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Vestibular PREHAB and gentamicin before schwannoma surgery may improve long-term postural function

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ABSTRACT

Background: Unilateral vestibular deafferentation (uVD), as performed in vestibular schwannoma surgery, results in a chronic vestibular deficit, though most of the insufficiency can be compensated by other sensory input. By vestibular training (prehabituation) performed before surgery, motor adaptation processes can be instigated before the actual lesion. The adaptation processes of the altered sensory input could be affected if the vestibular ablation and surgery was separated in time, by pre-treating patients that have remaining vestibular function with gentamicin.

Objective: To determine whether pre-surgical deafferentation would affect post-surgery postural control also in a long-term perspective (6 months).

Method: 41 patients subjected to trans-labyrinthine schwannoma surgery were divided into 4 groups depending on the vestibular activity before surgery (with no clinical significant remaining function n=17; with remaining function n=8), whether signs of central lesions were present (n=10), and if patients with remaining vestibular activity were treated with gentamicin with the aim to produce uVD before surgery (n=6). The vibratory posturography recordings before surgery and at the follow-up 6 months after surgery were compared.

Results: The subjects pretreated with gentamicin had significantly less postural sway at the follow-up, both compared to the preoperative recordings and to the other groups.

Conclusion: The results indicate that by both careful sensory training and separating the surgical trauma and the effects of uVD in time, can adaptive processes develop more efficiently to resolve sensory conflicts, not only resulting in a reduction of symptoms directly after surgery, but also perhaps up to 6 month afterwards.

Patients scheduled for vestibular schwannomas are subject to challenged rehabilitation processes, partly due to the intracranial surgery, but also to the effects of acute unilateral vestibular deafferentation (uVD). Acute uVD result in vertigo, malaise and postural imbalance, beside the physiological manifestations of spontaneous nystagmus, ocular torsion and disturbances of perceived vertical and horizontal.[1] Vestibular deafferentation also result in cognitive impairments, [2-4] and if performed bilaterally result in hippocampal atrophy. [4] Due to the usually slow growth rate of the tumors, the remaining function of the vestibular system before surgery differs considerably among patients, giving a varying spectrum of postsurgery illness and need for rehabilitation. The status of the vestibular system should be carefully assessed preoperatively. Recently a treatment procedure was reported, in which patients who had remnants of vestibular function before surgery were ablated by installing gentamicin in the middle ear. [5, 6] The treatment aim at achieving total uVD before surgery, giving the patients time to adapt to the altered sensory input. The symptoms and manifestations of uVD subside with time with the development of vestibular compensation,[7] dependent on central nervous system (CNS).[8] Although the symptoms decrease with time, the responses to passively imposed vestibular stimuli do not recover, and other sensory input must compensate the vestibular deficiency.[9] The ability to adapt and modify postural strategies to altered afferent sensory input (as in vestibular disorders), as well as to different external constraints, is essential for an operational postural control system. Postural control is maintained by both feedback and feed-forward mechanisms.[10] Feedback control depends on sensory input (vision, vestibular and somatosensory) that are processed, integrated and weighted to their relative importance and context in the CNS.[11] Feedforward mechanisms involve the concept of "internal models", whose output consists of preformed neuromuscular strategies activated in given situations automatically or voluntarily (anticipated movement). Learning how to withstand a postural perturbation, such as vibratory

proprioceptive stimulation, seem to follow the general paradigm of memory formation with a consolidation of a short-term memory into a longer lasting memory.[12] The generated response corresponds to an internal model designed to control posture when exposed to vibratory stimulation, and most likely involve areas within the CNS involved in adaptation and learning, i.e. cerebellum and hippocampus.[13, 14] Surgery, even minor abdominal, leads to inflammation in the CNS, indicated by elevated cytokines in the hippocampus and cerebellum, resulting in memory impairment when learning new tasks.[15] It can thus be conceivable that surgery and deafferentation performed at the same time would lead to delayed or impaired compensation and that separating the different traumas in time would generate a more efficient re-calibration process of the altered sensory information. In the present study we aimed to assess the recordings with vibratory proprioceptive stimulation before and after surgery of patients with varying vestibular function from the disease, with the specific aim to examine whether pre-surgical deafferentation and sensory training would affect post-surgery postural control also in the longer term, here up to 6 months after surgery.

