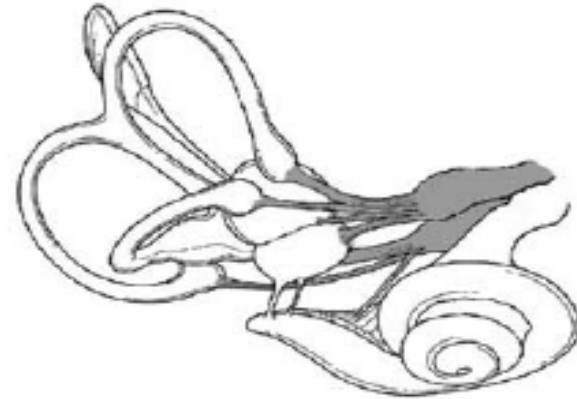


Fig. 2. Classification of vestibular neuritis. **a** Superior vestibular neuritis, **b** total (superior and inferior) vestibular neuritis

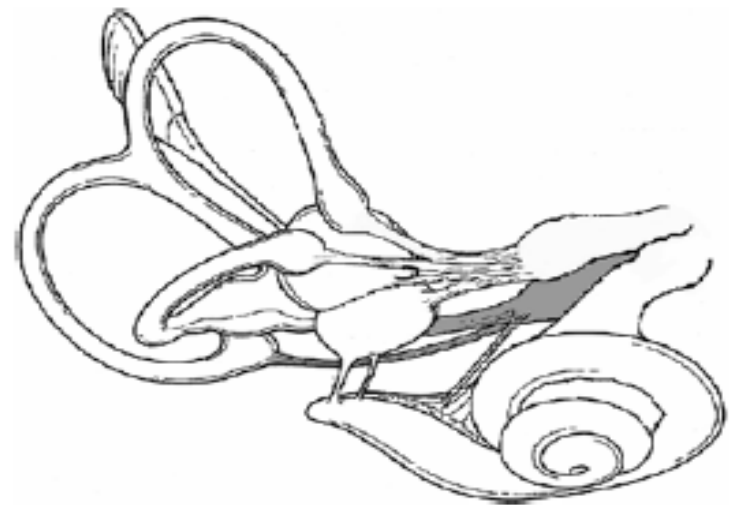
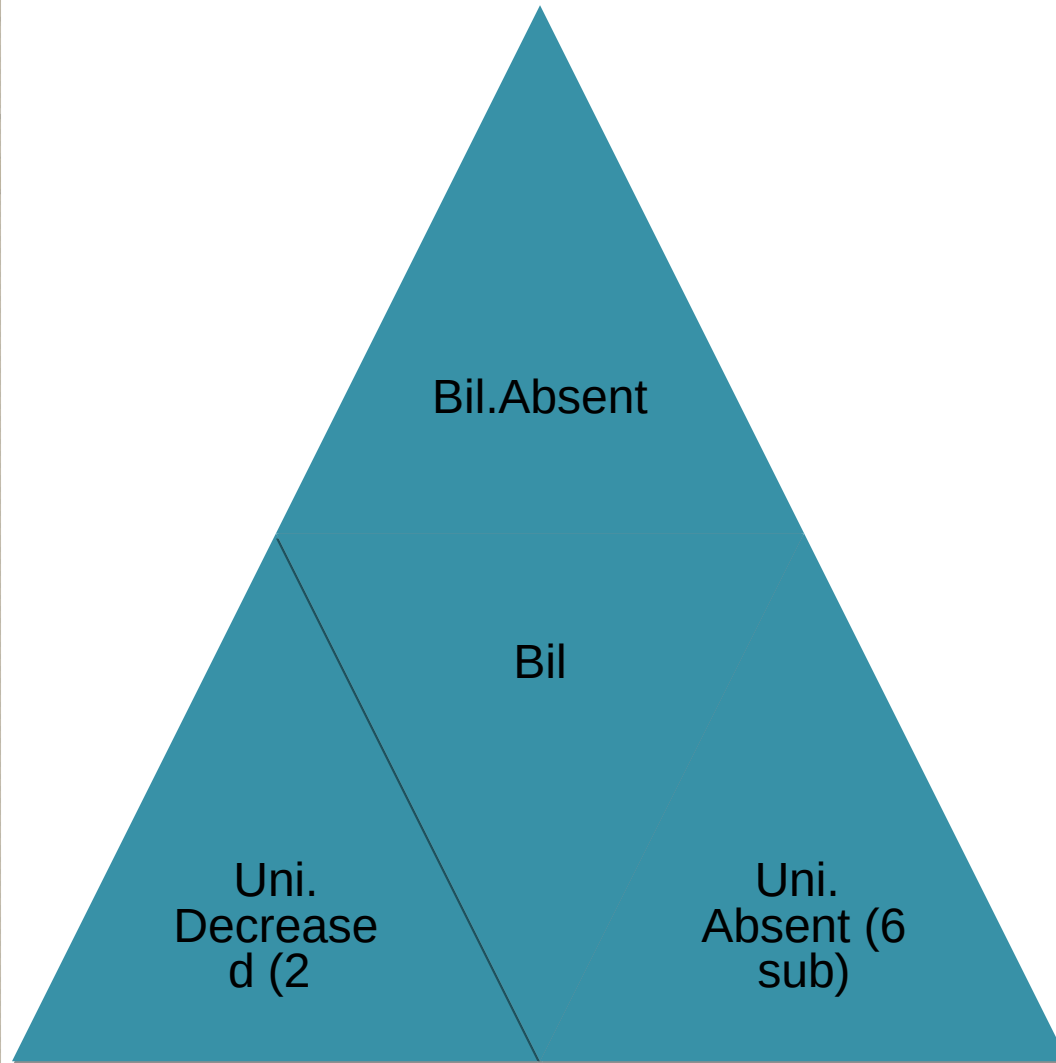


