**Sensitivity of nasal and temporal hemiretinas in latent nystagmus and strabismus evaluated using the light reflex**

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**Abstract**

To determine visual sensitivity in patients with latent nystagmus with or without esotropia or exotropia, and in patients with esotropia or exotropia, pupillary reactions to 5 degrees diameter light stimuli given to the retina at the fovea (centre), at 5 degrees nasal to the centre, and at 5 degrees temporal to the centre, were measured using electronic infrared pupillography; then pupillary constriction rates and maximum velocity of pupillary constrictions were calculated and analysed. In non-dominant eyes, visual sensitivities of the nasal retina and temporal retina were higher compared with normal eyes, and they also tended to be higher than the sensitivities at the fovea (centre), particularly in patients with latent nystagmus with esotropia. In patients with esotropia or exotropia, visual sensitivities in non-dominant eyes of the nasal retina, and temporal retina increased to a level similar to those at the fovea (centre). The magnitude of these abnormalities was greater in patients with latent nystagmus with esotropia, those with latent nystagmus, and those with latent nystagmus with exotropia, esotropia, and exotropia, in that order. From these results, it was assumed that this imbalance is transmitted to the motor system, resulting in the onset of latent nystagmus.

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Latent nystagmus is a nystagmus appearing with a fast phase beating toward the fixating eye while one eye is covered — that is, it develops when binocular viewing is disturbed. Although multiple abnormalities in the visual and motor systems are found to involve the onset of latent nystagmus, and many theories have been advocated, none of these theories fully explains the mechanism of this disease. Latent nystagmus is frequently complicated by esotropia or dissociated vertical deviation (DVD). An abnormality in the optomotor effect transmission pathway has been reported to be a cause of esotropia, whereas it is inferred from studies of visual evoked potential (VEP) that an abnormality in the optic chiasma, resembling that in albinos, causes DVD. In order to clarify visual dysfunction in latent nystagmus, we recorded and analysed light reactions according to selected retinal regions in patients with latent nystagmus, and also in those with strabismus. We obtained interesting results that indicated the presence of an imbalance in the visual system, as described here.

**Material and methods**

Sixteen patients with latent nystagmus without DVD, comprising four with latent nystagmus alone, seven complicated by esotropia, and five complicated by exotropia, were selected as subjects, excluding those found to have nystagmus under binocular viewing (manifest latent nystagmus) on photoelectric oculography, and six patients with esotropia and six with exotropia were also examined, in addition to six normal subjects with good visual acuity.

The eye with less nystagmus with monocular viewing was identified as the dominant eye by reference to visual acuity and the results of the hole in card test. The patients with latent nystagmus were 10–20 years of age, with a mean of 13-5 years; the corrected visual acuity of both dominant and non-dominant eyes was 20/200 to 20/16. Of these patients with latent nystagmus, those complicated by esotropia ranged from 9 to 19 years of age, with a mean of 13-2 years; the corrected visual acuity of the dominant eye was 20/30 to 20/16, and that of the non-dominant eye ranged from 20/200 to 20/20; the eye position was 6–45 prism dioptries, with a mean of 25-2 prism dioptries. The patients complicated by exotropia ranged from 7 to 13 years of age, with a mean of 10-8 years; the corrected visual acuity of the dominant eye was 20/25 to 20/20, that of the non-dominant eye was 20/200 to 20/20; the eye position was 18–30 prism dioptries, with a mean of 20-8 prism dioptries. The ages of the patients with esotropia ranged from 7 to 23 years, with a mean of 11-7 years; the corrected visual acuity of the dominant eye was 20/16, and that of the non-dominant eye was 20/25 to 20/16; the eye position was 20–60 prism dioptries, with a mean of 28-7 prism dioptries; they all had infantile esotropia. The patients with exotropia included three with constant exotropia and three with intermittent exotropia. The former patients were all 7 years old and had a corrected visual acuity of 20/16 in both dominant and non-dominant eyes; the eye position was 20–50 prism dioptries, with a mean of 31-6 prism dioptries. The latter patients with intermittent exotropia ranged from 11 to 12 years of age, with a mean of 11-6 years; the
corrected visual acuity was 20/20 to 20/16 in both dominant and non-dominant eyes; the eye position was 16 to 35 prism dioptres, with a mean of 27.0 prism dioptres. None of the patients had had treatment for amblyopia or surgical treatment.

