Study on Optokinetic Nystagmus

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Optokinetic nystagmus is a physiological response. It provides an example of the activity of the fixation and re-fixation reflexes. When an object centered on the fovea is moved to the periphery, the eyes slowly follow it in a series of rapid saccadic movements until it disappears or until other object of interest falls on the retinal periphery; then the eye turns back in a quick smooth movement and fixates the newcomer. If such objects in a series are successively brought across the visual field, the eyes travel outward and back in a biphasic jerk rhythm. In other words, optokinetic nystagmus occurs in a normal person when successive moving objects excite attention as they traverse the visual field.

Optokinetic nystagmus is commonly observed in everyday life as when looking out of the window of a train (railway or train nystagmus) or looking of processions of cavalry.

Since the beginning of this century the optokinetic nystagmus is of interest to ophthalmologist, the neurologist and the otologist. Wirths (1911)[1] is the first to describe the optokinetic nystagmus for making a diagnosis of conjugate paresis of the eyeballs. The clinical importance of the optokinetic nystagmus for neurology has been emphasized by Fox and Holmes (1926)[2] and Stenvers (1926)[3]. Stenvers reported that the lesion damaging either of the centers in the occipital and frontal lobes or the pathways between them, occurs loss of the optokinetic reaction towards the opposite side. Fox and Holmes deny the existence of the frontal center in this phenomenon.

It is of importance to note that in the otological field Barany (1921)[4] and Brunner (1932)[5] were the first to study optokinetic nystagmus, particularly for localization of a spontaneous nystagmus. They studied the influence of optokinetic nystagmus upon a spontaneous nystagmus, using a turning optical wheel. Working independently of each other, they came to the same conclusion: If a patient with spontaneous nystagmus showed a typical optokinetic reaction, the test could not be used for the localization of the spontaneous nystagmus. If the optokinetic reaction was atypical or inverse, the conclusion could be drawn that the spontaneous

nystagmus was of a non-labyrinthine origin.

As to the interference of spontaneous nystagmus with induced ocular and induced labyrinthine nystagmus, some otologists stated that any spontaneous ocular nystagmus can easily be influenced by an induced ocular nystagmus, but not so easily by a caloric nystagmus. On the other hand, spontaneous nystagmus of central labyrinthine origin can easily be influenced by caloric nystagmus, but not easily by an induced ocular nystagmus.

METHOD OF OPTOKINETIC TEST

The author come, now, to consider the method of optokinetic test. Before considering the method of optokinetic nystagmus the author will state an indication of this test. According to Fischer (1956)[6] if the otologist observes spontaneous nystagmus in a patient and cannot determine whether it is of a labyrinthine or nonlabyrinthine origin, he should employ the optokinetic test. His opinion seems to be reasonable as regards indication of this test.

In general, for tests of the optokinetic nystagmus an optic wheel, cylinder and drum are used. Brunner (1922)[7] devised the following apparatus: It is made of pasteboard in the shape of a flat cylinder, 70 cm. in diameter and 30 cm. in height. There are six vertical black stripes, 9 cm. wide and about 30 cm. apart. It can be placed on a tripod by means of a cross or hung from the ceiling.

In normal subjects an optokinetic nystagmus is elicited in the direction towards the opposite to that of turning cylinder. If the cylinder is turned in a horizontal plane, for example to the right, an optokinetic nystagmus to the left is elicited; on turning the cylinder to the left, a nystagmus to the right occurs. Physiologically the frequency of the optokinetic nystagmus increases with greater rotation speed of cylinder and with closer spacing of the black stripes. It is generally accepted by many authors that optokinetic nystagmus follows the same rules as the labyrinthine nystagmus. For example, it will be increased by looking in the direction of the fast phase and decreased by looking in the direction of the slow phase. But attention influences the optokinetic nystagmus and distraction of the fixating attention inhibits this nystagmus.