Fig. 3. Inferior vestibular neuritis

Idiopathic Bilateral Vestibulopathy

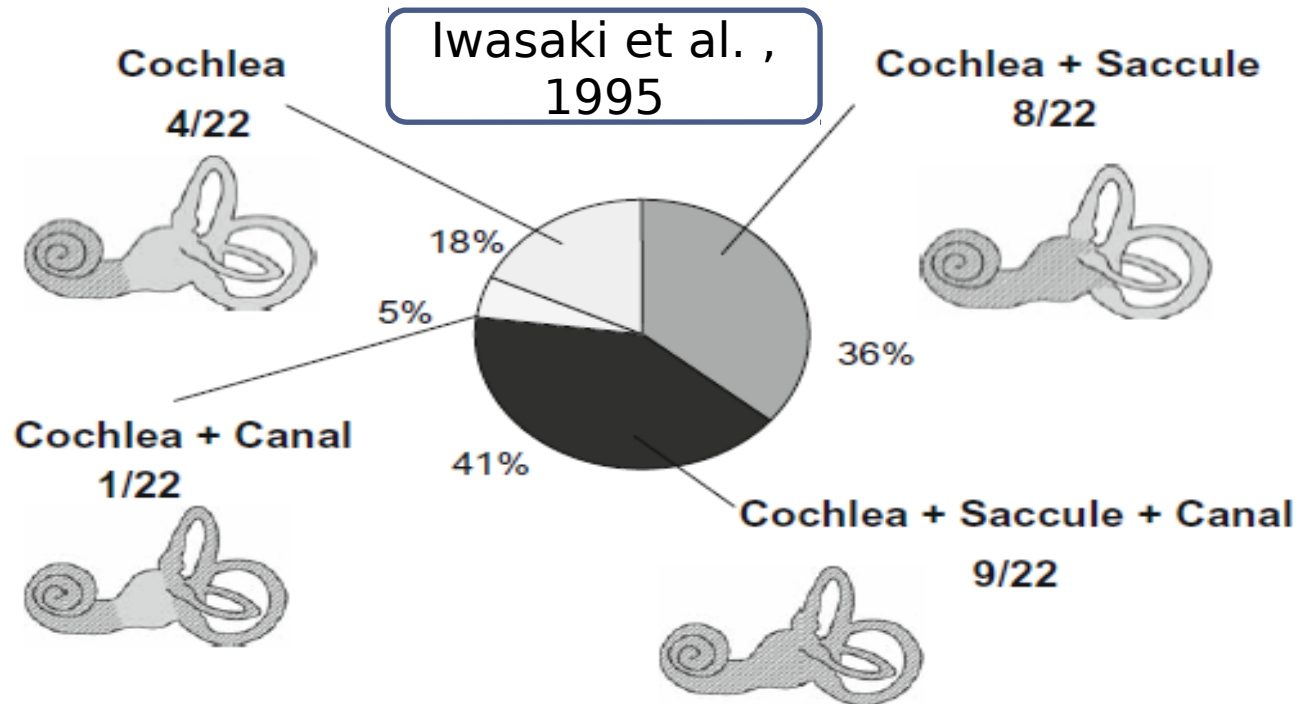
- Idiopathic bilateral vestibulopathy (IBV) is a clinical entity proposed by Baloh et (1987).
- Two types: Progressive and Sequential
- There could be a third type: One attack/progressive type (Murofushi et al. 2010).