The retina was stimulated with a light spot (5 degrees in diameter) at the fovea, at 5 degrees nasal to the centre of the fovea, and at 5 degrees temporal to the centre of the fovea, using the Maxwellian optical system under non-expectant condition; and direct pupillary reactions to light were measured five times each using electronic infrared pupillography (Takei Kiki Co, Ltd). After that, the pupillary constriction rate (CR %—pupil area before stimuli (mm²)/amplitude of constriction (mm²)) and the maximum velocity of pupillary constriction (MVC (mm²/s)) were calculated and analysed. In consideration of diurnal variations, measurement was almost always performed at about 3 pm in a dark room. After dark adaptation for 15 minutes, each eye, made to gaze at the centre using a staring light emitting diode, was first stimulated with light with both eyes open, to confirm the absence of a convergence reaction, and pupillography was then undertaken. Each light stimulus was provided at 2000 trolands for 0.5 seconds.

With the mean values in the dominant and non-dominant eyes of normal subjects stimulated at the centre (fovea) taken to represent 100%, values obtained from the dominant eye of each patient stimulated at the centre and on the nasal and temporal sides were compared with the mean value in the normal dominant eyes stimulated at the centre, and those obtained from the non-dominant eye of each patient were also compared with the mean value in the normal non-dominant eyes stimulated at the centre.

Results

LIGHT REFLEX OF NORMAL SUBJECTS (FIG 1)
An assessment was made regarding the light reflex in the normal subjects, to confirm if the light reflex can be a parameter to represent retinal sensitivity. Regarding the dominant eye in the normal subjects, the mean with standard deviation of CR in the centre was 100% (SD 22.8%), if the mean at the centre is set as representing 100%. CRs at the nasal and temporal sites were 72.9% (37.4%) and 75.1% (33.9%), respectively, showing that the rate is higher at the centre. MVCs were 100% (26.7%) at the centre, 67.5% (32.7%) at the nasal site, and 74.5% (26.7%) at the temporal site, showing that the velocity was highest in the centre. Regarding the non-dominant eye, the mean value at the centre of the non-dominant eye was used as a 100% criterion. CRs were 100% (25.7%) at the centre, 75.1% (20.8%) at the nasal site, and 64.8% (37.2%) at the temporal site, showing the highest rate at the centre. MVCs were 100% (35.1%) at the centre, 72.6% (18.9%) at the nasal site, and 59.4% (33.7%) at the temporal site, showing the highest velocity at the centre, similar to CR. In comparing the nasal and temporal sites between the dominant and non-dominant eyes, no evident difference was observed in a range of 5 degrees from the centre. Also no evident significant difference was observed between the dominant and non-dominant eyes.

LIGHT REFLEX WITH LATENT NYSTAGMUS (FIG 2)
Regarding the dominant eyes in patients with latent nystagmus, those with latent nystagmus with esotropia, and those with latent nystagmus with exotropia, esotropia, and exotropia, no significant difference was observed at the centre, nasal, or temporal site, compared with CR and MVC in the normal subjects, and the centre showed a trend that the reflex is larger, compared with those in the nasal and temporal sites. On the other hand, in the non-dominant eye of latent nystagmus patients, CRs (121.8% (10.5%)) and MVCs (107.5% (10.8%)) at the centre were observed within the range of the normal subjects, but CRs were 117.7% (21.3%) at the nasal site and 137.7% (17.7%) at the temporal site; these values were significantly high (p<0.05) compared with the rates at the nasal and temporal sites in the normal subjects, showing a trend of the rates being higher than at the centre. MVCs were 129.3% (14.8%) at the nasal site and 125.6% (16.1%) at the temporal site: these values were significantly higher than the velocities at the nasal and temporal sites in normal subjects (p<0.05). Furthermore, the visual sensitivities at the nasal and temporal sites showed a trend of being higher than at the centre in the non-dominant eye. It was found that the visual sensitivities at the nasal and temporal sites were about the same.