MATERIAL AND METHOD

Subjects

Between 2001 and 2007, 82 patients were subjected to trans-labyrinthine vestibular schwannoma surgery at Lund University Hospital. Only this surgical approach was considered as the procedure comprises a total destruction of the inner ear and is considered to be the method of choice for small tumors when no hearing-preservation is required. In half of the patients (n=41) the posturography data both pre and post surgery could be retrieved and these patients were included in the study. The vestibular function was assessed both before and after surgery, with video-recorded head impulse test of all 3 canals of each ear [16], bithermal calorics, vestibular evoked myogenic potential measured on the sternocleidomastoid muscle (cVEMP), subjective horizontal and vertical, rod and frame tests, posturography, eye movement analyses and audiometric with pure tone and speech discrimination evaluation. The patients were divided into 4 groups depending on the clinical status before and after surgery (figure 1).

Group 1 (n=17, 11 male and 6 female, median age 58, range 33-71) consisted of patients with no clinically significant vestibular function, defined as pathological head impulse test in all planes of the semicircular canals, an eye-velocity of $<7.5^{\circ}/s$ (mean 3.0, range 0.5-4.9) [17] during calorics and no registered cVEMP of the affected ear. Group 2 (n=8, 4 male and 4 female, median age 43.5, range 26-72) consisted of patients with either intact head impulse test and/or a recorded eye-velocity $>7.5^{\circ}$ /s (mean 10.0, range 1.9-19.4) during calorics. cVEMP varying but intact in 2 patients. The head impulse test was intact in 7 patients. Group 3 (n=10, 6 male and 4 female, median age 52.5, range 28-65) consisted of patients that, beside vestibular disorder, showed signs of central nervous disorder during the pre-surgical assessment (ataxia, pathological voluntary eye-movements, tilted subjective horizon to the contralateral side, Bruhns nystagmus etc.) or signs of severe ataxia post-surgery. The vestibular function before surgery varied from clinically total uVD to normal. Calorics gave a mean of 6.5 and range 0.9-18.6°/s. The head impulse test was intact in 5 patients. Group 4 (n=6, 4 male and 2 female, median age50, range 36-66) consisted of patients with intact vestibular function that were pretreated with gentamicin before surgery (for details see table 1). This regime was offered to all patients with remaining vestibular function after year 2003. All but one patient had measurable remnants of vestibular function of the affected ear. That patient suffered from vertiginous attacks and was regarded as having remaining vestibular function on the affected side. Calorics gave a mean of 11.8 and range 0.5-25.4°/s.

Patient	Vestibular function Prior to Gentamicin	Gentamicin treatment	Vestibular function after Gentamicin	Posturography Assessment
1	Sacculus + Posterior SCC	3 installations 6 months prior surgery	None	 1) 1 week prior gentamicin 2) 6 weeks post gentamicin 3) 5 months post surgery
2	Anterior and lateral SCCs + Utriculus	3 installations 5 months prior surgery	None	 2 months prior gentamicin 5 months post gentamicin 1 week post surgery 6 months post surgery
3	Sacculus + All SCCs	4 installations 3 months prior surgery	None	 1) 1 month prior gentamicin 2) 6 weeks post gentamicin 3) 6 months post surgery
4	Sacculus – All SCCs	3 installations 3 months prior surgery	None	 3 weeks prior gentamicin 6 weeks post gentamicin 1 week post surgery 6 months post surgery
5	None, but vertiginous attacks deemed to be caused by the affected ear	4 installations 3 months prior surgery	None	 1) 1 week prior gentamicin 2) 7 months post surgery
6	All SCCs	2 installations 3 months prior surgery	None	 1) 1 week prior gentamicin 2) 2 months post gentamicin 3) 8 months post surgery