VEMP Findings in Idiopathic Bilateral Vestibulopathy



Idiopathic Sudden Sensorineural Hearing Loss

- ✓ Approximately 50% of the subjects with idiopathic sudden sensorineural hearing loss could vestibular symptoms (Schuknecht, 1993).
- ✓ Histopathological study revealed by atrophy of the saccular macula with hair cells (Schuknecht, 1993).



Superior Canal Dehiscence Syndrome

- Superior canal dehiscence syndrome (SCDS) is a clinical entity introduced by Minor et al. (1998, 2000).
- Results from dehiscence of bone overlying the superior (anterior) semicircular canal.
- Characterized by vertigo or oscillopsia induced by pressure and/or a loud sound.
- SCDS is a newly established entity that induces Tullio phenomenon and/or a positive fistula sign.
- Subjects may have low tone hearing loss.

VEMP FINDINGS IN SCD

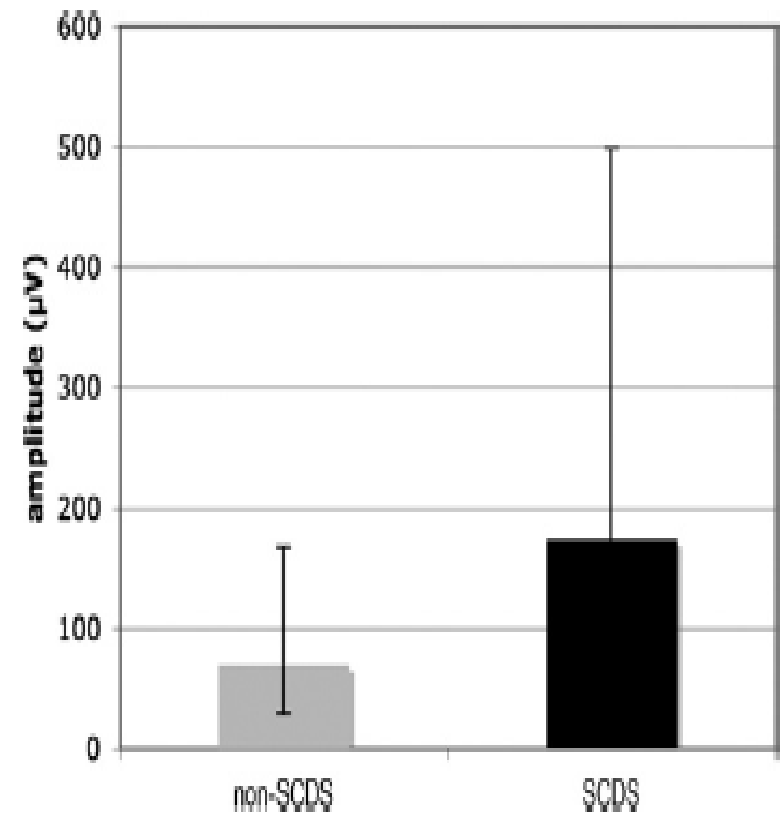
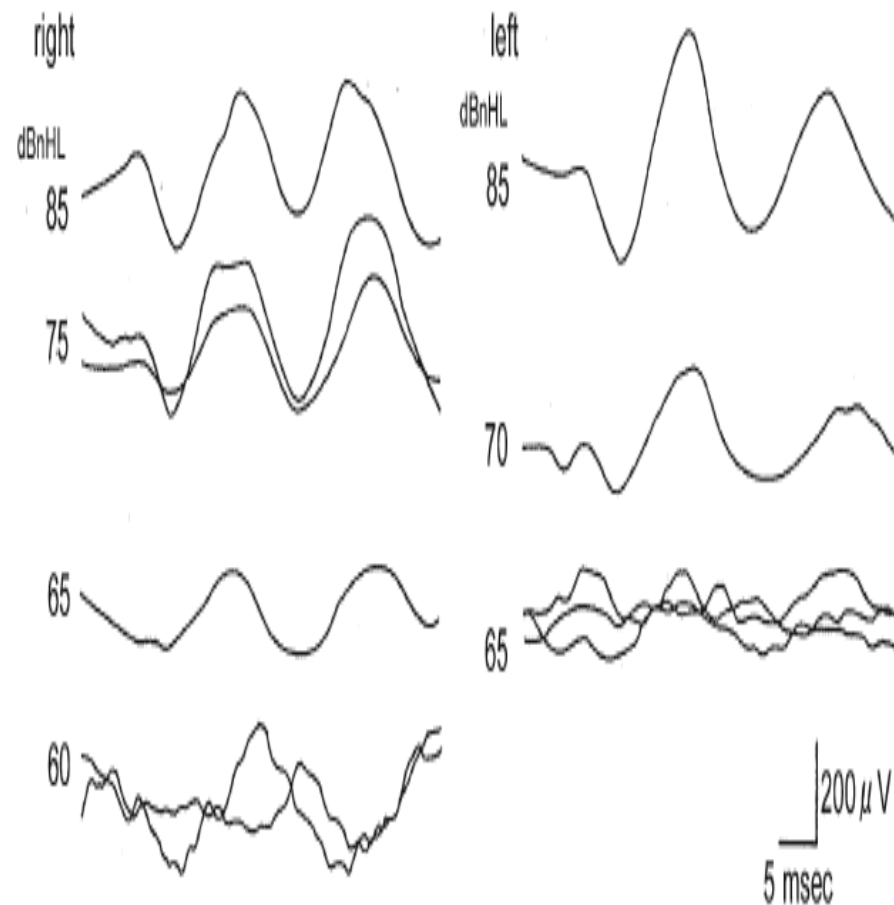
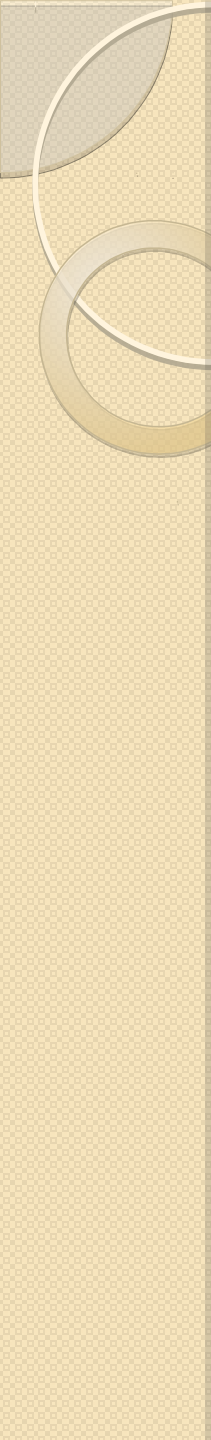


