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THE UNIVERSITY OF SOUTH FLORIDA
COLLEGE OF ARTS AND SCIENCES

**THE RELATIONSHIP BETWEEN OPTOKINETIC NYSTAGMUS AND
CALORIC WEAKNESS**

BY

D'Arcy D. Cyr

An Audiology Doctoral Project

Submitted to the Graduate Faculty of the
Department of Communication Sciences and Disorders
in partial fulfillment of the requirements
for the degree of

Doctor of Audiology

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ABSTRACT

Cyr, D'Arcy D., Audiology Doctoral Project, University of South Florida, December, 2003. The relationship between optokinetic nystagmus and caloric weakness.

Traditionally, results from caloric testing and optokinetic nystagmus (OKN) testing are analyzed separately because caloric testing is a measure of peripheral function and OKN testing is considered to be a measure of central function. However, there is a connection between the visual system and the vestibular system in the vestibular nucleus of the brainstem. The purpose of this paper was to determine whether a relationship exists between optokinetic nystagmus results and unilateral caloric weakness results. This was determined by conducting a retrospective study of forty patients who exhibited a unilateral caloric weakness greater than or equal to twenty percent and symptoms consistent with an uncompensated vestibulopathy. Patients were later divided into two groups based on involved side. A control group consisting of ten subjects with no reported hearing or vestibular problems was also recruited. When the data of all subjects with a unilateral caloric weakness was considered together, no correlation was found between caloric response (right and left ear) and optokinetic results (gain and slow phase velocity). However, a potential trend emerged at the slow stimulus velocity (15°/s) when comparing the patients with a right caloric weakness to those with a left caloric weakness. Subjects with a right caloric weakness showed decreased OKN gain for the right eye with a right-moving stimulus compared to the subjects with a left caloric weakness. Alternatively, subjects with a left caloric weakness showed decreased OKN gain for the left eye with a left-moving stimulus compared to the subjects with a right caloric weakness. We conclude that interpretation of OKN along with caloric results may offer potential for identification and tracking of compensation after a unilateral loss of vestibular function, but further research is needed.

INTRODUCTION

Assessment of optokinetic nystagmus (OKN) and caloric responses are common components of the vestibular assessment battery. Optokinetic nystagmus is an involuntary oculomotor (eye movement) reflex elicited during sustained head rotation (Hain, 1997). The purpose of this reflex is to hold images of the observed world steady on the retina, specifically the fovea, during head movement to maintain accurate visual perception (Leigh & Zee, 1999). Another responsibility of the optokinetic system is to supplement the vestibular-ocular reflex (VOR) at low frequencies (<0.5 Hz) during sustained self-rotation of head movement (Angelaki & Bess, 1994). Natural, everyday head movements occur in the frequency range of 0.5 Hz – 5.0 Hz (Leigh & Zee, 1999). The purpose of the VOR is to maintain clear vision during everyday head movements and it is mediated by the semicircular canals and the otoliths (Hain, Ramaswamy, & Hillman, 2000). For example, when an individual is continuously rotated at a constant velocity in the dark, the initial vestibular compensatory eye movements generated by the VOR decline after approximately 45 s due to the mechanical properties of the semicircular canals, specifically the elastic properties of the cupula (Leigh & Zee, 1999). In other words, the nystagmus stops as the response from the vestibular system diminishes. The optokinetic system, along with the smooth pursuit system serves as the back-up system for this VOR, allowing gaze to remain stable even after the mechanical response from the vestibular system declines (Cohen, Henn, Raphan, & Dennett, 1981; Miles, 1998; Schweigart, Mergner, Evdokimidis, Morand, & Becker 1997).