LIGHT REFLEX WITH LATENT NYSTAGMUS WITH ESOTROPIA (FIG 3)
CRs (116.1% (14.9%) and MVCs (111.0% (24.3%)) in the centre of the non-dominant eye were observed to be within the normal range, but CRs were 126.4% (15.3%) at the nasal site and 124.2% (14.1%) at the temporal site, showing a trend of being significantly high (p<0.01 and p<0.05, respectively) compared with the nasal and temporal sites in the normal subjects. MVCs
Sensitivity of nasal and temporal hemiretinas in latent nystagmus and strabismus evaluated using the light reflex

were 130.4% (21.3%) at the nasal site and 119.3% (16.9%) at the temporal site; these values were significantly high \((p<0.01\) and \(p<0.05\), respectively) compared with the nasal and temporal sites in the normal subjects. Furthermore, the visual sensitivities at the nasal and temporal sites showed a trend of being higher than at the centre. It was found that the visual sensitivities at the nasal and temporal hemiretinas were nearly the same.

**LIGHT REFLEX WITH LATENT NYSTAGMUS WITH EXOTROPIA (FIG 4)**

In the non-dominant eye, no significant difference was observed in CR or MVC in the centre or nasal and temporal sites, compared with the normal subjects, but CR showed a trend of being slightly high in the nasal and temporal sites in comparison with the values in the centre: 88.3% (16.5%) in the centre, 91.8% (12.9%) in the nasal site, and 88.0% (12.3%) in the temporal site. MCV also exhibited a trend of being slightly higher in the nasal and temporal sites than in the centre: 88.3% (8.1%) in the centre, 102.9% (22.5%) in the nasal site, and 99.4% (20.1%) in the temporal site. It was found that the visual sensitivities in the nasal and temporal hemiretinas were nearly the same.

**LIGHT REFLEX WITH ESOTROPIA (FIG 5)**

In the non-dominant eye, no significant difference was observed in CR or MVC in the centre or nasal and temporal sites, compared with the normal subjects, but CRs and MCVs in the nasal and temporal sites approached the levels of the respective values in the centre: CRs were 71.3% (4.4%) in the centre, 62.4% (14.5%) in the nasal site, and 76.1% (7.8%) in the temporal site; and MCVs were 97.2% (10.9%) in the centre, 92.1% (10.1%) in the nasal site, and 90.7% (19.1%) in the temporal site. It was found that the visual sensitivities in the nasal and temporal sites were nearly the same.

**LIGHT REFLEX WITH EXOTROPIA (FIG 6)**

No significant difference was observed in CR or MVC in constant exotropia and intermittent exotropia. In the non-dominant eye, a trend of the values being higher in the centre than in the nasal and temporal sites was observed, similar to the dominant eye. However, CR and MCV were significantly high \((p<0.05)\) in the nasal and temporal sites, compared with the normal subjects: CR was 121.0% (17.5%) in the nasal site and 126.4% (22.5%) in the temporal site, and MCV was 105.3% (23.9%) in the nasal site and 117.2% (22.2%) in the temporal site. The values approached the levels of CR (137.2% (12.3%)) and MVC (125.5% (22.1%)) in the centre. It was found that the visual sensitivities in the nasal and temporal sites were nearly the same.

**Discussion**

Although the mechanism of the onset of latent nystagmus has remained obscure, many theories have been advocated. For example, cortical binocularity disturbance,\(^\text{14}\) disorder of the subcortical optokinetic system,\(^\text{7}\) a disorder of proprioception,\(^\text{1}\) and egocentric localisation disorder\(^\text{16}\) have all been suggested as causes of latent nystagmus. Monocular optokinetic asymmetries seen in latent nystagmus are also observed in normal infants during a certain development period,\(^\text{11}\) as well as in lower mammals, such as the afoveate rabbit,\(^\text{10}\) in monkeys after removal of the occipital lobes,\(^\text{10}\) and in patients with infarction of bilateral occipital lobes.\(^\text{17}\) It is interesting to note that the direction of latent nystagmus alters in a patient who is blind in one eye when he attempts to look at an object with the blind eye.\(^\text{19}\) Thus, abnormalities over wide areas, from the visual input system to the motor system, through propriocep-
of the extraocular muscle, the high-levelled visual centre, and the association area in the cerebral cortex, are thought to be associated with latent nystagmus.

