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Electronystagmographic analysis of optokinetic and smooth pursuit eye movement disorders in vestibular lesions

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Abstract

The electronystagmographical analysis of the eye movements is an important method in the evaluation and topical diagnostic procedure of several vestibular lesions. The aim of the study was to compare the electronystagmographical results of the optokinetic and the smooth pursuit eye-movement, and their sensitivity in several vestibular disorders. The patients were divided into five groups: right and left unilateral and bilateral peripheral lesions, central vestibular dysfunction, and normal vestibular function. In patients with normal vestibular system the optokinetic eye movement was pathological in 9.53% of patients, while the smooth pursuit eye movements were pathological in 8.3% of patients with normal vestibular function. In unilateral lesions, 17.42% of the OKNs were pathological, compared with the smooth pursuit test's 20.3% pathological ratio. In the bilateral peripheral vestibular dysfunction the ratio of the pathological eye-movements was 28% equally with to two methods. In central vestibular lesions 22.72% of the patients had abnormal optokinetic eye movements, and the smooth pursuit eye movement was abnormal in 41.6%. Our results show that in the unilateral peripheral vestibular lesions the smooth pursuit eye movement examination seems to be more sensitive than the OKN test, while in central dysfunctions the smooth pursuit eye movement examination is more sensitive than OKN examination.

Keywords: electronystagmography, nystagmus optokynetic, vestibular neuropathy.

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INTRODUCTION

The accurate diagnosis and anatomic localization of the cause of the vestibular disorders usually requires a reliable assessment of general oculomotor function. Vestibular and optokinetic eye movements work together to keep the image of the world stationary on the retina during head rotation. Saccadic, pursuit eye movements change gaze so that images of objects of interest are bought to or kept on the fovea, where visual resolution is highest. Pursuit movements maintain on the fovea the image of an object that is already moving.

Normally the optokinetic eye movement holds images of the seen world steady on the retina during sustained head rotations, while smooth pursuit eye movement holds the image of a moving target on the fovea.[1]

Smooth pursuit eye movements present the foveal stimulation; optokinetic stimulation works is working as retinal stimulus. These could be "markers" of visual dependence in lack of equilibrium**.** [2]

The cerebellum plays an important role in both immediate on line and long term adaptive ocular motor control. The vestibulocerebellum regulates the eye movements in space during smooth pursuit tracking. Lesions of the flocculus impair smooth visual tracking. The vestibulocerebellum also participates in the long term prevention of ocular motor dismetria. The pathway of the smooth pursuit system runs from the cortical areas of the brainstem via the cerebellum.

Visual (optokinetic) inputs reach the vestibular nuclei, and by supplanting the fading labyrinthine signal, they help to maintain an accurate internal representation of the head's velocity.[1]

Observation of.optokinetic.nystagmus (OKN) may be of invaluable help in the diagnosis of.lesions.of the. vestibular.pathways at all levels from the labyrinth to the cerebral cortex and facilities for its observation are an essential item of modern otological equipment.[3]

Two mechanisms operate to overcome the vestibule-ocular reflex (VOR) and allow gaze to track the target: the smooth pursuit system provides a signal to cancel a normally-operating VOR and a reduction of the gain of the vestibulo-ocular reflex is achieved, an ability that is preserved even in patients with cerebral lesions that impair smooth pursuit.[4]

A directional preponderance of OKN slow phase velocity (SPV) was found corresponding to spontaneous nystagmus. This was due to enhancement of nystagmus SPV to the side of the lesion and depression of SPV in the opposite horizontal direction.[5] Compensatory.eye movements.in labyrinthine-deficient patients were always less than in normal subjects[6]**.**

The examination of.eye movements.is an even more sensitive method than magnetic resonance imaging for the diagnosis of acute vestibular.syndromes and for the differentiation of peripheral from central lesions.[7]

Optokinetic nystagmus could be pathological in unilateral supratentorial lesions. In most of the patients a directional preponderance to the ipsilateral side could be observed. Some cases showed marked inhibition of optokinetic.nystagmus of the contralateral side. These markedly inhibited cases are very similar to cases of pontine lesion. Pontine lesions show inhibition of the. optokinetic.nystagmus to the ipsilateral side.[8]

Smooth pursuit eye tracking test is a useful one in drug toxicity, central nervous system tumors, posterior or middle cerebral arteries thrombosis, encephalitis, whiplash injury, Parkinsonism. The test is rarely pathological in the peripheral vestibular damage, but the strong spontaneous nystagmus can produce abnormalities of pendular eye movement. Smooth pursuit and saccadic disorders are present mainly in central vestibular lesions.

Total ablation of the cerebellum was proved to be manifested by the disturbances of both saccades and sinusoidal eye movements[9].