It is not clinically feasible to measure OKN during sustained self-rotation without a rotational chair system. Therefore, artificial environments are typically created using optokinetic drums, lightbars, or video projectors. Of those three, video projectors are the only method capable of filling at least 90% of the visual field (Leigh & Zee, 1999). This is an important point because a visual field less than ninety percent will not provide the retinal stimulation that is needed to elicit true OKN (Hain, 1997). These types of instrumentation provide sensation of movement, while rotary chairs actually move the patient. Known as circularvection, this sensation of movement is caused by an illusion of self-rotation that is induced by optokinetic stimuli (Brandt, Dichgans, & Koenig, 1973). The eye movement of OKN is comprised of a slow component that moves in the direction of the stimulus and a fast component that moves in the direction opposite of the stimulus (Hain, 1997). The slow component of OKN is identified by the slow phase of the observed nystagmus, while the fast component of OKN is identified by the fast phase of the observed nystagmus. The fast phase of nystagmus defines the direction of nystagmus (Honrubia, 2000). While watching the moving stimuli, the optokinetic nystagmus gradually builds until the slow-phase velocity approximates the stimulus velocity (Collewyn, 1981). Eye velocity is simply the speed of the slow component of the OKN. Monitoring the eye movements with videonystagmography or electronystagmography allows the examiner to determine relationships between eye movement and optokinetic stimulus movement such as gain and eye velocity measures. In the case of OKN testing, gain is measured as the ratio of the slow phase component eye velocity to the visual target velocity (Hain, 1997).

Perhaps the most valuable subtest of the vestibular test battery is caloric response testing. This test allows the examiner to evaluate the right and left horizontal semicircular canals of the vestibular end organ separately. Through irrigation of the ear canal with either a water or an air stimulus, the temperature of the endolymph in the horizontal semicircular canal is manipulated. The change in temperature alters the direction of endolymph movement which, in turn, modulates neural activity originating from the ipsilateral vestibular end organ. The slow-phase velocities of the elicited nystagmus are compared using warm and cool irrigations of each ear. If the difference in the slow-phase velocities between ears is twenty percent or greater, a clinically significant unilateral caloric weakness is identified. The weakness is attributed to pathology within the horizontal semicircular canal system (the end organ itself), the vestibular portion of the eighth nerve, or the root entry zone of the eighth cranial nerve (Honrubia, 2000). While caloric testing will identify the damaged side, it will not further isolate the location of the damage (Jacobson, Newman, & Peterson, 1997).

Traditionally, results from caloric testing and OKN testing are analyzed separately because caloric testing is a measure of peripheral function and OKN testing is considered to be a measure of central function. However, there is a connection between the visual system and the vestibular system in the vestibular nucleus (VN) of the brainstem. Activity of the peripheral vestibular end organs affects the visual system through these connections. The majority of the cells in the VN respond to visual stimuli (Honrubia, 2000). Neurons in the VN that respond to head rotation are also excited by optokinetic stimuli in monkeys (Boyle, Büttner, & Markert, 1985; Henn, Young, & Finley, 1974; Waespe & Henn, 1977). It is important to mention that the VN also

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receives connections from other sensory systems and centers such as the proprioceptive system and the cerebellum. Therefore, information leaving the VN reflects the complex interaction of several afferent systems (Baloh & Honrubia, 2001).

There are four major divisions in the VN, the medial vestibular nucleus (MVN), the lateral vestibular nucleus (LVN), the inferior or descending vestibular nucleus (DVN), and the superior vestibular nucleus (SVN). All three semicircular canals have connections in a small area in the ventromedial portion of the SVN. This area contains vestibulo-ocular neurons that have projections to the abducens, oculomotor, and trochlear nuclei (Leigh & Zee, 1999). These neurons in the SVN run along the ipsilateral and contralateral medial longitudinal fasciculus (MLF) where they innervate the motor nuclei of the extrinsic eye muscles. Due to the afferent and efferent connection patterns, it is the SVN that operates as a major relay center for ocular reflexes that are governed by the semicircular canals (Baloh & Honrubia, 2001).

