Demêmes, D., Lleixa, A., Dechesne, C.J., 1995. Cellular and subcellular localization of AMPA-selective glutamate receptors in the mammalian peripheral vestibular system. Brain Res. 671, 83–94.

Devau, G., Lehouelleur, J., Sans, A., 1993. Glutamate receptors on type I vestibular hair cells of guinea-pig. Eur. J. Neurosci. 5, 1210–1217.

Fetoni, A.R., Sergi, B., Scarano, E., Paludetti, G., Ferraresi, A., Troiani, D., 2003. Protective effects of alpha-tocopherol against gentamicin-induced oto-vestibulo toxicity: an experimental study. Acta Otolaryngol. 123, 192–197.

Fetoni, A.R., Sergi, B., Ferraresi, A., Paludetti, G., Troiani, D., 2004. Protective effects of alpha-tocopherol and tiopronin against cisplatin-induced ototoxicity. Acta Otolaryngol. 124, 421–426.

Gilgun-Sherki, Y., Rosenbaum, Z., Melamed, E., Offen, D., 2002. Antioxidant therapy in acute central nervous system injury: current state. Pharmacol. Rev. 54, 271–284.

Hoffer, M.E., Kopke, R.D., Weisskopf, P., Gottshall, K., Allen, K., Wester, D., 2001. Microdose gentamicin administration via the round window microcatheter: results in patients with Meniere's disease. Ann. N. Y. Acad. Sci. 942, 46–51.

Horiike, O., Shimogori, H., Ikeda, T., Yamashita, H., 2003. Protective effect of edaravone against streptomycin-induced vestibulotoxicity in the guinea pig. Eur. J. Pharmacol. 464, 75–78.

Horiike, O., Shimogori, H., Yamashita, H., 2004. Effect of edaravone on streptomycin-induced vestibulotoxicity in the guinea pig. Laryngoscope 114, 1630–1632.

Itakura, A., Kurauchi, O., Takashima, S., Uchida, K., Ito, M., Mizutani, S., 2002. Immunological detection of 4-hydroxynonenal protein adducts in developing pontine and Purkinje neurons and in karyorrhexis in pontosubicular neuronal necrosis. Early Hum. Dev. 67, 19–28.

Kopke, R., Allen, K.A., Henderson, D., Hoffer, M., Frenz, D., Van de Water, T., 1999. A radical demise. Toxins and trauma share common pathways in hair cell death. Ann. N. Y. Acad. Sci. 884, 171–191.

Lees, K.R., Asplund, K., Carolei, A., Davis, S.M., Diener, H.C., Kaste, M., Orgogozo, J.M., Whitehead, J., 2000. Glycine antagonist (gavestinel) in neuroprotection (GAIN International) in patients with acute stroke: a randomised controlled trial. GAIN International Investigators. Lancet 355, 1949–1954.

Lipton, S.A., Rosenberg, P.A., 1994. Excitatory amino acids as a final common pathway for neurologic disorders. N. Engl. J. Med. 330, 613–622.

Matsubara, A., Takumi, Y., Nakagawa, T., Usami, S., Shinkawa, H., Ottersen, O.P., 1999. Immunoelectron microscopy of AMPA receptor subunits reveals three types of putative glutamatergic synapse in the rat vestibular end organs. Brain Res. 819, 58–64.

Puel, J.L., Saffiedine, S., Gervais d'Aldin, C., Eybalin, M., Pujol, R., 1995. Synaptic regeneration and functional recovery after excitotoxic injury in the guinea pig cochlea. C. R. Acad. Sci. III 318, 67–75.

Puel, J.L., d'Aldin, C., Ruel, J., Ladrech, S., Pujol, R., 1997. Synaptic repair mechanisms responsible for functional recovery in various cochlear pathologies. Acta Otolaryngol. 117, 214–218.

Shimogori, H., Yamashita, H., 2004. Peripheral vestibular disorder induced by (±)-alpha-amino-3-hydroxy-5-methyl-isoxazole-4-propionic acid (AMPA). Neurosci. Lett. 371, 69–72.

Shimogori, H., Yamashita, H., Watanabe, T., Nakamura, S., 1999. A role of glucocorticoid receptors in the guinea pig vestibular system. Brain Res. 851, 258–260.

Silverstein, H., 1999. Use of a new device, the MicroWick™, to deliver medication to the inner ear. Ear Nose Throat J. 78, 595–598.

Smith, P.F., Darlington, C.L., 1994. Pharmacology of the vestibular system. In: Baloh, R.W. (Ed.), Bailliere's Clinical Neurology. Bailliere Tindall, London, pp. 467–484.

Sugahara, K., Shimogori, H., Yamashita, H., 2001. The role of acidic fibroblast growth factor in recovery of acoustic trauma. NeuroReport 12, 3299–3302.

Tabuchi, K., Tsuji, S., Ito, Z., Hara, A., Kusakari, J., 2001. Does xanthine oxidase contribute to the hydroxyl radical generation in ischemia and reperfusion of the cochlea? Hear. Res. 153, 1–6.

Takemoto, T., Sugahara, K., Okuda, T., Shimogori, H., Yamashita, H., 2004. The clinical free radical scavenger, edaravone, protects cochlear hair cells from acoustic trauma. Eur. J. Pharmacol. 487, 113–116.

Takumida, M., Anniko, M., 2000. Glutamate-induced production of nitric oxide in guinea pig vestibular sensory cells. Acta Otolaryngol. 120, 466–472.

Tanaka, K., Takemoto, T., Sugahara, K., Okuda, T., Mikuriya, T., Takeno, K., Hashimoto, M., Shimogori, H., Yamashita, H., 2005. Post-exposure administration of edaravone attenuates noise-induced hearing loss. Eur. J. Pharmacol. 522, 116–121.

Vergun, O., Sobolevsky, A.I., Yelshansky, M.V., Keelan, J., Khodorov, B.I., Duchen, M.R., 2001. Exploration of the role of reactive oxygen species in glutamate neurotoxicity in rat hippocampal neurones in culture. J. Physiol. 531, 147–163.

Watanabe, T., Morita, I., Nishi, H., Murota, S., 1988. Preventive effect of MCI-186 on 15-HPETE induced vascular endothelial cell injury in vitro. Prostaglandins Leukot. Essent. Fat. Acids 33, 81–87.

Yamamoto, T., Yuki, S., Watanabe, T., Mitsuka, M., Saito, K.I., Kogure, K., 1997. Delayed neuronal death prevented by inhibition of increased hydroxyl radical formation in a transient cerebral ischemia. Brain Res. 762, 240–242.

Yoritaka, A., Hattori, N., Uchida, K., Tanaka, M., Stadtman, E.R., Mizuno, Y., 1996. Immunohistochemical detection of 4-hydroxynonenal protein adducts in Parkinson disease. Proc. Natl. Acad. Sci. U. S. A. 93, 2696–2701.

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Epidemiologic Characteristics of Definite Ménière's Disease in Japan

A Long-Term Survey of Toyama and Niigata Prefectures

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Key Words
Ménière's disease, epidemiology

Abstract
To identify epidemiologic characteristics of definite cases of Ménière's disease (DMD), we conducted retrospective surveys of the period 1990–2004 of the Nishikubiki district and of the period 1980–2004 of Toyama Medical and Pharmaceutical University. Three hundred and seventy-five patients (50 from Nishikubiki, 325 from Toyama) were diagnosed with DMD according to the diagnostic criteria proposed by the Japanese Society for Equilibrium Research. There was a slight increase in the prevalence of DMD during the period 1990–2004. However, incidence did not change significantly over time. The average annual prevalence and incidence were 34.5 and 5.0, respectively, per 100,000 population. Incidence and prevalence predominated in females. With respect to age at disease, the incidence in elderly patients was increased when we corrected for age distribution in the overall population.

Introduction

In 1974, the Ménière's Disease Research Committee of Japan was founded by the Ministry of Health and Welfare of Japan. Since then, several epidemiologic studies of the incidence and prevalence of Ménière's disease (MD) in Japan have been reported. In 1980, Nakae et al. [1] reported the prevalence of MD based on 1-day and 1-week surveys countrywide. The former was a random sampling survey of 811 hospitals and 729 clinics, and the latter was a survey of all university hospitals (69 hospitals) and general hospitals (121 hospitals) in Japan. In both surveys, diagnosis of MD was made by ENT doctors and doctors of internal medicine, and the prevalence of MD was 73 (1-day survey) and 3.5 (1-week survey) per 100,000 population. Later surveys of more limited regions such as prefectures, cities, and towns were conducted. In 1982, Tokumasu et al. [2] reported annual incidence and prevalence for Sagamihara city based on records of the Neuro-Otologic Department in Kitasato University Hospital for the period August 1971 through July 1978. Incidence and prevalence were 2.8 and 16.7, respectively, per 100,000 population. In 1997, Shojaku and Watanbe [3] reported a 1-year survey of ENT departments of all hospitals and clinics in the Hida district (1 city, 4 towns, and 10 villages) and the Nishikubiki district

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Table 1. Diagnostic criteria for DMD

Subjective symptoms (clinical history)

1 Repeated attacks of whirling vertigo
2 Fluctuating cochlear symptoms synchronized with vertiginous attack
3 No nervous symptoms except VIIIth nerve involvement
4 No histories that cause inner ear dysfunction such as otitis media, head injury, etc.