Figure 2 Mean amplitudes for the non-SCDS group and SCDS group at 500 Hz (measured at 95 dB nHL).

- 
- ✓ These features are consistent with hypersensitivity of the vestibular end-organs to sound in patients with SCDS. Although this tendency was also observed with bone-conducted sound.
 - ✓ Galvanic VEMPs showed a normal threshold for individual with SCD (Watson et al.,2000) . These findings suggested that the hypersensitivity can be found in vestibular end-organs.

Migraine associated vertigo and VEMP

Liao and Young (2004) studied 20 patients with Basilar type migraine

10 had bilaterally normal responses

two

patients showed bilaterally prolonged latencies

3 had bilaterally absent responses

4 had a unilateral absence of response

one patient showed a unilateral absence of responses and unilaterally prolonged latencies.

Acoustic Neuroma

- Acoustic neuromas (ANs) are schwannomas that arise mainly from the vestibular division of the eighth cranial nerve (vestibular nerve).
- The most frequent symptoms of ANs are unilateral hearing loss and tinnitus.
- Hearing loss is usually slowly progressive, although it may be of sudden onset.

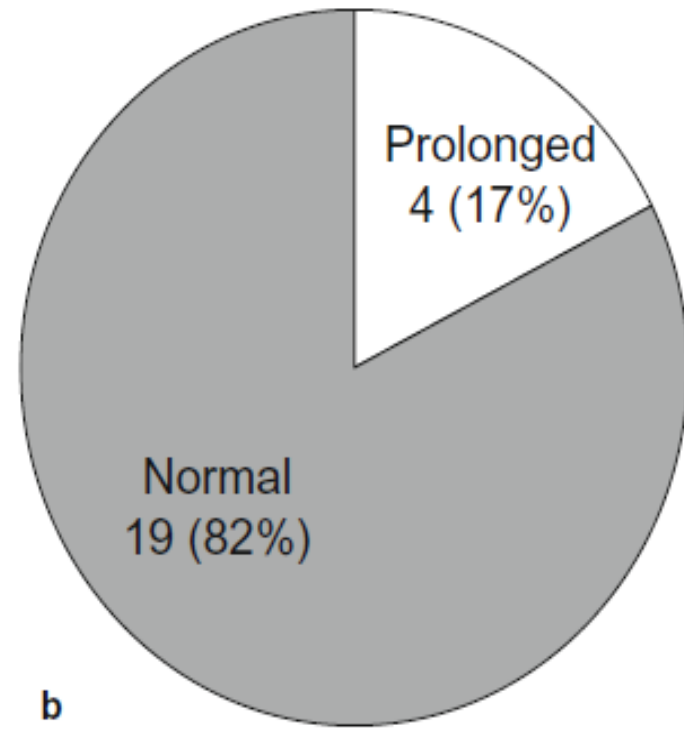
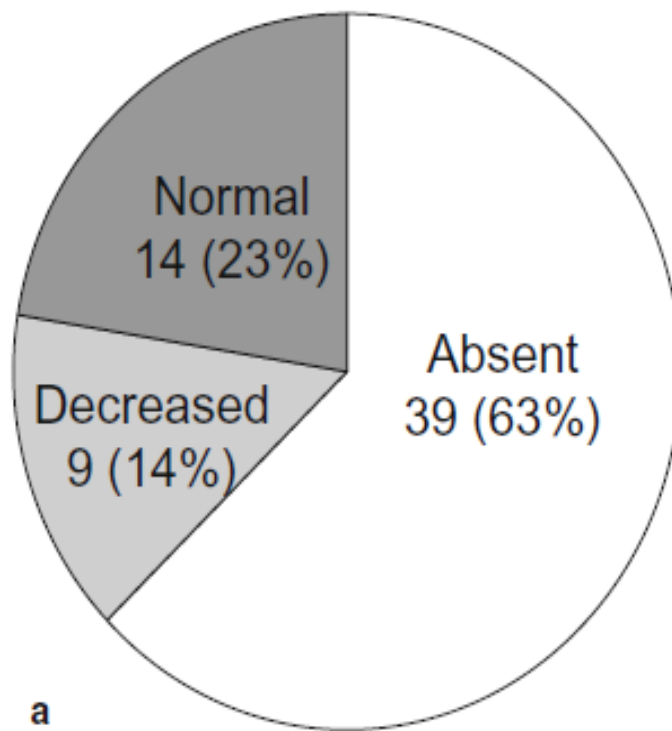


Fig. 3. Vestibular evoked myogenic potential (VEMP) responses in 62 AN patients. **a** Classification according to amplitudes. **b** Classification according to latencies

Murofushi et al. (2001)

Tumor Size and VEMPs

Table 2. Patients with acoustic neuroma and prolonged p13 and/or n23

No.	Amplitude	p13 (ms)	n23 (ms)	I-V (ABR) (ms)	Tumor size (cm)
1	Normal	14.9	31.2	4.92	2
2	Decreased	14.6	21.2	Only wave I	3
3	Normal	14.4	27.4	5.20	2
4	Decreased	15.0	26.0	5.48	2

Murofushi et al. (2001)

Noise Induced Hearing Loss

Table 3: VEMP responses in experimental group (NIHL subjects)

Total no. of ears	No. of ears with normal VEMP present	No. of ears with VEMP abnormal (prolonged latency/reduced amplitude)	No. of ears with VEMP absent
55	20 (36.4%)	19 (34.6%)	16 (29.0%)