As to the differentiation of labyrinthine and nonlabyrinthine spontaneous nystagmus, Fischer (1956)[6] stated that when examining a case with spontaneous nystagmus, the optokinetic nystagmus may occur in one of the three ways: 1. On turning the optical cylinder, a nystagmus

is elicited in the direction opposite to that of turning; normal optokinetic nystagmus. 2. On turning the cylinder, a nystagmus is observed in the same direction as that of the turning; atypical nystagmus or inversion. 3. On turning the cylinder, no eye movement; a typical reaction. The normal optokinetic nystagmus is of no help in diagnosing the origin of the spontaneous nystagmus of the case. The atypical nystagmus or inversion indicates a nonlabyrinthine origin of the spontaneous nystagmus of the case.

Recently Suzuki (1962)[8] presented a new type of clinical test utilizing optokinetic nystagmus, optokinetic pattern test (OKP test), which should greatly aid in diagnosis of central neural disorders including vestibular disturbances. According to his description his apparatus and test method are as follows: Optokinetic stimulation is given by an electrically controlled rotating drum, 1.8 m in diameter, 1.2 m in height, with a vertical rotatory axis and twelve black vertical stripes 3 cm wide interiorly. The drum can be rotated with a required constant angular velocity from 0° to 180° sec^{-1}. The angular speed of the drum is changed continuously with an angular acceleration of 4° sec^{-2}.

In our Department since five years we carried out the examination of optokinetic nystagmus by using Suzuki's apparatus mentioned above as a clinical routine test. The subject takes a chair at the center of the drum and is told to look at exactly the moving strips running horizontally across his visual field. Turning of the drum is accelerated from 0° to 150° sec^{-1} within a time period of 37.5 sec and immediately decelerated to zero with identical but negative acceleration. This is followed by another turning to the opposite direction.

Nystagmic movements induced by optokinetic stimulation were registered by a direct-writing, four-channel-recorder electronystagmograph (ENG). Paper was fed at 0.1 cm sec^{-1}. Time constant was 3 sec for eye movement and 0.03 sec in order to eye speed.

MATERIAL

In the present paper the author proposes to reconsider in great detail clinical features of optokinetic nystagmus, particularly, relationship between gaze nystagmus and optokinetic one, and this study is based on the examinations, made possible through the courtesy of our colleagues at Yamaguchi University Hospital, of 88 cases in which we have the following two groups: Group I, 47 cases complaining vertigo without gaze, spontaneous and positional nystagmus; and Group II, 41 cases complaining

vertigo with gaze nystagmus. These we have examined in the course of the last three years. Age distribution is as follows:

Age	0-20	21-30	31-40	41-50	51-60	61-70	70-	Total
Group I	3	5	17	6	4	9	3	47
Group II	6	4	5	11	8	5	2	41

We find 24 males and 23 females in Group I, and 27 males and 14 females in Group II.

RESULTS

Optokinetic nystagmus obtained by optokinetic pattern test (OKP test) are divided into the following patterns: 1. Normal pattern; and 2. Abnormal patterns type I, II, III and IV.

In normal pattern the author find out, during the slow phase of nystagmus, that the eyes move with a close approximation to the angular velocity of the drum, so that the electronystagmograph of this pattern shows the shape illustrated in Fig. I. It is of importance to note that in normal pattern no remarkable directional differences between turning to the right and to the left of drum.

In abnormal type I, the author find out a definite directional difference in both turning the drum as shown in Fig. I. This difference seems to be due to directional preponderance of optokinetic reaction.

In abnormal type II, the fusion limit of optokinetic nystagmus deteriorates in the center of ENG of both turning the drum as shown in Fig. I.

In abnormal type III, as illustrated in Fig. I, the author find out that the slow phase of optokinetic nystagmus is restrained in during whole response.

In abnormal type IV we find out that the direction of optokinetic nystagmus disappears in response.

Optokinetic patterns in 47 cases complaining vertigo without gaze, spontaneous and positional nystagmus are tabulated in Table I. In Table I, we find out normal pattern in twenty two of 47 cases, abnormal pattern type I in only two, type II in nine, type III in eleven. In addition we find out irregular patterns, such as normal by turning the drum to the right side and type II by turning the drum to the left side in two, and normal by turning the drum to the right side and type III by turning the drum to the left side in one.