Objective signs (examination)

5 Audiometrically documented characteristic hearing loss such as low-tone hearing loss, fluctuating hearing loss, etc.
6 Labyrinthine dysfunction shown using by the equilibrium test
7 Exclusion of central nervous system involvement except VIIIth nerve based on neurological examination
8 No clear evidence of cause of inner ear dysfunction based on neuro-otologic, physical, laboratory and radiologic examinations

Conformation to conditions 1–8 results in a definite diagnosis.

Table 2. Corrected percentages of elderly onset cases of DMD in Nishikubiki district

	1990–1994	1995–1999	2000–2004
Total population	56,626	54,969	52,577
≥ 65 years	10,912	12,762	14,402
≥ 65 years, %	19.3	23.2	27.4
Total DMD cases	18	9	11
≥ 65 years	2	4	7
≥ 65 years, %	11.1	44.4	64.0
Corrected DMD cases ≥ 65 years, %	11.1	36.9	44.7

Table 3. Average annual incidence and prevalence of DMD in Nishikubiki district

	1990–1994	1995–1999	2000–2004
Population	56,626	54,969	5,577
DMD cases	15.6	20.8	20.0
Prevalence (per 100,000 population)	27.6	37.8	38.0
DMD de novo	3.6	2.4	2.2
Incidence (per 100,000 population)	6.4	4.3	4.2

(1 city and 2 towns). Fifty-three (Hida) and 12 (Nishikubiki) cases of MD were documented in populations of 145,000 and 67,000, respectively, yielding a prevalence of 36.6 and 21.4, respectively, per 100,000 population. In a prefecture-based survey, prevalence was 16.5–18.4 per 100,000 population [4, 5].

In Japan, a rapid increase in cases of MD occurred after World War II [6]. However, prevalence varied in several epidemiologic surveys conducted by the above-mentioned Research Committee. The reasons for such discrepancies remain to be elucidated; there have been few reports of alterations in epidemiologic features over extended periods of time. In this study, changes in incidence and prevalence of MD over time were investigated in a 15-year retrospective survey of the Nishikubiki district, the westernmost region of Niigata prefecture. Other epidemiologic features, including sex ratio and age at onset were also evaluated in the Nishikubiki district and at the University Hospital of Toyama Medical and Pharmaceutical University (TMPU). Some of the data in this study were analyzed in a previous study [3].

Materials and Methods

Diagnostic Criteria

In 1976, the Ménière's Disease Research Committee of Japan proposed diagnostic criteria for MD [6]. In 1988, the Japanese Society for Equilibrium Research (JSER) proposed modified diagnostic criteria comprising not only subjective symptoms but also objective signs [7]. On the basis of the new criteria (table 1), we surveyed definite cases of MD (DMD) in the Nishikubiki district from January 1990 to December 2004 (15 years) and at TMPU from January 1980 to December 2004 (25 years). Diagnosis of DMD was made by 2 neuro-otologic specialists (Y.W. and K.M.).

Characteristics of Nishikubiki District and Toyama Prefecture

Nishikubiki district (Itoigawa city, Nou town and Oumi town) is located in the westernmost region of Niigata Prefecture and has a population of approximately 53,000. This district (746.25 km^2) is bordered by high mountains and the Sea of Japan, and the chief industry is cement manufacturing. The elderly segment of the population is expanding most rapidly. According to the 2000 census, the percentage of elderly individuals was 27.4% (table 2), which is almost 10% greater than that of Japan as a whole. During the years surveyed, there were two hospitals with an ENT department (Itoigawa General Hospital and Himekawa Hospital), and no ENT clinic. Due to geographic conditions, it is likely that the majority of patients suffering from ENT disorders in this district sought treatment at these hospitals [3, 8].

Toyama Prefecture (4,247 km^2) is located along the coast of the Sea of Japan and adjoins Nishikubiki district. The total population of Toyama Prefecture is approximately 1,120,000, and the chief

Table 4. Corrected percentages of elderly-onset cases of DMD in TMPU

	1980–1984	1985–1989	1990–1994	1995–1999	2000–2004
Total population	1,103,459	1,133,616	1,120,161	1,123,125	1,120,800
≥ 65 years	123,407	143,646	168,946	201,320	241,900
≥ 65 years, %	11.2	12.7	15.1	17.9	21.6
Total DMD cases	59	65	81	64	56
Onset ≥ 65 years	1	2	5	4	7
Onset ≥ 65 years, %	3.4	3.1	6.2	6.3	12.5
Corrected DMD cases					
Onset ≥ 65 years, %	3.4	2.7	4.6	3.9	6.5

industry is aluminum manufacturing. According to the 2000 census, the percentage of elderly individuals was 21.6% (table 4). The University Hospital of TMPU is the treatment center for patients with neuro-otologic disease in Toyama Prefecture.

Data Analysis

Survey data were stored and analyzed by database software at the Department of Otolaryngology at TMPU. To investigate changes in DMD incidence and prevalence and age of onset over time, data from 5-year periods (1980–1984, 1985–1989, 1990–1994, 1995–1999, 2000–2004) were compared. Incidence and prevalence were estimated from the data of the Nishikubiki district. Sex ratio and age at onset were analyzed from both the Nishikubiki and TMPU surveys. Data from the national censuses for 1980, 1985, 1990, 1995, and 2000 were used to correct the data for DMD cases.

For statistical analysis, χ^2 tests were performed on a personal computer with StatView for Windows (version 4.5, Abacus Concepts, Berkeley, Calif., USA).

In this study, the terms incidence and prevalence are as described by Phillips [9]. Incidence is the number of new cases diagnosed during a set time period, whereas prevalence is the total number of active cases (i.e., new cases plus existing cases).

Results

In the Nishikubiki district survey, 50 DMD cases were diagnosed at the ENT departments of Itoigawa General Hospital and Himekawa Hospital. Sixteen males (32%) and 34 females (68%) were diagnosed with DMD. The percentage of males among the total population was 48.2% (average male population 26,489, average total population 54,724). Thus, female patients were significantly predominant in the Nishikubiki district ($p < 0.05$).

Averaged annual incidence and prevalence for each of the 5-year periods in Nishikubiki district (1990–1994, 1995–1999, and 2000–2004) are shown in table 3. There was a slight increase in prevalence over time. In contrast, incidence did not change over time. Average annual incidence and prevalence between 1990 and 2004 were 5.0 and 34.5, respectively, per 100,000 population.

The age at onset peaked in the fifth and sixth decades for males and in the sixth and seventh decades for females. There were no DMD cases in the first 2 decades of life for either sex. The proportion of DMD cases with age at onset ≥ 65 years increased progressively for each of the 5-year periods ($p < 0.05$) (table 2).

In the TMPU survey, 13,059 patients were examined at the neuro-otologic section of the ENT department of TMPU Hospital, and 325 (2.5%) were diagnosed with DMD. One hundred and forty-seven (42.5%) were males and 178 (57.5%) were females. Age at onset peaked in the fifth decade for males and in the fourth decade for females. There were 5 patients (1.5%) with age at onset ≤ 14 years. The proportion of DMD patients with age at onset ≥ 65 years showed a slight increase over time (table 4). The percentage of DMD cases in the Nishikubiki and TMPU surveys during the period 2000 through 2004 was 4.18 (2.2/52,577) and 1.27 (56/1,120,800) per 100,000 population, respectively. There was no significant difference between the two surveys.

Discussion

In this study, changes in the incidence and prevalence, sex ratio, and age at onset of DMD over time were evaluated in Nishikubiki district and Toyama Prefecture. There was a slight increase in the prevalence of DMD during the period 1990 through 2004. However, incidence did not change over time. Average annual incidence and prevalence were 5.0 and 34.5, respectively, per 100,000 population. Incidence and prevalence in the female population predominated over those in the male population. With respect to age at onset, onset at ≥65 years of age increased when we corrected for differences in age distribution.

The prevalence of MD has been reported by researchers in several countries. In England, Cawthorne and Hewlett [10] reported 43 cases among 27,365 patients at 8 general practices in 1 year and estimated the frequency as 157 cases per 100,000 patients. A 1-year population-based survey in the Uppsala and Skane regions of Sweden by Stahle et al. [11] reported a prevalence of 46 per 100,000 population. In the United States, Wladislavosky-Waserman et al. [12] performed a 1-day survey of 40,000 Caucasian inhabitants of Rochester, Minnesota, and reported a prevalence of 218 per 100,000 population. In Italy, the annual prevalence in the district of Tuscany in 1992 was 3.3 per 100,000 population [13]. A Finnish study based on a 5-year survey of 2 university hospitals and 5 general hospitals reported a prevalence of 43.2 per 100,000 population.

Variations in diagnostic criteria may be one of the reasons for the wide variation in prevalence among population-based surveys. Wladislavosky-Waserman [12] used the 1972 AAOO criteria, in which not only classic MD but also vestibular MD and cochlear MD are included. Only 13 of 180 cases (7.2%) were initially classified as classic MD, but 117 cases (65%) were later reclassified as classic MD. Classic MD resembles definite MD according to the 1995 AAO-HNS criteria (used by Kotimaki et al. [14]) and DMD in the JSER criteria used in this study. Classic MD is also similar to the criteria for MD in Swedish [11] and Italian studies [13]. The prevalence in our study was almost equivalent to that in the Swedish and Finnish studies and between that of the American and Italian studies.

Incidence also varies among population-based surveys. A 30-year survey at the Mayo Clinic showed an incidence of 15.3 per 100,000 population [12]. In Europe, a relatively lower incidence has been reported. In Italy, Celestino and Ralli [15] reported a 13-year survey during the period 1973 through 1985, in which the incidence was 8.2 per 100,000 population. Another Italian survey reported an incidence of 1.2 per 100,000 population [13]. In Finland, the incidence was 4.3 per 100,000 population [14]. The incidence in Japanese studies (Tokumasu et al. [2]: 2.8 per 100,000 population; our study 5.0 per 100,000 population) was almost equivalent to that in European studies.