Considering the large inter-individual differences in the results of vestibulo-visual interaction, the actual values of nystagmus slow phase velocity have only limited diagnostic value in a single case. But by averaging the results obtained from various groups of patients very different but distinct patterns of vestibulo-visual interaction can be demonstrated[10]**.**

According to these facts the aim of our study was to evaluate what the normal average slow phase velocities of the spontaneous nystagmus in several diagnostic groups are.

Our second aim was to compare the optokinetic and the smooth pursuit eye-movement examination sensitivity and specificity in several vestibular disorders.

PATIENTS AND METHODS

The electronystagmographical data of 327 patients, who were examined between September of 2009 and February of 2011, were analyzed in this study.

The examination began with the detailed case history and routine oto-rhino-laryngological and neurological examinations. The cochleovestibular function of all the patients was examined by separate cochlear nerve and vestibular function tests. Cochlear function tests included the pure tone audiometry, acoustic reflex threshold and decay. The vestibular tests involved statokinetic tests (Romberg, sensitized Romberg and Babinski-Weil tests); spontaneous nystagmus with Frenzel's glasses and with ENG registration as well, positional and positioning

nystagmus examination using Frenzel's glasses. The saccadic and smooth pursuit eye movement tests and the optokinetic tests were performed by a computer-based ENG system. Electronystagmography was performed with ICS Chartr® electronystagmographical system.

In the OKN test the target points are moving horizontally with 20 degree/seconds.

In the smooth pursuit eye movement test: a stimulus for the test is a light target moving in a sinusoidal pattern including 3 cycles of each the following frequencies 2,3,4,5,6,7 Hz in a 34 degree screen. The amplitude of the movement is 16.7 degrees. Once completed, the sequence of sine waves repeats until the test is terminated.

During analysis the stimulus and the eye-movement elicited are compared by the ICS Chartr® software. During the computer-based analysis the phase of the fundamental frequency of the eye-movement is computed from a discrete Fourier transformation compared to the phase of the stimulus. If phase shift is greater than 3 degrees leading or more than 20 degrees lagging, the cycle is rejected as an artifact. This is based on empirical observations of the effects of patients' noncompliance with instructions. For gain, the velocity of the stimulus is over 250 milliseconds faster if compared to the eye-movements of more than 15 degrees/second faster than the stimulus, are eliminated for the calculations. The result is given in a graphic form.[11]

In most of the cases the optokinetic and smooth pursuit eye movement examinations were performed. In cases of severe loss of visual function, when the patient cannot see the moving target, the eye movement examination failed. These patients are excluded from this study.

Finally, bithermal caloric test was carried out by the computer-based ENG. The ears were stimulated with the 50°C and 25°C air insufflations for 40 seconds. The air caloric stimulation is the routine test battery of our otoneurological department even in the cases of normal external ear canal and normal ear drum, because the air caloric system is strictly connected to the computerized ENG system.

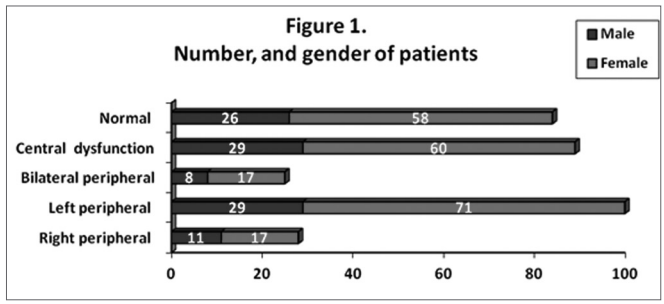
For the data analysis, the patients (n= 326) data were divided into five groups: right or left unilateral and bilateral peripheral lesions, central vestibular dysfunction, and normal vestibular function.

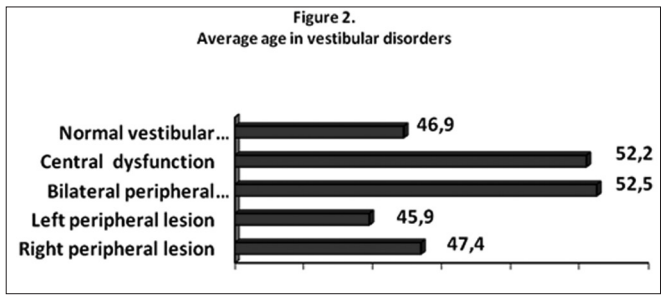
RESULTS

Figure 1 and 2 shows the demographical data of the patients.

The patients have several diagnoses in the background of the vestibular disorder.

In the normal vestibular system group (n=84), 23 patients had no vertigo, they were examined as an evaluation of sensorineural hearing loss. Sixteen patients





had intermittent vertebrobasilar insufficiency without vestibular lesion, while 26 patients had anxiety disorder. Nineteen patients had typical case history of BPPV, but at the time of the examination the vestibular system was normal.