Table 1. Vestibular status and timing of the posturography recordings in Group 4, pre-treated with gentamicin

The rationale behind the patient sub-division were that the vestibular function pre-surgery could affect the outcome on posturography follow-up, and that lesions within CNS could affect compensation processes as well as posturographic measurements, and as such be confounding. The largest tumor extra-meatal diameter differed between the groups as would be expected, with the largest in the group 3 (median 29mm, range 10-40mm) and smaller in group 2 (median 15.5mm, range 11-33mm) and group 4 (median 19mm, range 6-25mm). Group 1 had median size of 22mm, range 5-43mm.

Patients with remaining function of the vestibular system after year 2003 was offered presurgical deafferentation and accepted. They received 2-4 trans-tympanic injections of approximately 0.3-0.4 ml of gentamicin at each injection over 2 consecutive days.

Vestibular PREHAB

All patients were instructed to execute the home based vestibular training program (figure 1), adopted for patients with acute vestibular loss, 14 days before surgery and continue the training program the first postoperative weeks, until they considered themselves free of symptoms. For patients receiving gentamicin the 'PREHAB' constituted of 14 days of training after which gentamicin was installed in the middle ear on the tumour side. The training continued during installations and afterwards as the ototoxic effect of the gentamicin gradually took effect.[18] The subjects were encouraged to perform the program for at least 6 weeks after which they were assessed and considered compensated. The exercises in vestibular 'PREHAB' are identical to the vestibular training program and the term should ideally be restricted to be used in patients that had remaining vestibular function prior to surgery/gentamicin treatment (group 2 and 4). However to avoid confusion we term 'PREHAB' all sensory training performed prior to an intervention (figure 1).

Posturography

Postural control was evaluated by perturbing stance while standing on a force platform (400x400x75 mm) equipped with six strain-gauge sensors. Forces and torques actuated by the feet were recorded with six degrees of freedom by a force platform. Data were sampled at 50 Hz by a computer equipped with a 12-bit AD converter. The vibrations were applied to the muscles by two cylindrical vibrators (0.06 m long and 0.01 m in diameters), held in place with an elastic strap around each leg. The vibration amplitude was 1.0 mm amplitude at a constant frequency of 85 Hz. The vibratory stimulation was executed according to a computer controlled pseudorandom binary sequence (PRBS) schedule[19] for 205 seconds by turning on/off the vibratory stimulation. The PRBS schedule was composed of stimulation shift periods with random duration between 0.8 - 6.4 seconds (yielding an effective bandwidth of 0.1-2.5 Hz). Thus, the designated PRBS stimuli covered a broad power spectrum and the randomized stimulation reduced the opportunity to make anticipative and pre-emptive adjustments.

After information about the test procedure the subjects were instructed to stand erect but not at attention, with arms crossed over the chest and feet at an angle of about 30 degrees open to the front and the heals approximately 3 cm apart. Two tests were conducted at each trial occasion, eyes open, fixating on mark on the wall at a distance of 1.5 m, and eyes closed. The test order followed our set clinical procedure, always starting with eyes open followed by eyes closed. In order to minimize the external disturbances for the test subjects, the recordings were performed while the test subjects listened to classical music relayed through headphones.

Patients in group 1-3 were tested before surgery and after surgery (with exception for one patient that was tested twice before surgery due to prolonged time between the initial

examination and the surgery). Patients in group 4 were tested 2-4 times as illustrated in table 1, because of the necessity of evaluating the gentamicin-treatment efficacy.

Data Analysis

The variance of anteroposterior body sway was calculated for five periods for each test: a quiet stance period (0–30 seconds) before stimulation was applied, and for four periods (1–4) during the stimulation (30–80, 80–130, 130–180, and 180–230 seconds, respectively). Regression analysis of the torque variance showed dependence to the test subjects' squared mass and squared height. The torque variance data were therefore normalized by squared mass and squared height. As vibratory stimulation applied to calf muscles mainly affects muscles predominantly active in the anteroposterior direction[20], only those responses were analyzed. Statistics was calculated using non-parametric Wilcoxon for paired data (groups) and Mann-Whitney for unpaired data (between the groups). Non-parametric statistics were used since some values were not normally distributed and normal distribution could not be obtained after log-transformation.