With respect to sex ratio, Wladislavosky-Waserman et al. [12] reported a slight but not significant predominance in females. Celestino and Ralli [15] presented data on 52 (46.8%) males and 59 (53.2%) females. However, the majority (52.1%) of the inhabitants in the southeastern Latium region of Italy were females. Therefore, the incidence of MD was found to be similar between sexes. Stahle et al. [11] also reported a predominance of cases of MD in females (60%). A similar tendency has been reported in Japanese studies [4, 5] including the present study. Thus, the sex ratio varied among surveys.

The incidence of MD in elderly individuals has been reported to be between 10.8 and 37.8% of the total number of MD cases [4, 11, 12, 14]. In cases of MD in elderly patients (≥65 years of age), the incidence of reactivated, longstanding MD was less than that of de novo MD [16]. In addition, cases of elderly-onset MD have recently increased. The larger population of working elderly persons in recent years may explain this phenomenon. According to the 2000 census, 22% of elderly people in Japan were working. Ikeda and Watanabe [17] reported that 4 of 11 patients with MD of elderly onset ≥70 years of age held managerial jobs, suggesting that job-related mental and physiologic fatigue may be involved in the development of MD. Care of a sick spouse or parent by elderly individuals [17] may also be involved in the recent increase of elderly-onset cases. Elderly individuals are also healthier than those of previous generations.

Today there are more than 150 million septuagenarians on earth, and this number is likely to double over the next 30 years. We therefore need to be aware of potential for elderly-onset MD, which may induce falls resulting in serious injuries or life-threatening fractures.

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References

1 Nakae K, Nitta H, Hattori Y, Maeda K, Komatsuzaki A, Mizukoshi K, Watanabe I: The prevalence of Meniere's disease in Japan (in Japanese). Prac Otol (Kyoto) 1980;73(suppl 2):1023–1029.
2 Tokumasu K, Tashiro N, Goto K, Saito A, Kawano R, Hirose Y, Yoneda S: Incidence and prevalence of Meniere's disease in Sagamihara city, Kanagawa-ken (in Japanese). Prac Otol (Kyoto) 1982;75(suppl 3):1165–1173.
3 Shojaku H, Watanabe Y: The prevalence of definite cases of Meniere's disease in the Hida and Nishikubiki districts of central Japan: survey of relatively isolated areas of medical care. Acta Otolaryngol Suppl 1997;528:94–96.
4 Mizukoshi K, Watanabe Y, Shojaku H, Ito M, Naruse Y, Kagamimori S: Epidemiological studies on Meniere's disease in Toyama, Japan; in Filipo R, Barbara M (eds): Meniere's Disease – Perspectives in the '90s. New York, Kugler, 1994, pp 21–25.
5 Okamoto M, Morio S, Takita S, Nakayama H: Estimation of the number of Meniere's disease patients in Japan based on the 'patient survey' of a local self-governing body sent to all medical care institutions (in Japanese). Equilibrium Res 1993;9(suppl):1–4.
6 Watanabe I: Meniere's disease with special emphasis on epidemiology, diagnosis and prognosis. ORL 1980;42:20–45.
7 The Committee for Standardization of Diagnostic Criteria in Vertiginous Diseases: Meniere's disease: the guidelines for standardization of diagnostic criteria in vertiginous diseases, 1987 (in Japanese). Equilibrium Res 1988;47:247–249.
8 Shojaku H, Watanabe Y, Akaogi K, Ueda U, Mizukami Y, Yasumura S, Nagasaki M: Prevalence of definite cases of Meniere's disease in Niigata prefecture (in Japanese). Equilibrium Res 1998;57:487–490.
9 Phillips DS: Basic Statistics for Health Science Students. New York, Freeman, 1978.
10 Cawthorne T, Hewlett AB: Meniere's disease. Proc R Soc Med 1954;47:663–670.
11 Stahle J, Stahle C, Arenberg IK: Incidence of Meniere's disease. Arch Otolaryngol 1978;104:99–102.
12 Wladislavosky-Waserman P, Facer GW, Mokri B, Kurland LT: Meniere's disease: a 30-year epidemiologic and clinical study in Rochester, MN, 1951–1980. Laryngoscope 1984;94:1098–1102.
13 Nuti D, Biagini C, Passali D: Incidence and prevalence of Meniere's disease in Tuscany; in Filipo R, Barbara M (eds): Meniere's Disease – Perspectives in the '90s. New York, Kugler, 1994, pp 20–23.
14 Kotimaki J, Sorri M, Aantaa E, Nuutinen J: Prevalence of Meniere's disease in Finland. Laryngoscope 1990;109:748–753.
15 Celestino D, Ralli G: Incidence of Meniere's disease in Italy. Am J Otol 1991;12:135–138.
16 Ballester M, Liard P, Vibert D, Hausler R: Meniere's disease in the elderly. Otol Neurotol 2002;23:73–78.
17 Ikeda M, Watanabe I: Meniere's disease in the aged (in Japanese). Nippon Jibiinkoka Gakkai Kaiho 1995;98:380–390.

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ORIGINAL ARTICLE

Influence of visually induced self-motion on postural stability

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Abstract
Conclusion. Our results indicate that the illusion of self-motion is a significant factor leading to spatial disorientation. *Objective*. Under normal circumstances, self-motion is perceived in response to motion of the head and body. However, under certain conditions, such as virtual reality environments, visually induced self-motion can be perceived even though the subject is not actually moving, a phenomenon known as "vection". The aim of this study was to examine the possible influence of illusory self-rotation (circular vection) on postural adjustments. *Material and methods*. The subjects were 10 young females with no history of ocular or vestibular disease. Video-motion analysis was applied to measure postural movements during vertical optokinetic stimulation. *Results*. For most subjects, movement of the visual surroundings induced head and body displacements in the same direction as that of the visual stimulus, regardless of the onset of self-motion perception. However, there was a significant increase in postural instability after the subjects began to perceive false self-motion in the opposite direction to that of the visual stimulus.

Keywords: *Optokinetic stimulation, postural adjustment, spatial disorientation, vection, virtual reality*

Introduction

Self-motion is perceived by means of information from visual, vestibular and somatosensory systems, in response to head and body motion. Of these systems, visual information contributes largely to self-motion perception during motion at a constant velocity, as the vestibular system receives acceleration signals exclusively. Under certain circumstances, however, visually induced self-motion is known to be falsely perceived; in other words, the subject is not actually moving. This illusory perception, known as "vection", can be well illustrated by the train illusion, i.e. a passenger on a stationary train experiences a sensation of motion when a train alongside is moving, and more recently, by virtual reality systems. Vection can be induced in a sitting position in which head and body movements are stabilized using a chin cup and forehead rest [1]. When an erect subject is exposed to visual surround motion, body displacement coupled with vection is induced in the direction of the visual motion [2–7]. Under these conditions, the vection response is complicated by the fact that information from the vestibular and somatosensory systems, which signal that the head and body have not really moved, remains in conflict with information from the visual system and the resultant illusion of self-motion perception for as long as the visual stimulus endures. The first report of the correlation between vection and postural change was that of Dichgans and Brandt [4], but others [3] have shown that the onset latencies of postural change and vection are not identical. Later, Previc and Mullen [8] showed that the onset of roll vection and postural instability correlated strongly with one another by dividing changes in the center of foot pressure into two different components. Nevertheless, the influence of vection on postural instability was somewhat confusing: the onset of roll vection was delayed by several seconds with respect to the postural instability.

Vertical visual motion produces a strong self-rotation. For example, upward stimulation produces a sensation of a tumbling forward self-motion coupled with a sensation of a head and body tilt, and a real head and body tilt [9–12]. This study was designed to investigate the question of whether or not an illusory self-motion perception in the opposite direction to that of the visual motion could tip the balance in favor of maintenance of spatial orientation. Video-motion

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analysis was applied to measure head and body displacements during vertical visual stimulation.

Material and methods

The subjects were adult volunteers with no history of ocular or vestibular disease. We recorded data from 10 young female subjects whose height ranged from 145 to 155 cm (mean ± SD 152.7 ± 2.5 cm) and whose age ranged from 21 to 24 years (mean ± SD 22.0 ± 1.1 years). Subjects stood in Romberg's posture and faced a hemispherical dome with a radius 75 cm, with the center of the inside surface approximately at eye level a distance of 75 cm away (Figure 1). A random pattern of dots with diameters of 0.2–1.0° of visual angle was projected onto the inner wall of the hemispherical dome. The pattern subtended 90° in the horizontal and 180° in the vertical direction. The pattern was rotated about the subject's inter-aural axis (upward or downward) at a constant acceleration of $1°/s^2$ for 60 s in darkness. The subjects were instructed to stare straight ahead at the stimulus pattern without attempting to follow the moving dots while standing. Subjects were also instructed to signal the onset of self-rotation by pressing a hand switch.

Video-motion analysis was applied to measure postural movements during vertical optokinetic stimulation (OKS). For the detection of postural movements in the dark, small red fluorescent markers were placed on the right side of the head (parietal and temporal parts) and body (neck, shoulder, greater trochanter, knee and ankle). The motion of each marker was recorded using a charge-coupled device camera, and videotaped from the side at 60 fields (30 frames) per second. The video was digitized and sent to a PC for subsequent analysis (DKH; Frame-DIAS).