Given this connection and the fact that some neurons in the VN respond to head movement and optokinetic stimuli, it is potentially important to evaluate caloric and OKN results together. Interpretation of these results together may provide some insight into a patient's compensation of peripheral vestibular insult. Vestibular compensation is an additional role of the VN. Compensation is the ability of the central nervous system to recover from the neurological effects of a labyrinthine pathology (Curthoys & Halmagyi, 1995). In a normal system, the two vestibular end organs produce symmetrical output of afferent information that is routed to the VN. An acute unilateral pathology will cause an imbalance between the normally symmetrical neural activity produced by the peripheral end organs. Specifically, the ipsilateral neurons lose their spontaneous activity while

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healthy contralateral neurons lose their inhibitory input (Baloh & Honrubia, 2001). This causes an increase in spontaneous activity in the healthy vestibular system and a decrease of spontaneous activity in the pathological system (Precht & Dieringer, 1985). It is this mismatch in signals that causes the classic symptoms of vertigo, nystagmus, and nausea (Shepard & Telian, 1996).

The asymmetrical signal that arises from the labyrinths is sent to the VN where it is compared to input from the visual system (Honrubia, 2000). Because a mismatch exists between these two systems, the brainstem recognizes a conflict. This is why following complete vestibular lesions, a minor asymmetry in OKN occurs (Hain, 1997). The system will try to correct itself by restoring the tonic activity in the ipsilateral vestibular nucleus. Once compensation has occurred in the VN, the brainstem will no longer interpret the asymmetrical activity as a mismatch between the visual and vestibular systems and symmetrical OKN will be restored. In other words, when an individual first experiences a vestibular lesion, an OKN abnormality is expected. As compensation occurs over time, this abnormality in OKN eventually disappears. A caloric weakness will still be measured even though compensation has occurred because the restoration of neural activity happens above the peripheral level. Nevertheless, the peripheral end organ remains damaged (Shepard & Telian, 1996).

The purpose of this paper is to determine whether a relationship exists between optokinetic nystagmus and unilateral caloric weakness results. Since it is known that peripheral vestibular input does influence OKN, it is likely that another way to utilize information from the vestibular test battery would be to evaluate the caloric response along with the OKN response. If these test results are analyzed concurrently, it may be

possible to predict whether an individual has experienced vestibular compensation. We hypothesize that individuals with unilateral caloric weakness will demonstrate abnormalities on OKN responses due to the conflict of information between the visual and vestibular system that is occurring at the level of the vestibular nucleus.

METHOD

Subjects

The relationship between unilateral caloric weakness and optokinetic nystagmus was determined retrospectively for a group of 40 subjects previously seen for assessment at the American Institute of Balance. Patient charts were selected based on the presence of a caloric weakness greater than or equal to twenty percent and findings and symptoms consistent with an uncompensated vestibulopathy. Objective findings attributed to an uncompensated vestibulopathy included degraded dynamic visual acuity, positional nystagmus, post head-shake nystagmus, gaze nystagmus and/or an asymmetrical vestibular autorotation test (VAT). The number of subjects with a specific finding is shown in Figure 1. Subjective symptoms of an uncompensated vestibulopathy included, but were not limited to, motion-provoked dizziness (oscillopsia), unsteadiness, attack(s) of vertigo, and/or an overall sense of dizziness. The number of subjects exhibiting particular symptoms is shown in Figure 2. The category “Other” included those symptoms which were noted by only a few individuals, such as motion intolerance, visual dependence, surface dependence, nausea during testing, and a feeling of drifting or “being pulled”.