RESULTS

In figure 2 A+B the normalized sway variance in the anteroposterior direction for all groups are demonstrated. Group 1-3 did not decrease their induced torque variance from before surgery to the follow up 6 months after surgery in either test condition (except for group 3, period 3 with eyes open). Group 4 however decreased the induced torque significantly after surgery in both test conditions (closed eyes period 1-4, and open eyes period 1, 3 and the quiet stance period) as shown in the figure 2 A + B.

The torque variance in groups 1-3 could not be separated from group 4 statistically before surgery regardless test condition. At the follow up, group 1 had significantly more sway than group 4; with eyes closed in period 1 and 4 (p<0.05) and period 3 (p<0.01), and with eyes

open in period 1-3 (p<0.05). Group 2 had significantly more sway with eyes closed in period 1 (p<0.05). Group 3 had significantly more sway with eyes closed in period 1 (p<0.05) and period 3 (p<0.01).

The individual posturography recordings for group 4 are presented in figure 3. For most patients the induced torque variance increased on the test following gentamicin therapy in eyes closed condition. Only for patient 6 did the induced torque variance decrease on the test immediately after gentamicin installation. With eyes open, there was a stronger tendency for the induced torque variance to decrease with each test, although not uniformly so (patient 3 and 4).

DISCUSSION

Measuring torque variance during vibratory perturbation gives an appreciation of the energy that is spent on maintaining postural control while resolving a sensory conflict.[21] As energy conservation is a fundamental principle, it would seem plausible that a task is handled with more ease if less energy is spent. The results in this study clearly demonstrate that patients pre-treated with gentamicin needed or spent less energy at the vibratory posturography test at the time of follow up. Previous evaluations of vestibular compensation after deafferentation with posturography demonstrate that the performance deteriorates immediately after surgery and improves after 3 months to levels even better than before surgery.[22] This could be argued to be due to learning to handle the postural test itself and not necessarily development of more efficient postural strategies due to sensory re-weighting.[12] It has also been argued to the deafferentation itself but rather to the loss of the vestibular role as an orientational and internal reference to other conflicting sensory input.[23] It is conceivable that adaptation processes, essential for coping with the altered sensory information and also in resolving

arising sensory conflicts, are impeded by performing both deafferentation and major surgery at the same time. The vestibular training before surgery (PREHAB) aim at initiating motor learning at a cellular level before the actual lesion,[18] in line with multiple plasticity mechanisms active in cerebellar and hippocampal adaptation.[13, 24] Performing vestibular 'PREHAB' simultaneously with the gradual loss of the remaining vestibular function induced by gentamicin treatment,[18] enabled patients in group 4 to continuously compensate and adapt.[25] It has been shown that even minor surgery interfere with hippocampal function in aged rodents,[15] and post-operative cognitive function and memory function in particular, is impaired especially in elderly.[26, 27] Furthermore, the loss of vestibular input affects hippocampal function and integrity,[4] though that effect is greater in the case of bilateral deafferentation.[28] It is thus not improbable that the surgical trauma associated with translabyrinthine surgery would have similar effects on the CNS, delaying central neural adaptive processing.

It is possible to adapt and form long-term memory from the posturography test itself as described by Tjernstrom et al.[12]. It is also possible that the vestibular training (PREHAB) before surgery was more rigid in the gentamicin group as they were given a specific time to start the exercises and that the consultations at the time for gentamicin injections as well as the follow-up may have induced a better compliance as well as a longer period of executing the exercises. However, both these explanations would assume a consecutive improvement on each posturography,[12] which does not seem to occur. It seems therefore more likely that the difference between the groups is the separation in time of deafferentation and surgery.