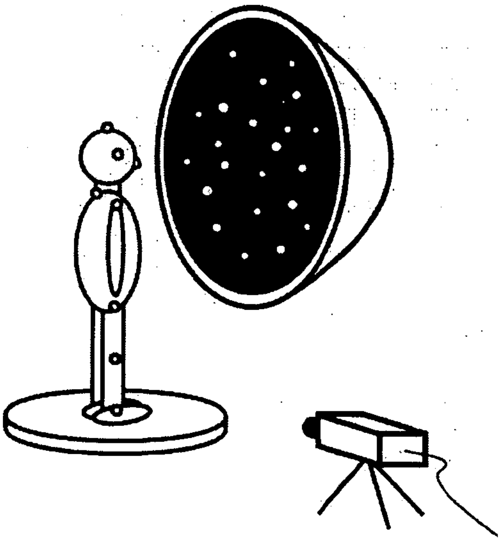

Figure 1. A schematic diagram showing the experimental set-up. Subjects stood in Romberg's posture and faced a hemispherical dome, the center of which was 75 cm away and approximately at eye level. Visually induced head and body sway was measured by monitoring seven reference points on the head and body using a video-motion analyzer.

We examined the relationships between the onset of self-rotation and the change in the temporal part of the head position, as the head motion in response to OKS was greater than that of other body parts for all cases. Two measures of head position changes were extracted from the raw data in a manner similar to that of Previc and Mullen [8]: the deviation component of postural change was obtained by smoothing the raw fore-and-aft movement using a 3-s averaging window, and the oscillation component was obtained by subtracting the deviation component from the raw data (Figure 2). The root-mean-square (r.m.s.) value of the oscillation was compared for 10 s before and after the onset of circular vection (CV). For statistical analysis, Student's paired *t*-test was applied. $p < 0.05$ was considered statistically significant. All subjects gave their informed consent to participate in the study.

Results

Figure 3 shows a typical dataset. As the downward visual stimulation progressed, the head and body gradually swayed forward; in this case the head swayed forward by >15 cm over the course of 60 s. For most subjects, postural change in response to vertical stimulation mainly occurred for the head and the upper half of the body. In one case, hip sway was also prominent (not shown). As the visual stimulation progressed, all subjects began to experience a compelling sensation of self-rotation in the opposite direction to that of the stimulus, i.e. upward rotational OKS produced a sensation of tumbling forward self-rotation and downward rotational OKS produced a sensation of backward self-rotation. Figure 2 shows the typical relationship between the two different components of the postural change and the onset of CV. The head essentially shifted in the same direction as that of OKS regardless of the onset of CV. However, the subjects showed apparent instability of the head after the onset of CV. The individual deviation component of the temporal part of the head position before and after the onset of CV is shown in Figure 4. For most subjects, the head swayed forward for downward OKS. In contrast, postural responses to upward OKS varied among individuals. For 7/10 subjects, the head sway for upward OKS was relatively small. For three subjects, there was a rapid backward sway at the time of onset of CV. However,

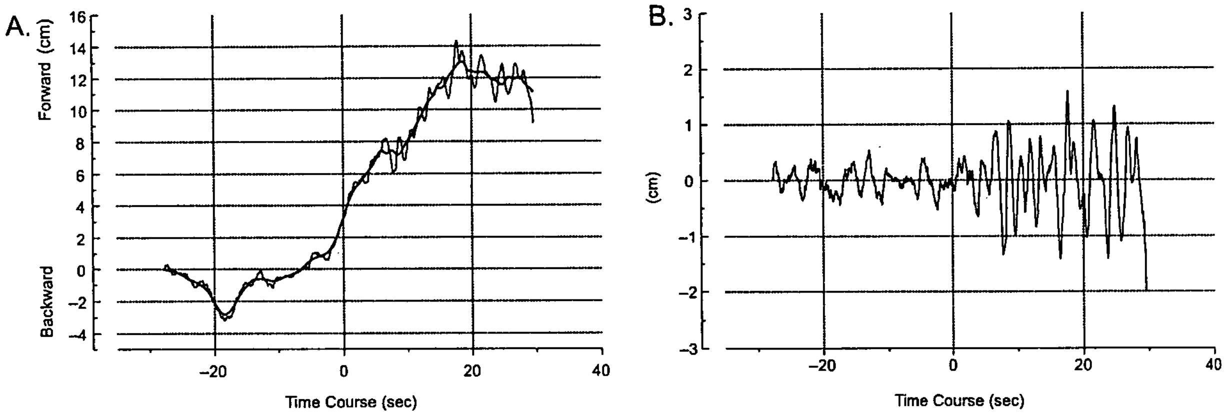

Figure 2. A typical example of postural changes: (A) the deviation component; (B) the oscillatory component. The subject's head shifted in the same direction as that of OKS regardless of the onset of CV. However, the subject showed apparent instability of the head after the onset of CV. The time "zero" indicates the onset of the self-motion perception.

the onset of self-motion perception did not change the direction of postural sway in either stimulus direction. The individual oscillation component of the temporal part of the head position before and after the onset of CV is shown in Figure 5. For the group tested, there were significant increases in postural instability after the onset of CV in both stimulus directions.

Discussion

In true motion, visually induced self-motion contributes largely to self-motion perception during motion at a constant velocity, as the vestibular system responds exclusively to accelerations during head and body motion and the deflected cupulae of the labyrinth return to their resting position by virtue of their elasticity after ≈20 s of rotation at constant velocity [13]. The dominant role of visually induced self-motion in postural adjustments has also been reported in subjects with bilateral vestibular deficits and in subjects in a condition of weightlessness [14,15].

By contrast, illusory self-motion perception may have an adverse impact on autonomic responses [16–18]. Vection is an essential factor in providing a compelling experience of "presence" in a virtual reality environment, but some individuals who experience vection may also experience motion sickness [17]. Vection is also known to induce vertigo and motion sickness when coupled with

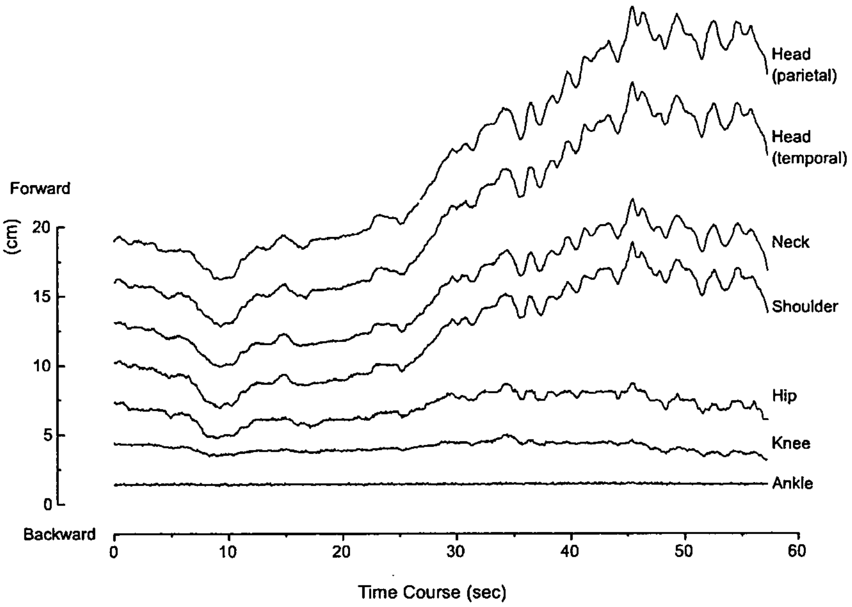

Figure 3. Video-motion analysis data. Changes in the subject's fore-and-aft movement over time are shown. Note that postural change in response to vertical stimulation occurred mainly for the upper half of the head and body.

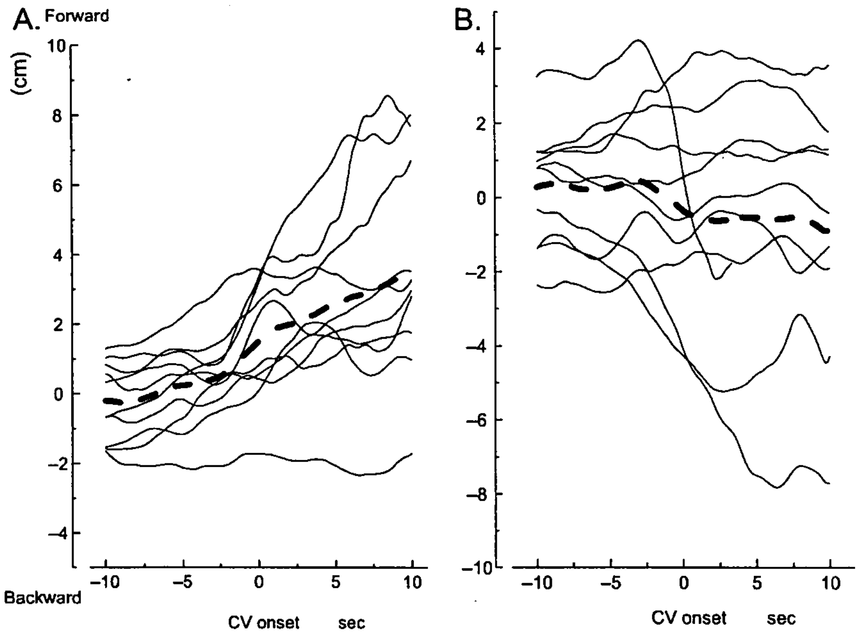

Figure 4. Changes in the deviation component for each subject over 20 s: (A) downward OKS; (B) upward OKS. The bold broken line indicates the mean of the 10 subjects.

incidental head movements [19]. The aim of this experiment was to investigate the effects of visually induced illusory self-rotation on postural responses. We used a new technique of video-motion analysis to determine which parts of the head and body react most to vertical motion in the visual surroundings.