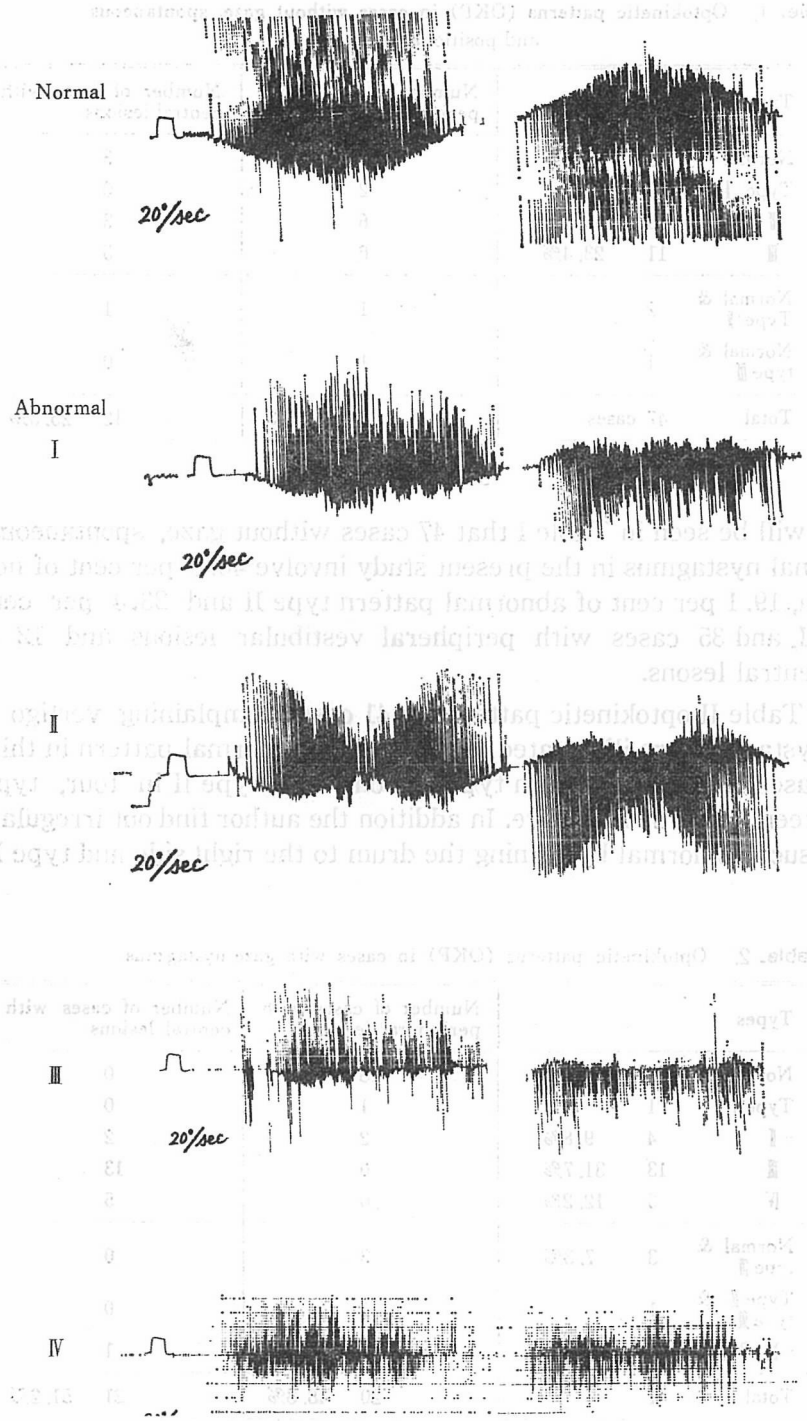

Fig. 1. Types of optokinetic pattern (OKP)

Table. 1. Optokinetic patterns (OKP) in cases without gaze, spontaneous and positional nystagmus

Types			Number of cases with peripheral lesions		Number of cases with central lesions	
Normal	22	46.8%	19		3	
Type I	2		2		0	
-II	9	19.1%	6		3	
-III	11	23.4%	6		5	
Normal & Type II	2		1		1	
Normal & type III	1		1		0	
Total	47 cases		35	74.5%	12	25.5%

It will be seen in Table I that 47 cases without gaze, spontaneous and positional nystagmus in the present study involve 46.8 per cent of normal pattern, 19.1 per cent of abnormal pattern type II and 23.4 per cent of type III, and 35 cases with peripheral vestibular lesions and 12 cases with central lesons.