Interesting to note is the high torque variance with eyes open generated in patients with remaining vestibular function, not treated with gentamicin, at the follow-up. In these patients the vestibular prehabituation could have been counter-productive, in the sense that the exercises, that mainly train the vestibulo-ocular reflex, are conducted in a sensory setting that will change at the time of surgery.

The patients regarded as clinically deafferent before surgery (group 1) differed from the patients treated with gentamicin. This could either be due to a better compliance to the exercises in the gentamicin group, but also due to a remaining (although hypoactive) vestibular function in the patients included in group1. Following the above reasoning, that could yield a counter-productive sensory training.

The patients showing signs of central disorder before surgery (group 3), had a tendency to decrease their torque variance to the follow-up. Although it is difficult to draw any hard conclusions from the data produced by group 3, given the heterogeneity both with regards to remaining vestibular function as the nature of central nervous malfunction, the data suggest that the adaptation processes were not impeded by any central nervous dysfunction. This could be ascribed to the fact that the pressure effects on the cerebellum and brain-stem from the tumor were alleviated at surgery.

Although the number of patients (6) are small, the results indicate that pre-treating patients that have remaining vestibular function with gentamicin before ablative surgery is of benefit not only for postoperative well-being as previously described, but also for long-term learning to withstand perturbed postural control, i.e. coping with sensory conflicts. Even if the present study is reasonably thorough in assessing the actual vestibular function before surgery, it is nevertheless conceivable that remnants of vestibular function exist though not detected. This perspective indicate a rationale for treating even more patients scheduled for vestibular schwannoma surgery with vestibular PREHAB and gentamicin, at least when no hearing-conservation is required. To corroborate such a conclusion however, prospective studies are needed.

REFERENCES

1. Karlberg M, Aw ST, Black RA, Todd MJ, MacDougall HG, Halmagyi GM. Vibrationinduced ocular torsion and nystagmus after unilateral vestibular deafferentation. *Brain*. 2003 Apr;**126**(Pt 4):956-64.

2. Talkomski ME, Redfern MS, Jennings JR, Furman JM. Cognitive Requirements for Vestibular and Ocular Motor Processing in Healthy Aduts and Patients with Unilateral Vestibular Lesions. *J Cogn Neurosci*. 2005;**17**(9):1432-41.

3. Yardley L, Burgneay J, Nazareth I. Neuro-otological and psychiatric abnormalities in a community sample of people with dizziness: A blind controlled investigation. *J Neurol Neurosurg Psychiatry*. 1998;65:679-84.

4. Brandt T, Schautzer F, Hamilton DA, et al. Vestibular loss causes hippocampal atrophy and impaired spatial memory in humans. *Brain*. 2005;**128**:2732-41.

5. Magnusson M, Kahlon B, Karlberg M, Lindberg S, Siesjö P. Preoperative vestibular ablation with gentamycin and vestibular 'prehab' enhance postoperative recovery after surgery for pontine angle tumours - first report. *Acta Otolaryngol*. 2007;**127**(12):1236-40.

6. Magnusson M, Kahlon B, Karlberg M, Lindberg S, Siesjö P, Tjernström F. Vestibular 'PREHAB'. *Ann N Y Acad Sci*. In Press.

7. Curthoys IS, Dai MJ, Halmagyi GM. Human ocular torsional position before and after unilateral vestibular neurectomy. *Exp Brain Res*. 1991;**85**(1):218-25.

8. Vidal PP, de Waele C, Vibert N, Muhlethaler M. Vestibular compensation revisited. *Otolaryngol Head Neck Surg*. 1998 Jul;**119**(1):34-42.

9. Curthoys IS. Vestibular compensation and substitution. *Curr Opin Neurol*. 2000 Feb;**13**(1):27-30.

10. Johansson R, Magnusson M, Akesson M. Identification of human postural dynamics. *IEEE Trans Biomed Eng.* 1988 Oct;**35**(10):858-69.