In this study, the head and body swayed in the same direction as that of the vertical visual stimulus. When monitored with respect to different parts of the head and body, postural change in response to vertical stimulation mainly occurred for the upper half of the body. These results are consistent with those obtained in previous studies on roll vection [3–5] and linear vection (LV) [2,3,6,7]. When the subject is standing in front of earth-stationary visual surroundings, retinal slip information helps to keep

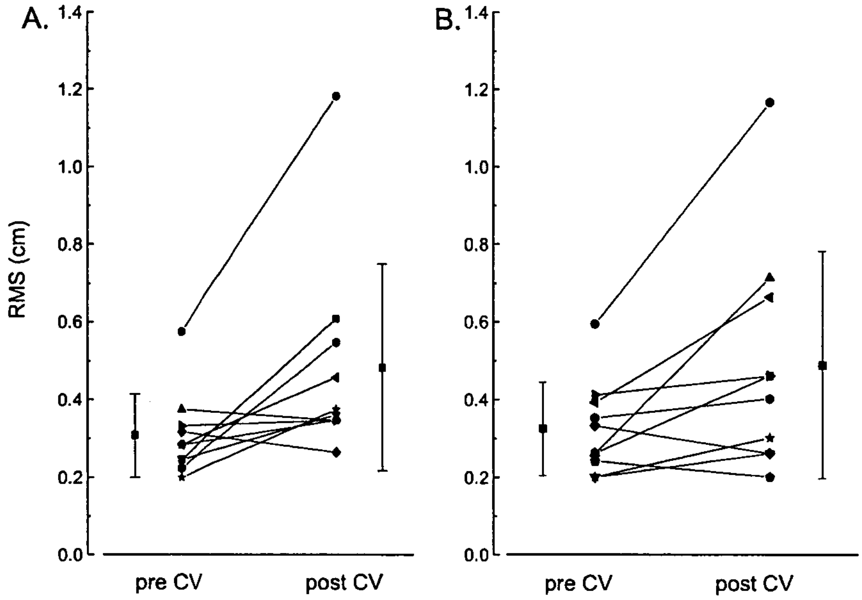

Figure 5. Individual oscillatory components for each of the 10 subjects: (A) downward OKS; (B) upward OKS. There was a significant increase in instability after the onset of CV in both stimulus directions. Each symbol corresponds to an individual subject. The vertical bars represent the SD of the mean (■) of 10 subjects.

the subject stationary. The amount of body sway with eyes open is ≈40–50% less than that with eyes closed [20,21]. Consequently, vision should control postural sway by generating eye rotation and postural reactions to stabilize the subject with respect to the visual world. The present study revealed an asymmetry in head and body displacement which was relatively larger for the forward direction. For LV, Lestienne et al. [7] observed that forward body tilt was larger than backward body tilt. Vertical visual motion is also known to induce up–down asymmetry in CV [11], illusory body tilt [12] and optokinetic nystagmus [22,23].

Our data showed that the direction of the displacements was primarily independent of the onset of self-motion perception. If the illusory self-motion did have a favorable impact on postural reactions such as real head and body motion in normal circumstances, the head and body might sway back in the direction opposite to that of the visual stimulus after the perception of self-motion. Instead, there was an increase in postural instability after the subjects began to perceive false self-motion in the opposite direction to that of the visual stimulus.

False perception of self-motion is reported to be a significant factor in certain operational environments and flight conditions. Pilots tend to respond to an illusion of vection with unnecessary aircraft control movements [24]. When an erect subject is exposed to a moving visual scene, the postural response is complicated by the fact that information from the vestibular and somatosensory systems continues to be in conflict with information from the visual system and the resultant illusion of self-motion perception. In conclusion, our results indicate that the illusion of self-motion could be a significant factor in inducing postural instability.

Acknowledgement

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References

[1] Fushiki H, Takata S, Watanabe Y. Influence of fixation on circular vection. J Vestib Res 2000;10:151–5.

[2] Bronstein AM. Suppression of visually evoked postural responses. Exp Brain Res 1986;63:655–8.

[3] Clement G, Jacquin T, Berthoz A. Habituation of postural readjustments induced by motion of visual scenes. In: Igarashi M, Black FO, editors. Vestibular and visual control on posture and locomotor equilibrium. Basle, Switzerland: Karger; 1985. p. 99–104.

[4] Dichgans J, Brandt T. Visual-vestibular interaction: effect on self-motion and postural control. In: Held R, Leibowitz HW, Teuber HL, editors. Handbook of sensory physiology, vol. 8. New York: Springer-Verlag; 1978. p. 755–804.

[5] Dichgans J, Held R, Young LR, Brandt T. Moving visual scenes influence the apparent direction of gravity. Science 1972;178:1217–9.

[6] Lee DN, Lishman JR. Visual proprioceptive control of stance. J Hum Mov Stud 1975;1:87–95.

[7] Lestienne F, Soechting J, Berthoz A. Postural readjustments induced by linear motion of visual scenes. Exp Brain Res 1977;28:363–84.

[8] Previc FH, Mullen TJ. A comparison of the latencies of visually induced postural change and self-motion perception. J Vestib Res 1990–91;1:317–23.

[9] Cheung BS, Howard IP, Nedzelski JM, Landolt JP. Circularvection about earth-horizontal axes in bilateral labyrinthine-defective subjects. Acta Otolaryngol (Stockh) 1989;108:336–44.

[10] Clement G, Lathan C. Postural reactions induced by vertical motion of visual scenes and the effects of weightlessness. Acta Otolaryngol (Stockh) 1998;118:466–73.

[11] Fushiki H, Takata S, Yasuda K, Watanabe Y. Directional preponderance in pitch circular vection. J Vestib Res 2000;10:93–8.

[12] Young LR, Oman CM, Dichgans JM. Influence of head orientation on visually induced pitch and roll sensation. Aviat Space Environ Med 1975;46:264–8.

[13] Cohen B, Henn V, Raphan T, Dennett D. Velocity storage, nystagmus, and visual-vestibular interactions in humans. Ann N Y Acad Sci 1981;374:421–33.

[14] Bronstein AM, Yardley L, Moore AP, Cleeves L. Visually and posturally mediated tilt illusion in Parkinson's disease and in labyrinthine defective subjects. Neurology 1996;47:651–6.

[15] Young LR, Shelhamer M. Microgravity enhances the relative contribution of visually-induced motion sensation. Aviat Space Environ Med 1990;61:525–30.

[16] Hettinger LJ, Berbaum KS, Kennedy RS, Dunlap WP, Nolan MD. Vection and simulator sickness. Mil Psychol 1990;2:171–81.

[17] Hettinger LJ, Riccio G. Sickness in virtual environments. Presence 1993;1:114–8.

[18] Regan EC, Price KR. The frequency of occurrence and severity of side-effects of immersion virtual reality. Aviat Space Environ Med 1994;65:527–30.

[19] Dichgans J, Brandt T. Optokinetic motion sickness and pseudo-Coriolis effects induced by moving visual stimuli. Acta Otolaryngol (Stockh) 1973;76:339–48.

[20] Diener HC, Dichgans J, Bacher M, Gompf B. Quantification of postural sway in normals and patients with cerebellar diseases. Electroencephalogr Clin Neurophysiol 1984;57:134–42.

[21] Paulus WM, Straube A, Brandt T. Visual stabilization of posture. Physiological stimulus characteristics and clinical aspects. Brain 1984;107:1143–63.

[22] Murasugi CM, Howard IP. Up-down asymmetry in human vertical optokinetic nystagmus and afternystagmus: contributions of the central and peripheral retinae. Exp Brain Res 1989;77:183–92.

[23] Van den Berg AV, Collewijn H. Directional asymmetries of human optokinetic nystagmus. Exp Brain Res 1988;70:597–604.

[24] Ungs TJ. The occurrence of the vection illusion among helicopter pilots while flying over water. Aviat Space Environ Med 1989;60:1099–101.

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ORIGINAL ARTICLE

Head and body sway in response to vertical visual stimulation

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Abstract

Conclusions. Postural responses differed according to the stimulus direction, i.e. vertical visual stimulation induced head rather than trunk displacements. Accordingly, it could be that center of foot pressure (COP) responses tended to underestimate the postural sway during visual stimulation. *Objectives.* To investigate head and body sway in response to vertical visual surround motion, and to examine the correlation between the displacements of head and body segments derived from video-motion analysis and COP measurements. *Material and methods.* Postural sway was assessed in 10 young female subjects by video-motion analysis of four different head and body segments, and by use of force-plate posturography. Head and body sway in the pitch plane was induced by rotating a random pattern of dots about the subject's inter-aural axis at a constant acceleration of $1^\circ/s^2$ or a constant velocity of 60°/s in darkness. *Results.* Generally, head displacement was greater than that of other body parts during vertical optokinetic stimulation (OKS). In most subjects, maximum head displacements were induced in the same direction as the visual motion. Downward OKS induced a forward head and body sway. The COP trajectory correlated well with the displacements of each head and body segment during downward OKS. In contrast, postural responses to upward OKS were complicated in terms of their time course. The correlation coefficient between each head and body segment and the COP varied among individuals for upward OKS.