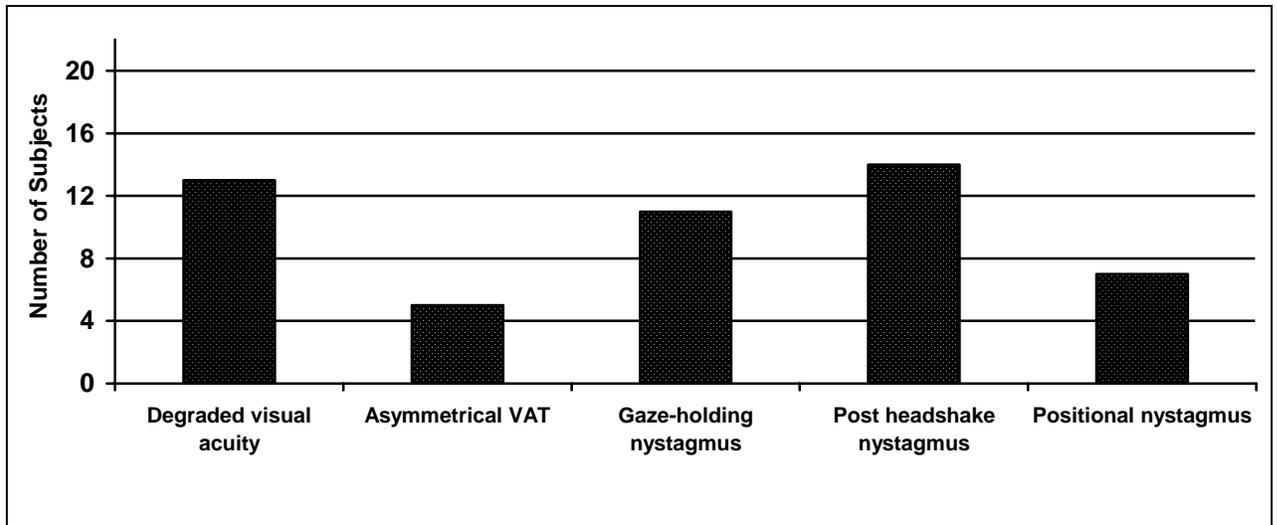


Figure 1. Number of subjects included in the study with specific test findings are shown.

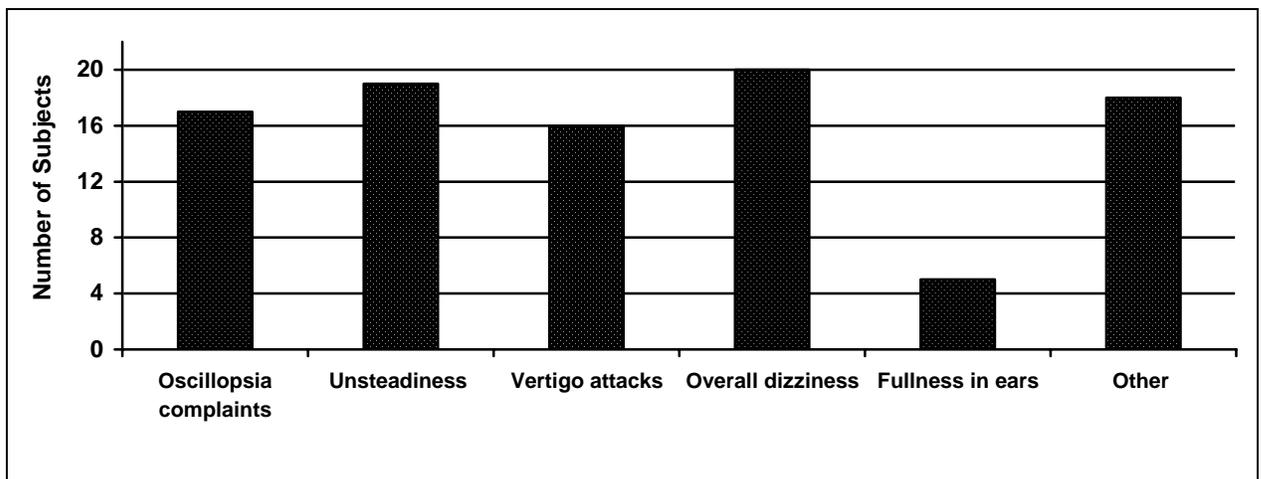


Figure 2. Number of subjects included in the study with particular reported symptoms are shown.