11. Peterka RJ. Sensorimotor integration in human postural control. *J Neurophysiol*. 2002 Sep;**88**(3):1097-118.

12. Tjernstrom F, Fransson PA, Hafstrom A, Magnusson M. Adaptation of postural control to perturbations--a process that initiates long-term motor memory. *Gait Posture*. 2002 Feb;**15**(1):75-82.

13. Boyden ES, Katoh A, Raymond JL. Cerebellum-dependent learning: the role of multiple plasticity mechanisms. *Annu Rev Neurosci*. 2004;**27**:581-609.

14. Nadel, Bohbot. Consolidation of memory. *Hippocampus*. 2001;**11**:56-60.

15. Rosczyk HA, Sparkman NL, Johnson RW. Neuroinflammation and cognitive function in aged mice following minor surgery. *Exp Gerontol*. 2008;**43**:840-46.

16. Karlberg M, Annertz M, Magnusson M. Acute vestibular neuritis visualized by 3-T magnetic resonance imaging with high-dose gadolinium. *Arch Otolaryngol Head Neck Surg*. 2004 Feb;**130**(2):229-32.

17. Bohmer A, Rickenmann J. The subjective visual vertical as a clinical parameter of vestibular function in peripheral vestibular diseases. *J Vestib Res*. 1995 Jan-Feb;**5**(1):35-45.

18. Magnusson M, Padoan S. Delayed onset of ototoxic effects of gentamicin in treatment of Meniere's disease. Rationale for extremely low-dose therapy. *Acta Otolaryngol*. 1991;**111**:671-6.

Johansson R. System Modeling and Identification. NJ: Prentice Hall Englewood Cliffs;
 1993.

20. Eklund G. Further studies of vibration-induced effects on balance. *Ups J Med Sci*. 1973;**78**(1):65-72.

21. Magnusson M, Johansson R, Wiklund J. Galvanically induced body sway in the anterior-posterior plane. *Acta Otolaryngol*. 1990 Jul-Aug;**110**(1-2):11-7.

22. Parietti-Winkler C, Gauchard GC, Simon C, Perrin PP. Sensorimotor postural rearrangement after unilateral vestibular deafferentation in patients with acoustic neuroma. *Neurosci Res.* 2006 Jun;55(2):171-81.

23. Nashner LM, Black FO, Wall C, 3rd. Adaptation to altered support and visual conditions during stance: patients with vestibular deficits. *J Neurosci*. 1982 May;**2**(5):536-44.

24. Jorntell H, Hansel C. Synaptic memories upside down: bidirectional plasticity at cerebellar parallel fiber-Purkinje cell synapses. *Neuron*. 2006 Oct 19;**52**(2):227-38.

25. Cohen HS. Disability and rehabilitation in the dizzy patient. *Curr Opin Neurol*. 2006 Feb;**19**(1):49-54.

26. Caza N, Taha R, Qi Y, Blaise G. The effects of surgery and anesthesia on memory and cognition. *Prog Brain Res.* 2008;169:409-22.

27. Howland JG, Wang YT. Synaptic plasticity in learning and memory: stress effects in the hippocampus. *Prog Brain Res.* 2008;169:145-58.

28. Hüfner K, Hamilton DA, Kalla R, et al. Spatial memory and hippocampal volume in humans with unilateral vestibular deafferentation. *Hippocampus*. 2007;**17**:471-85.