Keywords: *Center of foot pressure, optokinetic stimulation, postural adjustment, up–down asymmetry, video-motion analysis*

Introduction

Human posture is controlled by sensory integrative information from the visual, vestibular and somatosensory systems. Stabilization of posture by the visual system has been confirmed by the results of experiments in which an erect subject is exposed to a stationary or moving visual surround; the amount of body sway decreases by 40–50% with eyes open compared to with eyes closed [1,2]. When an erect subject is exposed to a moving visual surround, the body has been reported to sway in the direction of the visual motion so as to stabilize the subject with respect to the visual world. While most studies of postural stabilization by vision have concerned lateral sway to linear or roll visual motion [3–9], several studies have also been conducted on fore–aft sway to linear visual motion [10,11]. For example, Lestienne et al. [11] have observed that linear visual motion in the fore-and-aft direction induces postural sway in the direction of the visual motion. The responses, however, were asymmetric in terms of the amplitude of fore-and-aft body tilt.

A force-measuring platform has been a successful tool in these studies of balance and postural control, as the center of foot pressure (COP) measurements derived from the platform are often used as an indicator of "body sway" [12]. However, postural responses to a moving visual surround are relatively complicated because information from the vestibular and somatosensory systems, which signal that the head and body have not really moved, remains in conflict with information from the visual system and the resultant illusion of self-motion perception for as long as the visual stimulus endures. We [13] have recently reported that visual self-motion perception leads to significant "head" instability, based on use of a video-based system. We report herein the head and body responses to vertical rotational visual stimulation, and examine the extent to which conventional COP measurements reflect head and body sway during vertical visual stimulation.

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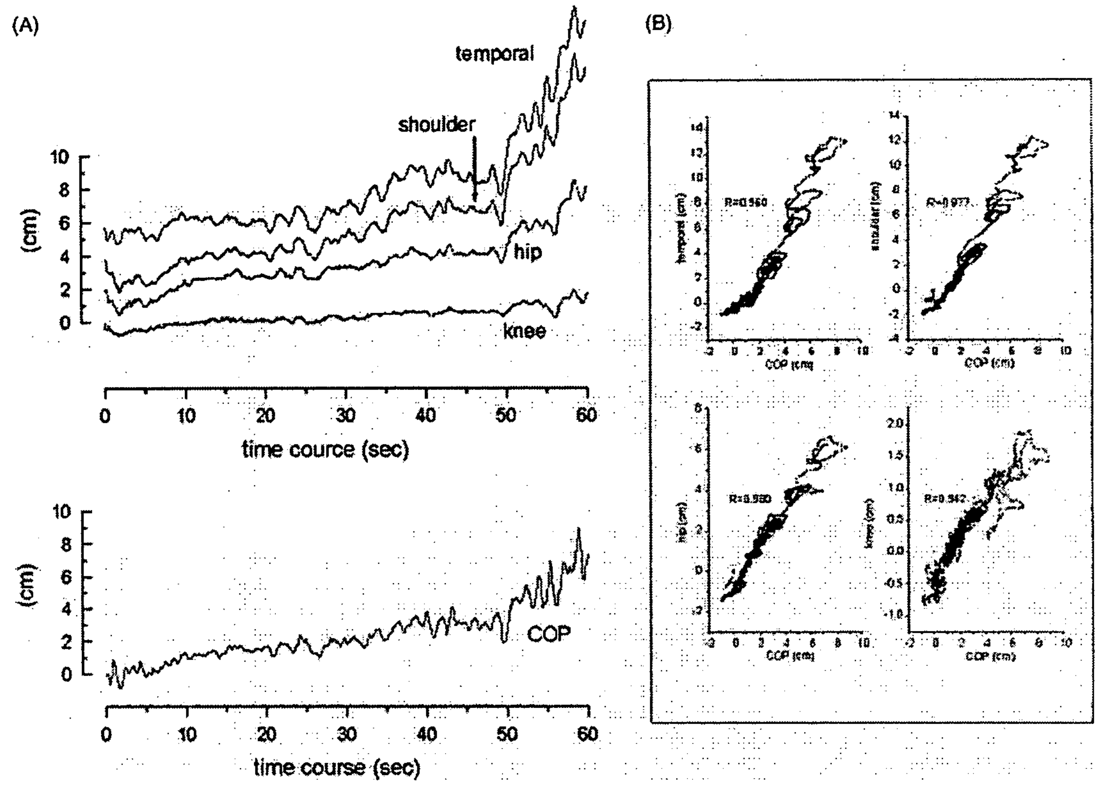

Figure 1. (A) An example of video-motion analysis and posturographic data for downward OKS. Changes in the subject's fore-and-aft movements over time are shown. From top to bottom on the upper graph, the waves show the movements of the head, shoulder, greater trochanter and knee for downward OKS at a constant acceleration. The lower graph shows the time course of the COP. (B) Scatter plots of the correlation and correlation coefficient between each segment of the body and the COP in the same subject. In this case, as the downward visual stimulation progressed, each segment of the head and body gradually swayed forward. The COP shifted in a similar trajectory for each head and body segment.

Material and methods

Ten young female adults ranging in height from 145 to 155 cm (mean 152.7 cm; SD 2.5 cm) and in age from 21 to 24 years (mean 22.0 years; SD 1.1 years) participated in this study. The subjects were volunteers with no history of ocular or vestibular disease, and were the same as those used in a previous study [13]. Subjects stood on a force-measuring platform in Romberg's posture and faced a hemispherical dome of radius 75 cm, with the center of the inside surface approximately at eye level at a distance of 75 cm. A random pattern of dots with diameters of 0.2–1.0° of the visual angle was projected onto the inner wall of the hemispherical dome. The pattern subtended 90° in the horizontal and 180° in the vertical direction. Two different stimulus patterns were used for induction of vertical head and body sway: the pattern was rotated about the subject's inter-aural axis (upward or downward) at a constant acceleration of $1°/s^2$ for 60 s or at a constant velocity of 60°/s for 60 s in darkness. The subjects were instructed to stare straight ahead at the stimulus pattern without attempting to follow the moving dots while standing.

The method of video-motion analysis was the same as described previously [13]. In brief, small red fluorescent markers were placed on the right side of the head (temporal part) and body (shoulder, greater trochanter and knee). The motion of each marker was recorded by a charge-coupled device camera, and was videotaped from the side at 60 fields (30 frames)/s. The video was digitized and sent to a PC for subsequent analysis. Force platform data were synchronously sampled at 30 Hz by a PC.

The head and body maximum displacement (amplitude between the extreme forward and backward positions) and the head and body mean position (distance from the initial position) during visual stimulation for 60 s were calculated from the displacements of the head and body segments for each subject. The head and body displacements were compared with the COP trajectory derived from the force-platform data by measuring the coefficient of correlation between them.

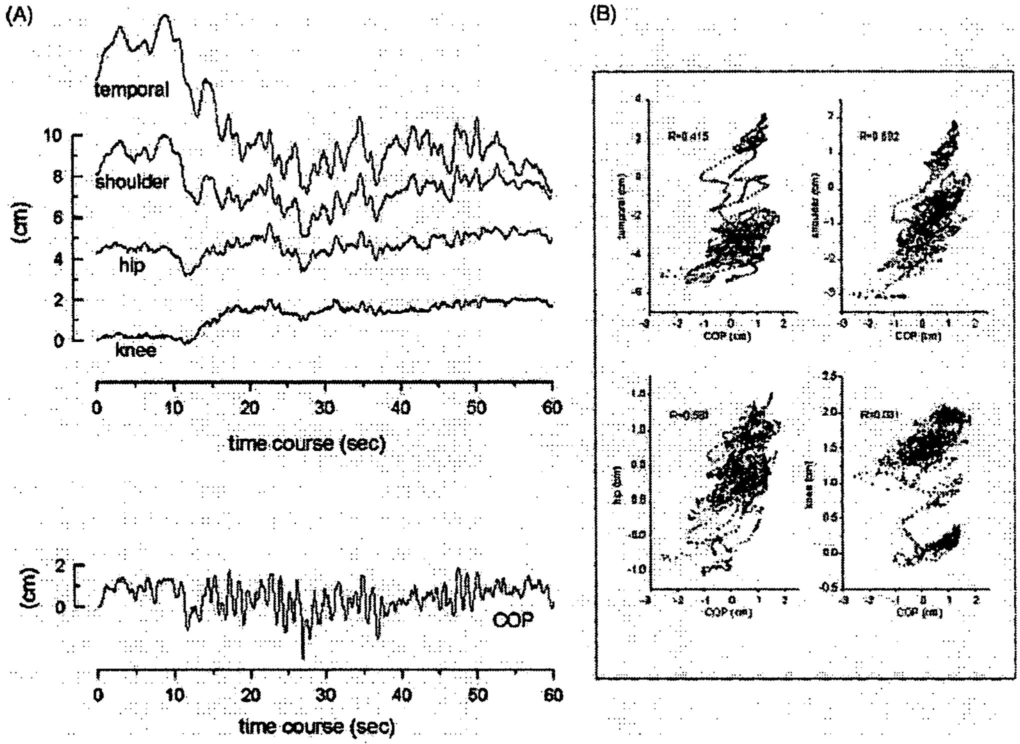

Figure 2. (A) An example of video-motion analysis and posturographic data for upward OKS. Changes in the subject's fore-and-aft movement during upward OKS at a constant velocity are shown. The composition of the Figure is the same as that of Figure 1. The direction of the head and shoulder changed as the visual stimulation progressed. The COP trajectory showed little displacement. (B) Scatter plots of the correlation and correlation coefficient between each segment of the body and the COP in the same subject.