Using these criteria, 21 females and 19 males ranging in age from 21 – 87 years (mean = 62.0; standard deviation = 16.0) were selected for participation in the study. Subjects were later divided into two groups based on involved side. The right caloric weakness group consisted of 13 females and 10 males, aged 21 to 87 years (mean = 59.9; standard deviation = 18.1). The left caloric weakness group consisted of 8 females and 9

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males, aged 43 to 82 years (mean = 64.8; standard deviation = 12.2). Average unilateral weakness was 49.11% and 50.93% for the right and left weakness groups, respectively.

Finally, a control group of 10 subjects with no reported hearing or vestibular problems was recruited. All subjects passed a hearing screening of ≤ 25 dB HL at the octave frequencies from 250 to 8000 Hz. These subjects had no unilateral weakness and no OKN abnormalities. The subjects included 7 females and 3 males aged 21 to 55 (mean = 30.9; standard deviation = 10.4). Their OKN and caloric test data was compared to that of the groups with unilateral caloric weakness.

Procedures

Participants with unilateral caloric weakness received a complete audiological and vestibular assessment. Results from this assessment were used to determine eligibility to participate. Objective test findings, including caloric and OKN test results, and subjective symptoms were recorded from each chart.

Optokinetic Nystagmus and Unilateral Weakness

During the examination of chart records, values were recorded for OKN testing and caloric testing. For OKN, left and right eye responses were documented for a slow ($15^\circ/s$), medium ($30^\circ/s$), and fast ($45^\circ/s$) stimulus velocity. Optokinetic stimuli were presented to the subjects using a projector that filled 90% of their visual field.

Nystagmus slow phase eye velocity and gain was recorded for each eye and stimulus velocity. For caloric testing, left and right ear results were recorded for both warm and cool responses obtained with an air caloric irrigator. A unilateral weakness was determined if there was a twenty percent or greater interaural difference of the mean maximum slow-phase velocity.

RESULTS

First, the data of all subjects with unilateral caloric weakness was considered together. Correlations were determined for all factors including caloric response (right and left ear), and optokinetic results (gain and slow phase velocity) for the right and left eyes for the three stimulus conditions. Following this analysis, the subjects were divided into two groups based on the involved side, right side weakness or left side weakness. Data were averaged within each group and examined for trends. Statistical analysis was subsequently performed on an interesting pattern of results that emerged and this pattern was compared to the results of a control group.

Initially, data were analyzed to determine any relationship between unilateral weakness (right ear or left ear) and optokinetic nystagmus (slow phase eye velocity and gain for each eye at all stimulus conditions). None of the correlations were significant at the $\alpha < 0.05$ probability level.

Next, the data of the twenty-three subjects with right caloric weakness were compared to the data of the seventeen subjects with left caloric weakness. A potential trend emerged during inspection of the results obtained for the slow stimulus velocity ($15^\circ/\text{s}$). Subjects with a right caloric weakness showed decreased OKN gain for the right eye with a right-moving stimulus compared to the subjects with a left caloric weakness. Alternatively, subjects with a left caloric weakness showed decreased OKN gain compared to the subjects with a right caloric weakness for the left eye with a left-moving stimulus. No other trends were identified. This pattern was not evident in the data of the control group although it was observed that the control group had gains much closer to 1.0 than the other two groups for both of these conditions. This is shown in Figure 3.