In the present study, we recorded reactions to light stimuli given to selected retinal regions, which are used to evaluate the visual system in latent nystagmus and strabismus, and then we calculated CR and MVC. As a result, it was revealed for the first time that an imbalance of the visual system exists in latent nystagmus and strabismus; namely, the visual sensitivity of the retina on both nasal and temporal sides was higher in the patients than in the normal subjects, and it was also higher than the sensitivity at the centre. The magnitude of these abnormalities was greater in patients with latent nystagmus with esotropia, those with latent nystagmus, and those with latent nystagmus with exotropia, esotropia, and exotropia, in that order. These differences are thought to correspond to affected areas in the visual system.

Affrent visual and light reflex tracts run together through the retina, optic nerve, and the optic chiasma; they run two thirds of the way up the optic tract; and then they separate and take their own pathways. The visual fibre reaches the cerebral cortex through the lateral geniculate nucleus and optic radiation. The light reflex tract enters through the pretectum of the midbrain into the Edinger-Westphal nucleus. Although the light reflex is thought to be an important objective parameter for the determination of vision, particularly retinal function, the contribution of other cortical centres, such as optic radiation and the occipital lobe, has not been reported. In 1975, Aoyama measured objectively the distribution of visual sensitivity based on CR calculated from the light reflex in patients with retinohoroidal disease, optic neuropathy, and cerebral disease with bitemporal hemianopsia and homonymous hemianopsia. He reported that measurement of visual sensitivity distribution determined by light reflex was closely correlated with subjective visual field measurements and was very sensitive, even to a minute change in visual function, compared with subjective measures. Further, pupillary reactions seem to reflect not only reactions up to the lateral geniculate nucleus or subcortical and cortical reactions, but other reactions as well, because the pupil has been determined to respond to pattern and colour stimuli.

Visual system abnormality has also been reported in relation to esotropia, which is highly associated with latent nystagmus. Koenig observed that only one eye moved medially in infants with esotropia when the retina of that eye was lighted on the temporal side, whereas both eyes showed a conjugate movement towards the light when the retina was lit on the nasal side. He stated that monocular adduction became predominant in addition to conjugate movements, if these eye movements repeatedly occurred, leading to a tendency towards severer esotropia. Mitsu noted that monocular light stimulation in a dark room produced adduction in the stimulated eye in the presence of intermittent esotropia, whereas it did not cause any change or it produced abduction in the eye contralateral to
the stimulated eye in the presence of intermittent exotropia. He postulated that esotropia occurs because of a reflex of the medial rectus muscle of the corresponding eye, as Keiner mentioned, whereas esotropia is caused by contraction of the lateral rectus muscle of the contralateral eye as a result of optomotor effect of the extraocular muscle of one eye under light stimulus. He suggested deep involvement of the optomotor effect transmission pathway in strabismus.

The visual imbalance observed in this study was most remarkable in patients with latent nystagmus complicated by esotropia, and in those with latent nystagmus, which is often complicated by esotropia or DVD. As reported by Keiner and Mitsui, a disorder of the optomotor effect transmission pathway is thought to cause esotropia, and measurement of VEP suggests a disorder of the optic chiasma, resembling that seen in albinos, as a cause of DVD. From the results of the present study, it is inferred that the abnormal visual sensitivity determined by pupillary reaction in latent nystagmus appears as a primary phenomenon of an increase in visual sensitivity on the nasal and temporal sides, and that latent nystagmus occurs as a corrective nystagmus in response to esotropia caused by Keiner’s reflex, as observed in esotropia with monocular viewing. Based on the results of the present study and reports in the literature, we think that the four above mentioned factors are cooperatively involved in the onset of latent nystagmus, rather than that latent nystagmus is caused by an abnormality in visual sensitivity alone.

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