In the right peripheral vestibular system group (n=27) one patient had Ménière's disease, 4 patients had vestibular neuronitis, 8 had BPPV (benign paroxysmal positional vertigo), 3 patients had labyrinthine concussion. In 11 cases patients suffered from sudden sensorineural hearing loss with vestibular dysfunction.

In the left peripheral group (n=100), 21 patients had Ménière's disease, and 27 patients had vestibular neuronitis. Twenty patients had BPPV, and sudden loss of cochleovestibular function was diagnosed in 14 cases. In some cases migrainous vertigo (n=2), and acoustic neuroma (n=2). One-one patient had trauma, labyrinthitis, a herpes zoster infection, cochlear otosclerosis. Two patients had autoimmune inner ear disorder, and 8 patients' final diagnosis remained unevaluated. We didn't find any explanation during the examination period, why there were more peripheral lesions on the left side than the right one.

In the bilateral group (n=25) 2 patients had bilateral vestibular neuronitis, 2 patients had BPPV with decreased caloric responsiveness. Two patients suffered ototoxic damage and in one patient a posttraumatic lesion occurred. Vascular origin of the vestibular lesion was suspected in 11 cases. Two patients have bilateral pontocerebellar angle space-occupying lesion, and 5 patients' bilateral lesion remained unqualified.

In the central vestibular lesion group (n=89) 56 patients had vertebrobasilar insufficiency, 13 patients

had central vestibular lesion of migrainous origin. Multiple sclerosis was diagnosed in 3 cases, while 5 patients had post infectious vestibular lesion. Posttraumatic central dysfunction was diagnosed in one, and cervical vertigo in 5 patients. The exact cause of central vestibular lesion was still unclear in 6 cases.

The average slow phase velocities of the spontaneous nystagmus in several diagnostic groups were shown on Table 1.

Table 1. Average SPV of spontaneous nystagmus.

Vestibular disorder	Average	Standard deviation
Normal vestibular system	11,08	13,76
Right peripheral lesion	12,88	14,81
Left peripheral lesion	5,01	4,85
Bilateral peripheral lesion	8,08	17,02
Central vestibular dysfunction	16,35	17,10

Using the Kruskal-Wallis One Way Analysis, the differences in the median values among the groups were statistically significant (P = <0,001). Using the Dunn's method (Multiple Comparisons versus Control Group), the SPV of spontaneous nystagmus in right peripheral group versus normal vestibular system group, the bilateral lesion versus normal group, and the central vestibular lesion versus normal vestibular function were statistically significant (P<0.05).

The average frequencies of the spontaneous nystagmus in the above mentioned groups are shown on Table 2.

Table 2. Average frequencies of spontaneous nystagmus.

Vestibular disorder	Average	Standard deviation
Normal vestibular system	7,73	7,754
Right peripheral lesion	9,26	10,102
Left peripheral lesion	6,83	13,043
Bilateral peripheral lesion	4,24	7,457
Central vestibular dysfunction	11,16	10,401

The differences in the median values among the groups are greater than would be expected by chance; there is a statistically significant difference (P = <0,001) (Kruskal-Wallis One Way Analysis of Variance). Using the Dunn's Method, the multiple comparisons versus normal vestibular system the examination of the nystagmus frequencies, the values of normal vestibular system versus left peripheral lesion, and the values of bilateral lesion versus normal vestibular system, the difference was statistically significant.

The caloric irrigation was performed in every patient during the diagnostic process. The average caloric

Table 3. Optokinetic nystagmus slow phase velocities.

Lesion	OKN SPV to right	OKN SPV to left	Difference absolute values
Right peripheral lesion	30,82	33,75	7,18
Left peripheral lesion	27.08	26.87	5.18
Bilateral peripheral lesion	26,36	26.12	6,72
Central vestibular dysfunction	33,93	35,44	7,83
Normal vestibular function	32,11	33,65	4,60

nystagmus SPV in normal vestibular system was 17.4 degree/sec in unilateral vestibular lesions, it is 14.65 and in compensated bilateral lesions it is decreased (9.56 degree/sec).

In the central dysfunctions the average caloric ASPV is increased (19.7 degree/sec)

The statistical analysis was made by Mann-Whitney test.

Neither in normal vestibular system nor in unilateral right or left peripheral or bilateral peripheral lesions or in central lesions was the difference between right and left OKN-SPV statistically significant.

Although the differences between the right and left SPV of OKN were not statistically significant, in the everyday practice we often see the difference between the ASPV of right and left optokinetic nystagmus. When the difference is more than 10 degree/sec, we often estimate it as a pathological difference. Based on this estimation, in our practice the optokinetic eye movement was pathological in 9.53% of patients with normal vestibular function. In unilateral lesions, 17.42% of the OKNs were pathological.