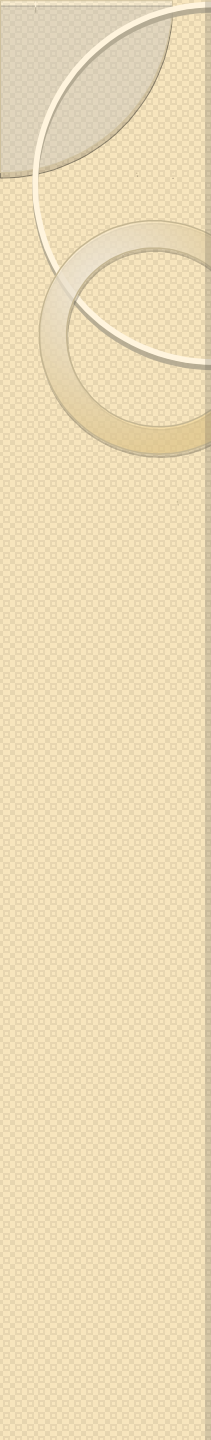
Table 1: Mean and standard deviation of VEMP responses in control group and experimental group

Parameters	Mean		Standard deviation	
	Control group	Experimental group	Control group	Experimental group
P1	11.54 msec	12.96 msec	1.07	1.43
N1	19.20 msec	22.24 msec	2.26	2.64
Peak to peak amplitude	21.67 microvolt	17.57 microvolt	11.03	5.06

Kaushlendra, Christina, & Bhat (2010)

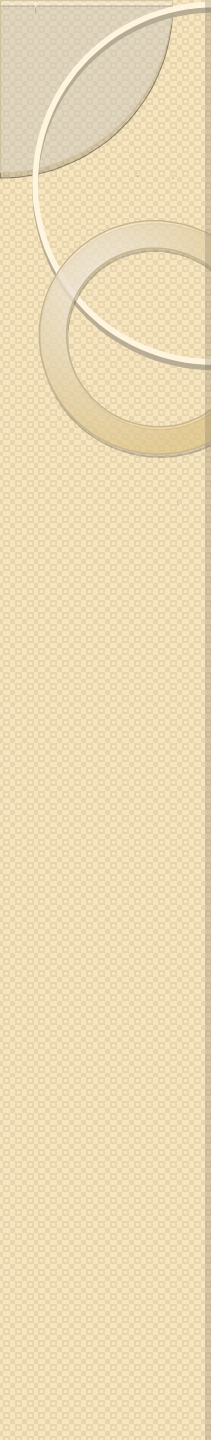
Auditory Neuropathy

- ❖ The incidence of vestibular neuropathies in patients with AN is not known.
- ❖ Difficulties in trying to define vestibular nerve involvements in AN are the gradual and insidious onset of minimal symptoms and the attribution of symptoms such as gait imbalance and unsteadiness to the accompanying peripheral or cranial neuropathies.
- ❖ Starr et al.(1996) described three subjects in whom a lateral gaze nystagmus was present and in one subject caloric responses were absent.

- 
- ✓ Fujikawa (2000) found Vestibular abnormalities in 9 of the 14 patients, 7 of whom had concomitant peripheral neuropathies.

Kumar, Sinha & Barman (2007)

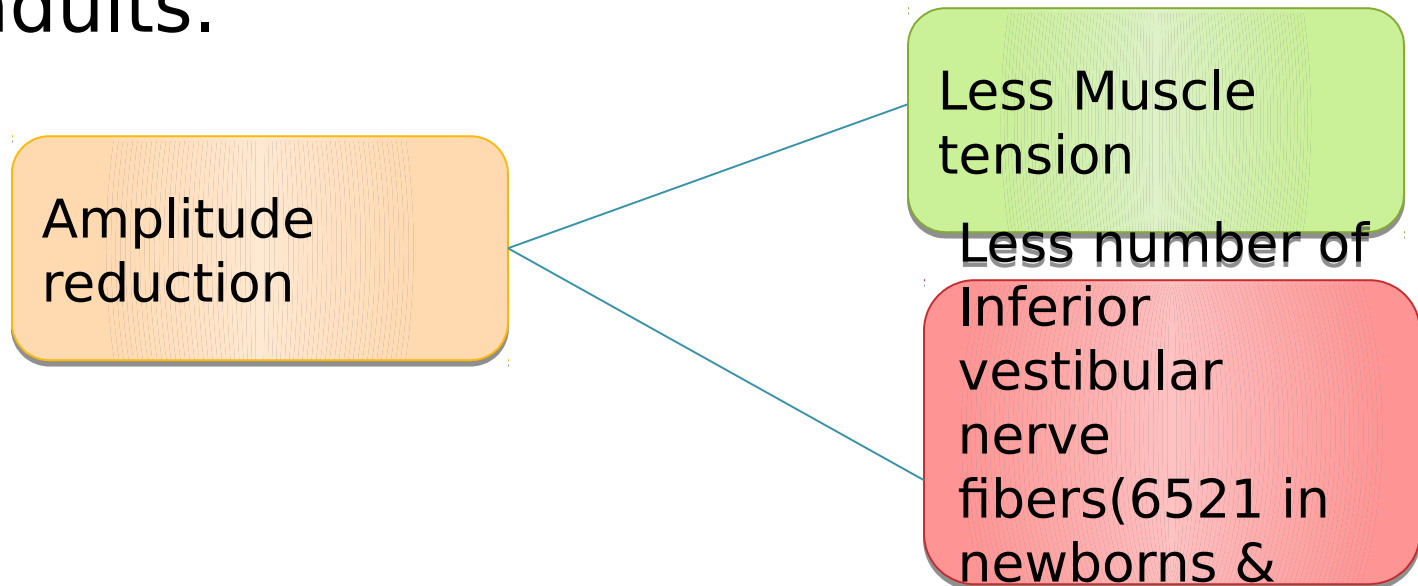
Nine out of the 10 subjects showed abnormal or absent VEMP; (2) there is no one-to-one correlation between the abnormal or absent VEMP and the vestibular symptoms that these subjects present; and (3) 80% of the ears with auditory neuropathy showed abnormal VEMP.