Results

Figure 1 shows a typical data set. Downward visual surround motion induced a forward head and body sway for as long as the visual stimulus endured. The head displacement was greater than that of other body segments. In this case, the COP shifted in a similar trajectory for each head and body segment, with a high correlation. In contrast, postural responses were relatively complicated for upward optokinetic stimulation (OKS). For example, the head and shoulder initially swayed forward and, as the upward stimulation progressed, they were gradually directed backward, with a smaller hip and knee sway (Figure 2). In this case, there was little displacement of the COP monitored from the force platform, and the movement of the COP was ≈40% of head displacement. The correlation coefficient between the COP and each head and body segment was relatively low.

Figure 3 shows plots corresponding to the time at which maximum head sway occurred for each subject. In most subjects, the maximum head displacements were induced in the same direction as the vertical visual motion for both stimulus conditions. In some cases, however, the head was positioned in the opposite direction to the vertical visual motion. Head displacement was, on average, greater than that of other body parts for both downward and upward OKS (Table I). The mean positions of each head and body segment during downward OKS were, on average, in the forward direction. The mean positions of the head, shoulder and hip were, on average, in the backward direction during upward OKS, but knee position remained in the forward direction. Correlation coefficients between each head and body segment and the COP are shown in Table II. The correlation coefficients between the head and body segments and the COP were relatively high for downward OKS. In contrast, the correlation coefficient between each head and body segment and the COP varied among individuals for upward OKS. The correlation coefficient between the knee and the COP was lower than that for other segments of the head and body.

Discussion

The aim of this experiment was to examine the postural responses to vertical visual stimulation, and to investigate relationships between COP measurements and displacements of head and body segments elicited by visual surround motion.

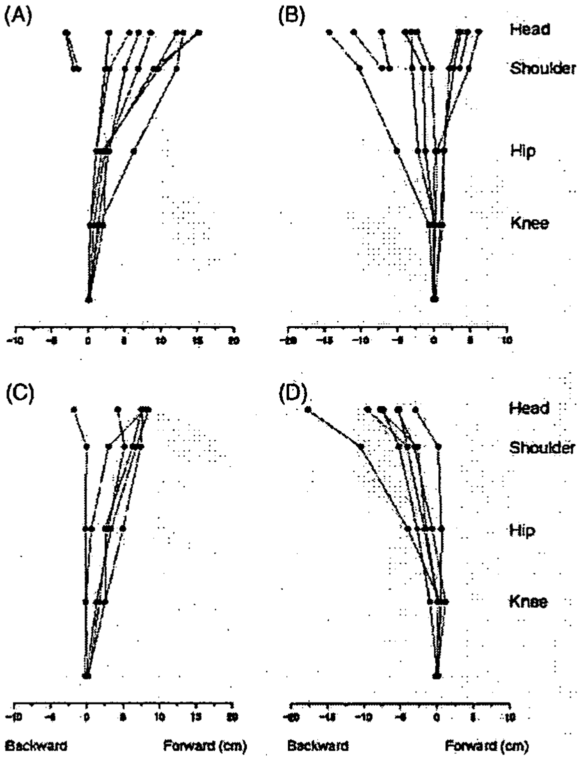

Figure 3. Plots showing the postural movement pattern at the time at which maximum head sway occurred. (A) Downward and (B) upward OKS at a constant acceleration. In 3/10 subjects, data for the hip and knee are not presented. (C) Downward and (D) upward OKS at a constant velocity.

Head and body pitch sway in the pitch plane was induced by rotating a large-field visual pattern at a constant acceleration of 1°/s^2 or a constant velocity of 60°/s. Under both stimulus conditions, for most subjects, vertical visual surround motion induced maximum head displacements in the same direction as that of the visual stimulus in a manner so as to stabilize the subject's head with respect to the visual world. The head and body were positioned in the forward direction during downward OKS, and the head, shoulder and hip were positioned in the backward direction during upward OKS. In this regard, the results are consistent with those obtained from previous studies of roll and linear visual motion [3–9]. However, we found that the postural response showed an asymmetry in its time course between the two stimulus directions. Head and body displacement was induced in the forward direction as long as the downward OKS endured. In contrast, postural responses to upward OKS over time were complicated. Some subjects showed postural instability and dissociative movements between the head and body during upward OKS.

Exposure to a moving visual surround not only leads to conflict with information from the vestibular and somatosensory systems, which signal that the head and body has not really moved, but also produces illusory self-motion perception. Vertical visual motion is known to induce up–down asymmetry in a sensation of illusory self-rotation [14] and body tilt [15]. One might therefore think that the asymmetry of postural instability over time would be related to the up–down asymmetry of self-motion perception. We [13] recently found that the illusion of self-motion perception leads to significant head instability. However, the degree of instability was found to be symmetric in the fore-and-aft direction. As such, the instability during upward OKS in some subjects may be explained in part by biomechanical factors. The base of support by the foot is asymmetric with respect to its center of rotation [16]. For linear visual motion in the fore-and-aft direction, the degree of inclination is larger in the forward than in the backward direction [11]. Head extension backward is known to increase postural instability [17]. Thus, postural responses to a visual scene may be

Table I. Average values of maximum displacement and mean position during visual stimulation for 60 s. The forward direction is indicated as positive for mean position.

	Downward OKS		Upward OKS	
	Max. displacement (cm)	Mean position (cm)	Max. displacement (cm)	Mean position (cm)
Acceleration ($n=10$)				
Head	10.18±5.00	1.82±2.28	7.47±3.85	−0.53±2.68
Shoulder	7.77±4.13	1.43±1.44	5.56±2.70	−0.48±2.08
Hip	4.21±1.93	0.70±0.63	2.47±1.31	−0.33±1.27
Knee	1.90±0.85	0.29±0.43	1.26±0.34	0.01±0.40
Constant velocity ($n=6$)				
Head	6.80±2.36	2.32±2.82	9.90±4.60	−3.22±3.46
Shoulder	5.49±1.89	2.23±2.24	7.16±2.78	−0.99±2.45
Hip	3.13±1.27	1.48±1.22	3.43±1.25	−0.06±1.35
Knee	2.00±0.88	0.93±0.77	1.77±0.53	0.49±0.76

Table II. Correlation coefficients between each head and body segment and the COP.

	Downward OKS			Upward OKS		
	Mean ± SD	Max.	Min.	Mean ± SD	Max.	Min.
Acceleration ($n = 10$)						
Head	0.90 ± 0.08	0.98	0.72	0.70 ± 0.21	0.90	0.25
Shoulder	0.92 ± 0.06	0.98	0.79	0.81 ± 0.13	0.97	0.52
Hip	0.94 ± 0.04	0.98	0.88	0.73 ± 0.15	0.93	0.55
Knee	0.85 ± 0.17	0.95	0.46	0.46 ± 0.30	0.79	−0.05
Constant velocity ($n = 6$)						
Head	0.87 ± 0.08	0.97	0.75	0.76 ± 0.18	0.90	0.42
Shoulder	0.90 ± 0.08	0.98	0.74	0.89 ± 0.09	0.97	0.69
Hip	0.92 ± 0.06	0.98	0.84	0.85 ± 0.14	0.95	0.58
Knee	0.81 ± 0.14	0.98	0.63	0.43 ± 0.43	0.86	−0.02

more stable in the forward direction, and suitable for forward locomotion.

COP measurements are a useful tool in studies of balance and postural control. Our data, however, show that vertical visual stimulation evokes head rather than trunk displacements. Consequently, in some cases, the COP responses tend to underestimate postural responses to visual motion.

References

[1] Diener HC, Dichgans J, Bacher M, Gompf B. Quantification of postural sway in normals and patients with cerebellar diseases. Electroencephalogr Clin Neurophysiol 1984; 57:134–42.

[2] Paulus WM, Straube A, Brandt T. Visual stabilization of posture. Physiological stimulus characteristics and clinical aspects. Brain 1984;107:1143–63.

[3] Bronstein AM, Buckwell D. Automatic control of postural sway by visual motion parallax. Exp Brain Res 1997; 113:243–8.

[4] Clement G, Jacquin T, Berthoz A. Habituation of postural readjustments induced by motion of visual scenes. In: Igarashi M, Black FO, editors. Vestibular and visual control on posture and locomotor equilibrium. Basle, Switzerland: Karger; 1985. p. 99–104.

[5] Dichgans J, Held R, Young LR, Brandt T. Moving visual scenes influence the apparent direction of gravity. Science 1972;178:1217–9.

[6] Ehrenfried T, Guerraz M, Thilo KV, Yardley L, Gresty MA. Posture and mental task performance when viewing a moving visual field. Brain Res Cogn Brain Res 2003;17:140–53.

[7] Lee DN, Lishman JR. Visual proprioceptive control of stance. J Hum Mov Stud 1975;1:87–95.

[8] Previc FH. The effects of dynamic visual stimulation on perception and motor control. J Vestib Res 1992;2:285–95.

[9] Thurrell AEI, Bronstein AM. Vection increases the magnitude and accuracy of visually evoked postural responses. Exp Brain Res 2002;147:558–60.

[10] Clement G, Lathan C. Postural reactions induced by vertical motion of visual scenes and the effects of weightlessness. Acta Otolaryngol (Stockh) 1998;118:466–73.

[11] Lestienne F, Soechting J, Berthoz A. Postural readjustments induced by linear motion of visual scenes. Exp Brain Res 1977;28:363–84.

[12] Winter DA. Sagittal plane balance and posture in human walking. IEEE Eng Med Biol Mag 1987;6:8–11.