In Table II optokinetic patterns in 41 cases complaining vertigo with gaze nystagmus are illustrated. Table II shows normal pattern in thirteen of 41 cases, abnormal pattern type I in only one, type II in four, type III in thirteen and type IV in five. In addition the author find out irregular patterns, such as normal by turning the drum to the right side and type III by

Table. 2. Optokinetic patterns (OKP) in cases with gaze nystagmus

Types			Number of cases with peripheral lesions		Number of cases with central lesions	
Normal	13	31.7%	13		0	
Type I	1		1		0	
-II	4	9.8%	2		2	
-III	13	31.7%	0		13	
-IV	5	12.2%	0		5	
Normal & type III	3	7.3%	3		0	
Type II & type III	1		1		0	
-III & IV	1		0		1	
Total	41		20	48.8%	21	51.2%

turning the drum to the left side in three, type II by turning the drum to the left side and type III by turning the drum to the right side in one, and type III by turning the drum to the right side and type IV by turning the drum to the left side in one.

It is of importance to note that in Table II 41 cases with gaze nystagmus in the present study contain 31.7 per cent of normal pattern, 9.8 per cent of abnormal pattern of type II, 31.7 per cent of type III and 12.2 per cent of type IV, and 20 cases with peripheral vestibular lesions and 21 cases with central lesions.

Classification of normal pattern of optokinetic nystagmus in 22 cases

Table. 3. Normal of optokinetic pattern(OKP) in caes without gaze, spontaneous and positional nystagmus

Number of cases with peripheral lesions		Number of cases with central lesions	
Vestibular neuritis	7	Head trauma	2
Meniere's disease	6	Cerebral arteriosclerosis	1
Harada's disease	2	Total	3
Hunt syndrome	1		
Circumscribed labyrinthitis	1		
Deafness	1		
Labyrinthine lesion due to cervical trauma	1		
Total	19	Sum total	22

Table. 4. Abnormal types Ⅰ and Ⅱ of Optokinetic pattern (OKP) in cases without gaze, spontaneous and positional nystagmus

	Number of cases with peripheral lesions		Number of cases with central lesions	
Type Ⅰ	Meniere's disease	2		0
	Total	2		
Type Ⅱ	Meniere's disease	4	Head trauma	2
	Vestibular neuritis	1	Hypertension	1
	Lbyrinthine lesion due to head trauma	1		
	Total	6	Total	3
			Sum total	9

without gaze, spontaneous and positional nystagmus is given in Table III and in 13 cases with gaze nystagmus is shown in Table IV. As shown in Table III 22 cases involve 3 cases with central lesions, although 13 cases with gaze nystagmus contain no case with central lesion.

Table IV and V illustrate classifications of abnormal patterns type I, II and III of optokinetic nystagmus in cases without gaze, spontaneous and

Tab e. 5. Abnormal type Ⅲ and others of optokinetic pattern (OKP) in cases without gaze, spontaneous, and positional nystagmus

	Number of cases with peripheral lesions		Number of cases with central lesions	
Type Ⅲ	Meniere's disease	2	Cerebral arteriosclerosis	2
			Cardiovascular lesion in the brain	1
	Harada s disease	2	Hg. poisoning	1
			Hormonal lesion	1
	Vestibular neuritis	1	Total	5
	Streptomycin poisoning	1		
	Total	6	Sum total	11
Normal and type Ⅱ	Head trauma	1	Cerebral arteriosclerosis	1
Normal and type Ⅲ	Vestibular neuritis	1		

Table. 6. Normal of optokinetic pattern (OKP) in cases with gaze nystagmus

Number of cases with peripheral lesions		Number of cases with central lesions
Vestibular neuritis	2	
Meniere's disease	2	
Sudden deafness	2	
Hunt syndrome	1	
Benign positional nystagmus	1	
Harada's disease	1	
Chronic middle ear infection	3	
Inner ear disturbance	1	
Total	13	

Table. 7. Abnormal types Ⅰ, Ⅱ and Ⅲ of Optokinetic pattern (OKP) in cases with gaze nystagmus