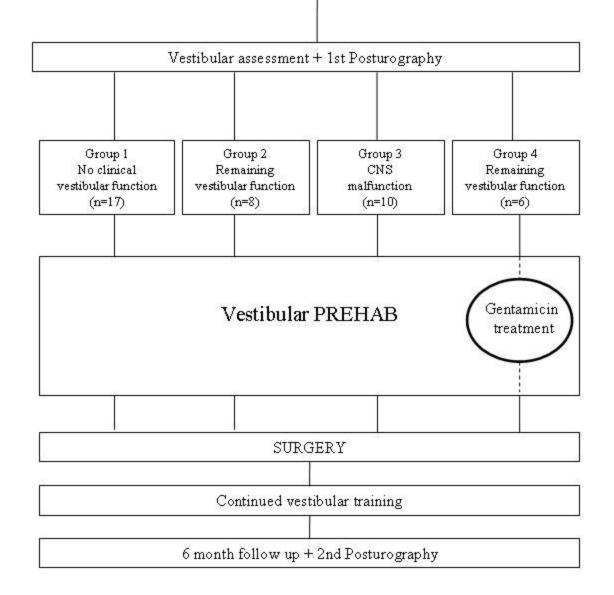
Figure 1. Table of sequence describing group division and the interventions for each group. 'PREHAB' is further described in the method section and the exercises are described in detail in figure 2. **Figure 2**. Vestibular training program constituting the vestibular 'PREHAB' as well as the sensory training before and after surgery as well as after gentamicin treatment, which all patients were encouraged to execute. The principle to the exercises is to use sensory mismatch to enforce recalibration of vestibular reflexes on a cellular level.[25]

Figure 3. Normalized anteroposterior torque variance in all groups, closed eyes (A) and open eyes (B), at the time of primary assessment (black line) and at the follow-up after 6 months (grey line).

Figure 4. Individual posturography recordings in the gentamicin group as further defined in table 1. The data consists of normalized anteroposterior torque variance. For detailed information as to the timing of the posturography recordings in each individual, please see table 1.

Black lines=before surgery, Dashed Black lines=after gentamicin, Dashed grey line=after surgery, Grey lines=At the follow-up 6 months after surgery

Patients selected for translabyrinthic approach



daily3 times

Note! You will experience some dizziness when you do the exercises! A home-based training program for Dizziness



1. Sit down, Fixate on an object 1,5 - 2 m away. Shake your head horizontally from side to side - still fixeating the object. Repeat twice a second for 15 seconds. Count one-thousand-one; one-thousand-two ... one-thousend -fifteen to keep pace and time

2. Stand up and put a finger on a stabile onject (chain/table), Fixate on an object 1,5 - 2 m away. Shake your head horizontally from side to side - still fixeating the object. Repeat twice a second for 15 seconds. Count one-thousand-one; onethousand-two ... one-thousend -fifteen to keep pace and time. .

3. Stand up with out support or touching any object. Fixate on an object 1,5 - 2 m away Shake your head horizontally from side to side - still fixeating the object fixate on an object 1.5 - 2 m away. Shake your head horizontally from side to side - still fixeating the object. Repeat twice a second for 15 seconds.

4. Stand up and close your eyes (with and then without support) .

Fixate on an object 1.5 - 2 m away. Shake your head horizontally from

side to side - still fixeating the object. Repeat twice a second for 15 seconds.

5. Stand up, fixate on an object 1.5 - 2 m away. Shake your head vertically from side to side - still fixeating the object. Repeat twice a second for 15 seconds.

6. Walk forwards fixate on an object 1.5 - 2 m away. Shake your

head horizontally from side to side - still fixating the object.

Repeat twice a second for 15 seconds.