We suggest to use the term “acoustic neuropathy” be used to indicate those patients in whom only the acoustic nerve is affected and “vestibuloacoustic neuropathy” to label those patients who also show involvement of the vestibular system.

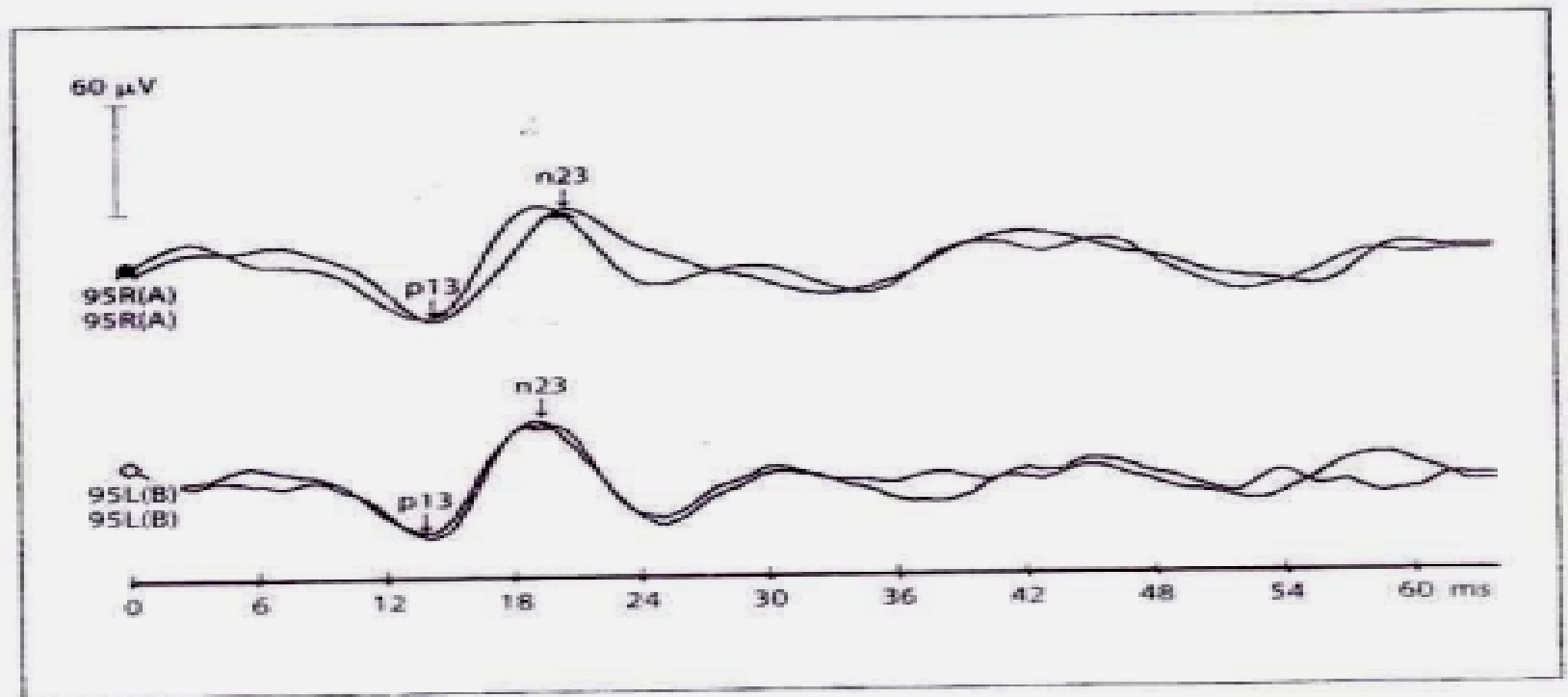
VEMPs in Newborns

- In newborns latency is delayed and amplitude is reduced compared to the adults.



Vestibulo collic reflex normalizes by 2 months of age and matures further till 2 yrs of life(Fife et al.2000). Investigating VEMPs in Newborn may help to study the sacculocollic reflex at birth

VEMPs in Newborns contd:



	Response Rate	P13 latency	N23 latency	amplitude (μv)	P13-N23 interval
Newborns	30(75%)	17.7 ± 4.1	23.9±4.5	27.5	6.3± 1.5
Adults	34(85%)	14.5±1.3	22.3±	59.1	7.8± 1.3

With courtesy from Chen et al. 2007

Development of VEMPs in Preterm Baby

- Vestibular nerve myelination begins at the 20th Fetal week and myelinated at birth(Barkovich,2005).
- Vestibular pathway may be completely responsive but end organs may not be

	Preterm	Fullterm
Response rate	26 %	72%
P13 latency	14.2± 0.4	13.3±0.4
N23 latency	20.1±1.3	18.3±0.6
P13-N23 amplitude (μv)	29.8±12.9	27.5±13.7

Data with courtesy from Wang et al. 2008.

Effect of Aging on VEMPs

Table II. Parameters of VEMP: latencies, amplitude, and VEMP asymmetry in subjects sorted by age group.