	Number of cases with peripheral lesions		Number of cases with central lesions	
Type Ⅰ	Meniere's disease	1		
—Ⅱ	Vestibular neuritis	1	Head trauma	1
	Circumscribed labyrinthitis	1	Cardiovascular lesion in the brain	1
	Total	2	Total	2
—Ⅲ			Sum total	4
			Arteriosclerosis in the brain	3
			Head trauma	2
			Congenital nystagmus	2
			Cerebellar lesion	2
			Fischer's syndrome	1
			Wallenberg's syndrome	1
			Hg. poisoning	1
			Strabismus	1
			Total	13

Table. 8. Abnormal type Ⅵ and others of optokinetic pattern (OKP) in cases with gaze nystagmus

	Number of cases with peripheral lesions		Number of cases with central lesions	
Type Ⅳ			Congenital nystagmus	2
			Cerebellar tumor	1
			Head trauma	1
			Central vestibular disturbance	1
			Total	5
N;rmal and type Ⅲ	Streptomycin poisoning	1	Hormonal disease	1
	Meniere's disease	1		
	Total	2	Total	1
			Sum total	3
Type Ⅱ and Ⅲ	Vestibular neuritis	1		
—Ⅲ and Ⅳ			Brain stem lesion	1

positional nystagmus. Table VI gives classification of abnormal patterns type I, II and III of optokinetic nystagmus in cases with gaze nystagmus. As shown in Table V, 11 cases with abnormal pattern type III involve 6 cases with peripheral vestibular lesions and 5 cases with central lesions, on the other hand as shown in Table VII, 13 cases with abnormal pattern type III contain no case with peripheral vestibular lesion.

In addition, as shown in Table VIII, 5 cases of abnormal type IV of optokinetic nystagmus with gaze nystagmus involve no case with peripheral vestibular lesion.

COMMENT

As shown in the results of this paper (Table II), all cases with gaze nystagmus showing normal pattern of optokinetic nystagmus were peripheral in origin, notwithstanding no central lesions in origin. On the other hand all cases with gaze nystagmus providing abnormal patterns type III and IV of optokinetic nystagmus were central lesions in origin.

As above mentioned in this paper, Barany (1921)[5] and Brunner (1932)[5] already stated that if cases with spontaneous nystagmus show atypical or inverse optokinetic reactions, the spontaneous nystagmus was of a non-labyrinthine origin.

It can be concluded, beyond any doubt, that the results obtained by this study agree with their opinions.

Finally, judging from the comparison of percentage (31.7%) of normal pattern in cases presenting gaze nystagmus (Table II) with percentage (46.8%) of normal pattern in cases presenting no nystagmus (Table I), it seems to be emphasized in all probability that the gaze nystagmus inhibits the optokinetic reactions.

SUMMARY

Optokinetic reactions were studied by OKP test in 88 cases complaining vertigo; Group 1:47 cases with no nystagmus; and Group 2: 41 cases with gaze nystagmus. It seems to be concluded that the gaze nystagmus inhibits the optokinetic reaction, judging from the results obtained.

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REFERENCES

1) Wirths, M.: Beitrag zum klinischen Bilder assoziierten Blicklaehmung. *Ztschrif. Augenh.*, **26** : 318, 1911.
2) Fox, L., and Holmes, G.: Optic nystagmus and its value. *Brain*, **49** : 333, 1926.
3) Stenvers, W.: On the optic nystagmus. *Acta oto-laryng.*, **8** : 545, 1926.
4) Barany, R.: Klinik und Theorie des Eisenbahner Nystagmus. *Acta oto-laryng.*, **3** : 260, 1921.
5) Brunner, H.: Zur klinischen Bedeutung der Inversion. *Arch. f. Ophth.*, **129** : 30, 1932.
6) Fischer, J.: *The labyrinth.* P. 133. Graune & Stratton, London, 1956.
7) Brunner, H.: Zur klinischen Bedeutung des optischen Drehnystagmus. *Klin. Monatsh. Klin. f. Augenh.*, **68** : 783, 1922.
8) Suzuki, J.: Optokinetic pattern test. *Acta oto-laryng.*, **54** : 49, 1962.