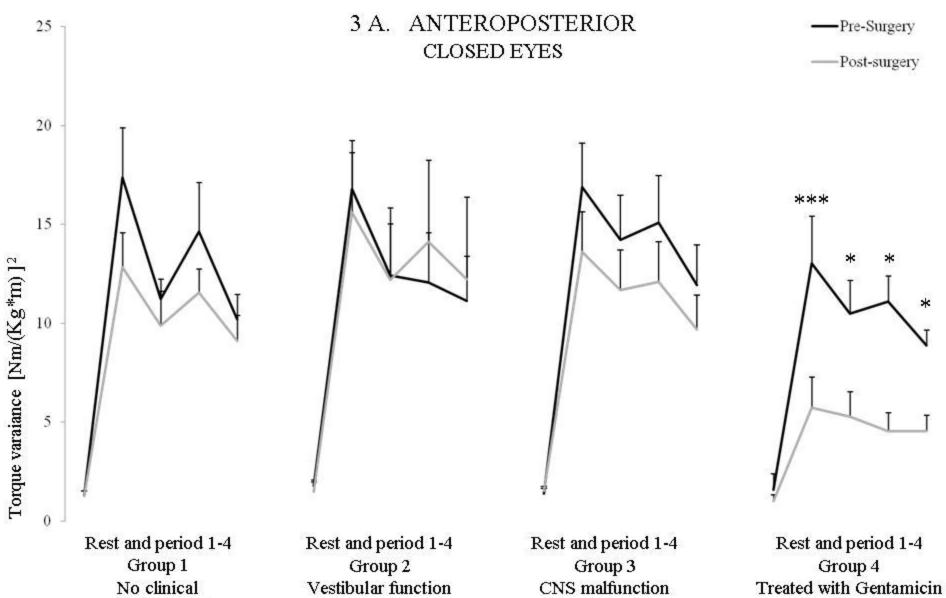
7. Stand on a pallow from your coach in a corner of the room. Remain there fore 1 minute then close your eyes and remain standing like this for another minute. If it is difficulte place a chair in front of you and initially you may put a fingertip on the chair.

8. Stand up holding a glass of water which is filled halfways again in a corner of the room. Remain there fore 1 minute than close your eyes and remain standing like this for another minute. If it is difficulte pu a chair in front of you and initially you may put a fingertip on the chair.

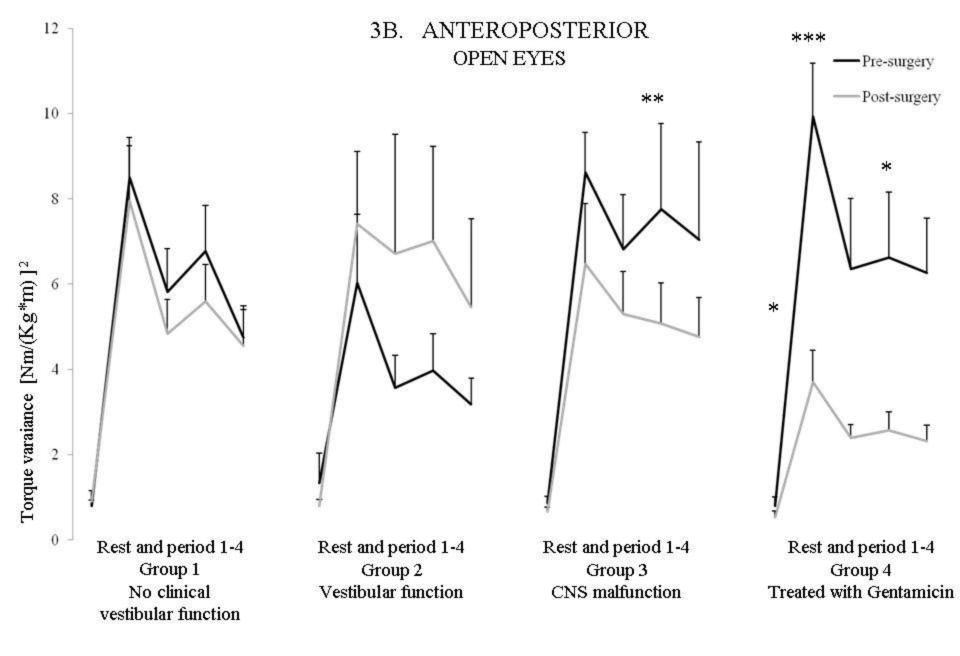
9. Take walk outdoor for at least 30 minutes. Try windowshopping which will have you turning your head from side to side when you walk

How to execute the head movements:

Begin to smoothly shake your head. Increase the speed until vision gets blurred. Decrease speed to regain clear vision, then increase again. The idea is to push the limit were vision gets blurred.



vestibular function



4.A. ANTEROPOSTERIOR CLOSED EYES