In the bilateral peripheral vestibular dysfunction the ratio of the pathological eye-movements was 28%. In central vestibular lesions 22.72% of the patients had abnormal optokinetic eye movements.

The statistical analysis of the OKN-SPV shows that OKN to the right in left peripheral lesion is significantly decreased, compared with normal vestibular system OKN-SPV. The difference in the median values is statistically significant (P = 0,007). The decreasing of the right OKN- SPV in the bilateral peripheral lesion is statistically significant (Mann-Whitney test) (P = 0,005), so is the left OKN-SPV in the left peripheral lesion. (P = 0,010).

The left OKN is significantly decreased in the bilateral vestibular lesion (P = 0,012), while in the right vestibular lesion the left OKN-SPV changes are not significant.

According to the software of ICS computer system, the smooth pursuit eye movement disorder can be seen in a graphic form (Figure 3).

Figure 3.
Smooth pursuit eye movement
ICS Chartr® graphic result

up: normal smooth pursuit
down: destroyed smooth pursuit in central vestibular lesion.

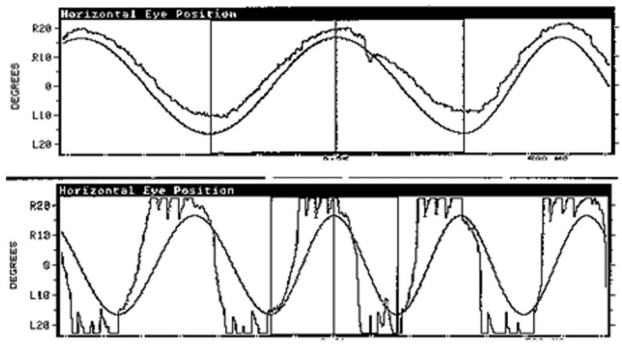


Table 4. Smooth pursuit eye movement results.

Vestibular disorders	Pathological %
Right peripheral	17,9
Left peripheral	22,8
Bilateral peripheral	28,0
Central dysfunction	41,6
Normal vestibular system	8,3

DISCUSSION

We didn't find explanation for the fact that during the same examination period there were many more peripheral lesions on the left side than on the right one.

There is an interesting fact that the slow phase velocity difference of the spontaneous nystagmus in left peripheral lesion is not statistically significant, while either in the right peripheral or in the bilateral peripheral lesion the spontaneous nystagmus SPV is significantly different from the spontaneous nystagmus SPV of normal vestibular system. In the same database with the same multiple comparisons test the nystagmus frequencies in the left peripheral and in the bilateral lesion were statistically different from the normal.

The statistical analysis of the OKN - SPV shows, that OKN to the right in left peripheral lesion is significantly decreased, compared with normal vestibular system OKN-SPV. The left OKN is significantly decreased in bilateral vestibular lesion ($P = 0,012$), while in right vestibular lesion the left OKN-SPV changes are not significant.

Do these facts suggest the suspicion that some cortical or subcortical influence might regulate the slow phase velocity in the left peripheral lesion? Why doesn't this mechanism work in the right peripheral lesions? Do these mechanisms have any connection with the brain dominance? These questions need further investigation.

We often observe in the everyday clinical practice that patients with central vestibular lesion have a hypersensitivity of caloric response with severe vegetative symptoms. According to our observations, in the central dysfunctions the average caloric ASPV is increased (19.7 degree/sec). It suggests that in central vestibular lesions the central inhibiting mechanisms of the caloric response are impaired.

Our question was the sensitivity of the optokinetic nystagmus compared with the smooth pursuit eye movement in several disorders of the vestibular system.

The results (Table 5) show that in cases of normal vestibular system, the ratio of pathological eye movements was fewer than 10% with both the OKN and the smooth pursuit tests.

Table 5. Sensitivity of eye movement examinations.

Vestibular disorders	Optokinetic eye movements	Smooth pursuit eye movements
Normal vestibular system	9.53%	8.3%
Unilateral peripheral lesions	17.42%	20.3%,
Bilateral peripheral	28%.	28%
Central dysfunction	22.72%	41.6%.

The visual system disturbances or neuro-ophthalmological disturbances can cause these results.

In unilateral peripheral lesion the smooth pursuit eye movement examination is more sensitive than OKN, but the difference between the two methods is small. In the bilateral lesion the ratio of the pathological OKN and smooth pursuit eye-movement is the same. In peripheral lesion the frequency of the pathological eye-movement disorder is higher than in normal vestibular system, examined either with OKN, or with smooth pursuit.

In central vestibular dysfunction the OKN disturbance occurred more frequently than in unilateral peripheral lesion, and normal vestibular system, but less frequently than in bilateral lesion. The pathological ratio of smooth pursuit eye movement is higher in central vestibular dysfunction than in the other types of vestibular lesion. It means that in central vestibular dysfunction the smooth pursuit eye movement examination is more sensitive than the OKN.

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