These data were statistically analyzed using two separate one-way Analyses of Variance (ANOVAs) to determine any differences between the groups. An ANOVA on the data obtained for the left eye and 15°/s left moving stimulus indicated a significant effect of group [$F(2,47) = 12.39, p < 0.001$]. Post-hoc analysis (Tukey test) revealed that the control group had significantly higher ($p < 0.001$) gain than either of the other two groups and the other groups were not different in terms of gain ($p > 0.05$). A separate ANOVA on the data obtained for the right eye and 15°/s right moving stimulus also indicated a significant effect of group [$F(2,47) = 14.28, p < 0.001$]. Post-hoc analysis (Tukey test) revealed that the control group had significantly higher ($p < 0.01$) gain than either of the other two groups and the other groups were not different in terms of gain ($p > 0.05$).

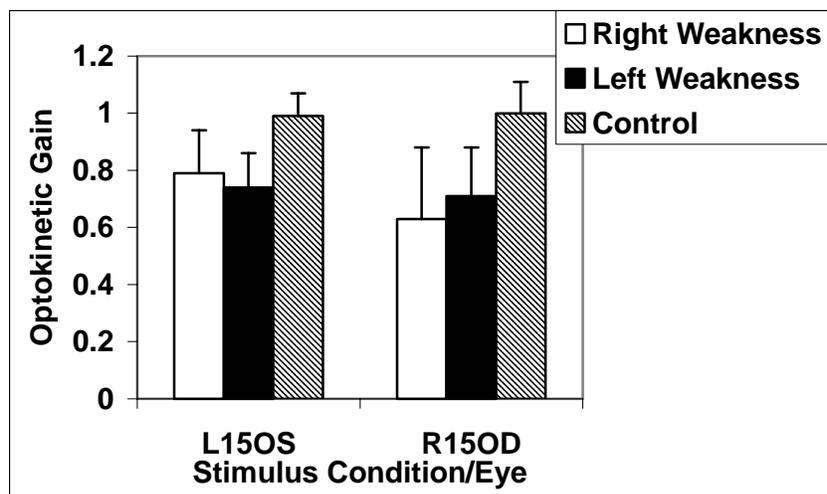


Figure 3. Optokinetic gain is shown for the groups with right unilateral weakness, left unilateral weakness, and the control group. Results are shown for the left and right eyes for the left and right moving stimuli. Error bars represent standard deviation.

DISCUSSION

The purpose of this paper was to determine whether a relationship exists between unilateral caloric weakness and optokinetic nystagmus findings. It was expected that we

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would observe OKN abnormalities in subjects with a caloric weakness because of the potential mismatch of sensory information between the vestibular and visual systems that converge at the level of the vestibular nucleus.

Results indicated no correlation between caloric response (right and left ear) and optokinetic results (gain and slow phase velocity) for any stimulus velocity. One reason for a lack of correlation may be due to the method in which optokinetic nystagmus was elicited. The subjects were seated in a chair approximately 6 feet from a wall upon which the optokinetic stimuli were projected, as is typical of such testing. Since the subject was seated, there was no actual motion or acceleration for the vestibular system to detect. Therefore, the only signals sent to the vestibular nucleus from the peripheral labyrinths would be that of the tonic, resting firing rate of the semicircular canals. Although an uncompensated vestibulopathy would be expected to produce a decrease in the tonic firing rate on the lesioned side and an increase in the tonic firing rate on the healthy side, this change in the resting firing rate may not have created enough of a mismatch in the VN to cause an asymmetrical OKN. Perhaps a relationship may emerge if the vestibular systems were activated with movement either before or during the OKN stimulation. This is possible with rotary chair testing.

Another possibility is to measure optokinetic after-nystagmus (OKAN). Optokinetic after-nystagmus is observed in the dark with eyes open after removing the OKN stimuli (Leigh & Zee, 1999). Neurons in the VN continue to discharge for several seconds after OKN stimuli have been removed, thus leading to the phenomenon of OKAN (Waespe & Henn, 1977). OKAN lasts for approximately thirty seconds (Hain, 1997). Patients with unilateral peripheral vestibular lesions typically exhibit abnormal

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OKAN, specifically, asymmetric OKAN in the direction the spontaneous nystagmus is observed (Baloh & Honrubia, 2001). Although OKAN velocity is more variable than OKN velocity in young, normal subjects, observing OKAN offers the potential for assessment of activity occurring in the VN in the absence of head movement (Leigh & Zee, 1999).