Age group* (number of ears)	Latency (ms)		Interlatency (ms)	Amplitude (mV)	Vemp asymmetry
	p13	n23			
I (26)	12.6±1.9	18.5±1.4	5.9±1.6	24.1±6.2	-0.2±7.1
II (34)	13.1±1.6	18.8±1.8	5.6±1.4	19.4±8.6	5.1±10.9
III (28)	13.1±1.6	18.3±1.6	5.2±1.0	16.3±5.5	4.6±6.8
IV (26)	12.9±2.1	18.2±2.1	5.3±1.2	15.1±6.1	-2.1±13.8
V (38)	14.1±2.4	19.8±2.5	5.7±1.3	14.8±6.9	-3.4±9.3
VI (28)	15.8±2.7	22.0±2.7	6.2±1.6	13.8±5.1	-4.1±13.2
VII (14)	15.6±2.2	21.5±3.0	5.9±1.7	14.9±5.8	1.4±8.1
Total (197)	13.8±2.4	19.5±2.6	5.7±1.4	17.0±7.3	0.1±10.8

*Group I, 10–19 years ($n=13$); group II, 20–29 years ($n=17$); group III, 30–39 years ($n=14$); group IV, 40–49 years ($n=13$); group V, 50–59 years ($n=19$); group VI, 60–69 years ($n=14$); and group VII, 70–79 years ($n=7$).

With Courtesy from Lee et al. (2008)

Effect of Aging on VEMPs contd:

<i>Group</i>	<i>Parameters</i>	<i>Mean</i>	<i>Std. Deviation</i>
Group I	P13 latency	11.46	1.28
	N 23 latency	19.25	2.31
	PP amplitude	32.32	18.95
Group II	P13 latency	12.04	1.50
	N 23 latency	19.74	2.46
	PP amplitude	30.32	17.86
Group III	P13 latency	11.98	1.42
	N 23 latency	20.25	2.35
	PP amplitude	27.03	17.97
Group IV	P13 latency	12.53	1.48
	N 23 latency	20.51	2.46
	PP amplitude	21.38	13.22
Group V	P13 latency	13.44	1.52
	N 23 latency	22.38	2.02
	PP amplitude	14.82	5.80

Sinha, Kaushlendra & Bhat, 2010

Thanks for Listening