Takemori (1997) studied whether compensation could be evaluated by observing OKAN in individuals undergoing a unilateral labyrinthectomy or vestibular neurectomy. Takemori measured the duration of OKAN before and after surgery. The responses of OKAN were heavily affected by the direction of spontaneous nystagmus. He reported that OKAN to the ipsilateral side was abolished soon after the procedure. With time, however, OKAN to the ipsilateral side recovered. Therefore, Takemori concluded that the stage of compensation could be assessed by measuring OKAN. Since OKAN appears to be a promising method of assessing compensation, perhaps future studies should focus on more precise methods of obtaining and quantifying OKAN in order to reduce the variability that is currently seen among subjects.

Although no relationship was observed between the caloric findings and the OKN results, a trend was observed when the data of the subjects with a right unilateral weakness was compared to that of the subjects with a left unilateral weakness. The group with a right caloric weakness showed decreased OKN gain in the right eye for the 15°/s right moving stimulus. Subjects with a left unilateral weakness showed decreased OKN gain in the left eye for the 15°/s left moving stimulus. The data of the control group (with no unilateral weakness) did not reveal such a trend for stimuli moving in either direction. Although the control group had higher gain than either of the groups with a weakness,

there was no significant difference between the groups with left and right caloric weakness.

It has been reported that disconjugate nystagmus is produced whenever a lesion exists from the VN to the oculomotor neuron pathways. In fact, a lesion in the medial longitudinal fasciculus (MLF) will produce impaired slow and fast components of nystagmus controlled by the ipsilateral medial rectus muscle (Baloh & Honrubia, 2001). The slow and fast components produced by the contralateral lateral rectus muscle remain normal. Therefore, it is possible that a unilateral weakness may lead to a deficit with the extraocular muscles similar to that of a lesion in the MLF in the VN on the side of the weakness. This may explain the decreased gain that was observed in this study. It is possible that the subjects in the current investigation may have been at a level of vestibular compensation that caused a trend for OKN abnormalities in the data, but was not strong enough to create group differences. Incorporating patients with acute vestibular lesions may allow investigators to explore this possibility to a greater extent.

It remains difficult to explain why slow-phase eye velocity was not affected and why only the 15°/s stimulus velocity indicated a trend. Although it is difficult to explain why slow-phase velocity was not affected, it is known that OKN gain reduces as stimulus frequency (or speed) increases (Holzman et al., 1975). Also, it is known that older subjects have greater difficulty tracking the OKN stimulus at higher speeds (Calder, 2000). Given that the current groups of subjects with unilateral weakness were older, it is possible that their performance deteriorated at higher speeds to an extent that any group differences were not apparent. This would also explain the higher OKN gains observed for the control group that was younger than the other two groups. Using stimulus

velocities even lower than 15°/s could possibly differentiate these two groups with unilateral weakness further. The potential significance of this is that the asymmetry in gain may be useful in tracking compensation of patients over time.

SUMMARY AND CONCLUSIONS

The purpose of this investigation was to determine any relationship between unilateral caloric weakness and optokinetic nystagmus. The data of forty subjects with a unilateral weakness was analyzed together and then for groups of participants with a right unilateral weakness and a left unilateral weakness. Group results were compared with data collected from 10 participants with normal hearing and vestibular function. Specific conclusions were: 1) no correlation exists between unilateral weakness and measures of optokinetic nystagmus and 2) a trend exists showing reduced OKN gain in the ipsilateral eye of the side of the weakness (for both left and right sides) for slow moving stimuli in the direction of the weakness. Interpretation of OKN along with caloric results may offer potential for identification and tracking of compensation after a unilateral loss of vestibular function, but further research is needed.

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