[13] Fushiki H, Kobayashi K, Asai M, Watanabe Y. Influence of visually induced self-motion on postural stability. Acta Otolaryngol 2005;125:60–4.

[14] Fushiki H, Takata S, Yasuda K, Watanabe Y. Directional preponderance in pitch circular vection. J Vestib Res 2000; 10:93–8.

[15] Young LR, Oman CM, Dichgans JM. Influence of head orientation on visually induced pitch and roll sensation. Aviat Space Environ Med 1975;46:264–8.

[16] Eklund G, Lofstedt L. Biomechanical analysis of balance. Biomed Eng 1970;5:333–7.

[17] Brandt T, Krafczyk S, Malsbenden I. Postural imbalance with head extension: improvement by training as a model for ataxia therapy. Ann N Y Acad Sci 1981;374:636–49.

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Research Report

Efficacy of tilt-suppression in postrotatory nystagmus in cats

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ABSTRACT

The phenomenon of tilting the head away from an upright position immediately after a horizontal head-rotation, thus reducing the duration of postrotatory nystagmus (PRN), has more than once been called "tilt-suppression." It represents an example of the semicircular canal–otolith interaction in the central vestibular system. In the present study we investigated how head roll-tilt influences the time constants of PRN in the horizontal and vertical planes in cats. The head/body was roll-tilted by 30° toward the upright or the side down from initial roll positions immediately after termination of earth-vertical axis (EVA) rotation. Changes in head orientation either towards or away from the EVA reduced horizontal PRN. The reducing effect was small when the head was roll-tilted toward the EVA. Vertical nystagmus decreased only when the head orientation moved toward alignment with the EVA. Otolithic "tilt-suppression" may be a central neuronal mechanism that is activated to minimize the tumbling sensation of turning about a tilted axis and postural instability, but our results indicate that tilt-suppression of PRN depends on a change in head orientation with respect to the EVA.

1. Introduction

With sustained head rotation at a constant velocity, post-rotational vestibulo-ocular reflex responses (postrotatory nystagmus, PRN) last much longer than the time that would be predicted from knowledge of the mechanical properties of the semicircular canals (Blanks et al., 1975). This prolongation of PRN has been implicated as being a build-up of vestibular signals by the central vestibular system, which has been called a "velocity storage integrator (VSI)" (Raphan et al., 1979). Studies in monkeys have suggested that the semicircular canals, the otoliths, and the visual systems couple to the integrator, and that the VSI functions to maintain image-stabilizing nystagmus during rotation and counter destabilizing after nystagmus generated by opposite vestibular stimulation at cessation of rotation (Cohen et al., 1977; Raphan et al., 1979; Waespe et al., 1983).

Furthermore, studies in monkeys have demonstrated that VSI is responsible for encoding of the spatial vertical during motion (Angelaki and Hess, 1994a; Dai et al., 1991; Jaggi-Schwarz et al., 2000; Merfeld et al., 1993; Raphan and Cohen, 1988). For example, when the head is tilted away from an upright position immediately after termination of earth-vertical axis (EVA) head-rotation at a constant velocity, a cross-coupled component emerges gradually after the head tilts, and the rotation axis of PRN is finally directed toward the gravitational axis (spatial reorientation) (Angelaki and Hess, 1994a). In monkeys, the realignment toward the gravitational axis is accurate up to 90° of head tilt, and is independent of the plane of the initial VOR as well as the plane of head tilt (Angelaki and Hess, 1994a), suggesting that in monkeys VSI is a function of the 3-dimensional nature. In humans and cats, on the other hand, the axis shift of PRN toward gravity is limited to a much lower degree of head tilt. For head-roll tilt, the axis shift

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reaches its maximum at 15–30° of head tilt in humans (Fetter et al., 1992; Fetter et al., 1996) and at 30–45° of head tilt in cats (Yasuda et al., 2002). For head-pitch tilt, there is little reorientation (humans: (Fetter et al., 1992, 1996); cats: (Yasuda et al., 2003)). We found that in cat vertical PRN, there is little emerging horizontal component to reorient the direction of eye movements toward gravity (Fushiki et al., 2004). During and after the head tilt, a human subject experiences a tumbling sensation of turning about a tilted axis and has accompanying postural instability (Fitger and Brandt, 1982; Schrader et al., 1985a,b).

Tilting the head away from an upright position also suppresses PRN (tilt-suppression), as might be the strategy if the central nervous system recognizes that the head is in fact no longer rotating and therefore decides that it is best to cancel past estimates of motion. A post-rotatory tilt reduces the PRN duration to close to the prediction from knowledge of the mechanical properties of the semicircular canals (Angelaki and Hess, 1994b; Fetter et al., 1992). Since the 1960s, this phenomenon has been repeatedly observed in many species (e.g., humans: (Benson and Bodin, 1966; Fetter et al., 1992; Schrader et al., 1985a,b); monkeys: (Angelaki and Hess, 1994a; Merfeld et al., 1993; Waespe et al., 1985); cats: (Fushiki et al., 2004; Yasuda et al., 2002,2003)). For example, tilt-suppression for the horizontal PRN has been observed when the head is tilted toward the side down from the upright (Angelaki and Hess, 1994a; Benson and Bodin, 1966; Fetter et al., 1992; Merfeld et al., 1993; Schrader et al., 1985a,b; Waespe et al., 1985; Yasuda et al., 2002), as well as when the head is tilted forward or backward from the upright (Angelaki and Hess, 1994a; Benson and Bodin, 1966; Fetter et al., 1992; Merfeld et al., 1993; Schrader et al., 1985b; Yasuda et al., 2003). Tilt-suppression for the vertical PRN has also been observed when the head is tilted toward the upright from the side down (Angelaki and Hess, 1994a; Fushiki et al., 2004). Consequently, PRN may be suppressed by any deviation from the axis of rotation in which the initial vestibular stimulation has occurred.

The present study investigates whether tilt-suppression occurs in response to a post-rotatory tilt, irrespective of whether the rotation axis moves toward or away from the EVA. PRN was induced with the head rolled during EVA rotation in cats. The head was roll-tilted either toward the upright or toward the side-down during a PRN. Under this condition, either the head-horizontal or-vertical component of the induced PRN moves toward the EVA, and the other component moves away from the EVA by a post-rotatory tilt. Although a PRN is induced by progressively greater activation of the vertical canals and lesser activation of the horizontal canals as the head-roll during the EVA rotation progresses toward the side down, it is assumed that the head-horizontal eye movement is essentially induced by horizontal canal activation, while head-vertical eye movement is predominantly induced by vertical canal activation (Hain and Buettner, 1990). The decay changes of the head-horizontal and head-vertical components were separately examined after a head-roll tilt.

2. Results

Fig. 1 represents the typical time course of the horizontal and vertical SPV components of PRN evoked from a cat. The animal was maintained at a roll angle of 45° during vestibular stimulation. When stimulation was stopped, the cat's head was maintained in one of three orientations: (1) The head remained in the same 45° roll orientation with respect to the EVA (Figs. 1B, E). (2) The head orientation was shifted by 30° to a more extreme orientation of 75° with respect to the EVA (Figs. 1C, F). (3) The head orientation was shifted by 30° to a less extreme orientation of 15° with respect to the EVA (Figs. 1A, D).

When the head was maintained at a roll angle of 45° after the EVA rotation, the horizontal and vertical SPV decayed gradually with TCs of 5–6 s (Figs. 1B, E). When the head was roll-tilted by 30° toward the side down after the EVA rotation, the horizontal component decayed rapidly compared to that when the head was maintained at a roll angle of 45° (Fig. 1C). When the head was roll-tilted by 30° toward the upright after the EVA rotation, the tilting effect on the horizontal component was not prominent (Fig. 1A). The TC was similar to that when the head maintained its initial head position in this case. The vertical component, on the other hand, decayed rapidly when the head was roll-tilted by 30° toward the upright after the EVA rotation (Fig. 1D), but decayed gradually when the head was roll-tilted by 30° toward the side down after the EVA rotation (Fig. 1F).

The average TCs for horizontal and vertical SPV components for the four cats are shown in Fig. 2A. For both components, the significant tilting-suppression of the PRN was seen in one-way relative to the tilt-direction. That is, the horizontal TCs for tilts toward the side down were significantly shorter than those for no-tilts. The horizontal TCs for tilts toward the upright were slightly shorter than those for no-tilts, but they were not statistically significant. The vertical TCs for tilts toward the upright were significantly shorter than those for no-tilts. Conversely, the vertical TCs for tilts toward the side down tended to be longer. This tendency was also observed in cases of initial head positions of 30° and 60° (Table 1). A vectorial direction of PRN plane was initially directed to the earth-horizontal (Fig. 2B, thick line). After a head tilt toward either the upright or the side down, the direction of the PRN plane changed gradually, and it was ultimately directed to the earth-horizontal (Fig. 2B, dashed line). There were no significant differences in amplitude between no-tilts and post-rotatory tilts under any of the stimulus conditions.

3. Discussion

We examined the effects of changing head orientation on PRN. Tilt-suppression of PRN depended on a change in head orientation with respect to the EVA. Horizontal PRN decreased after roll-tilts that moved the head away from its position during vestibular stimulation. For a comparison of the same head orientation in space, TCs of the horizontal PRN after a post-rotatory tilt were much shorter than those maintained with an initial position (no post-rotatory roll tilt). Changes in head orientation either towards or away from the EVA were effective in reducing the horizontal PRN. But the reducing effect was small when the head was roll-tilted toward the upright. On the other hand, vertical PRN decreased only when the head orientation moved toward alignment with the EVA.

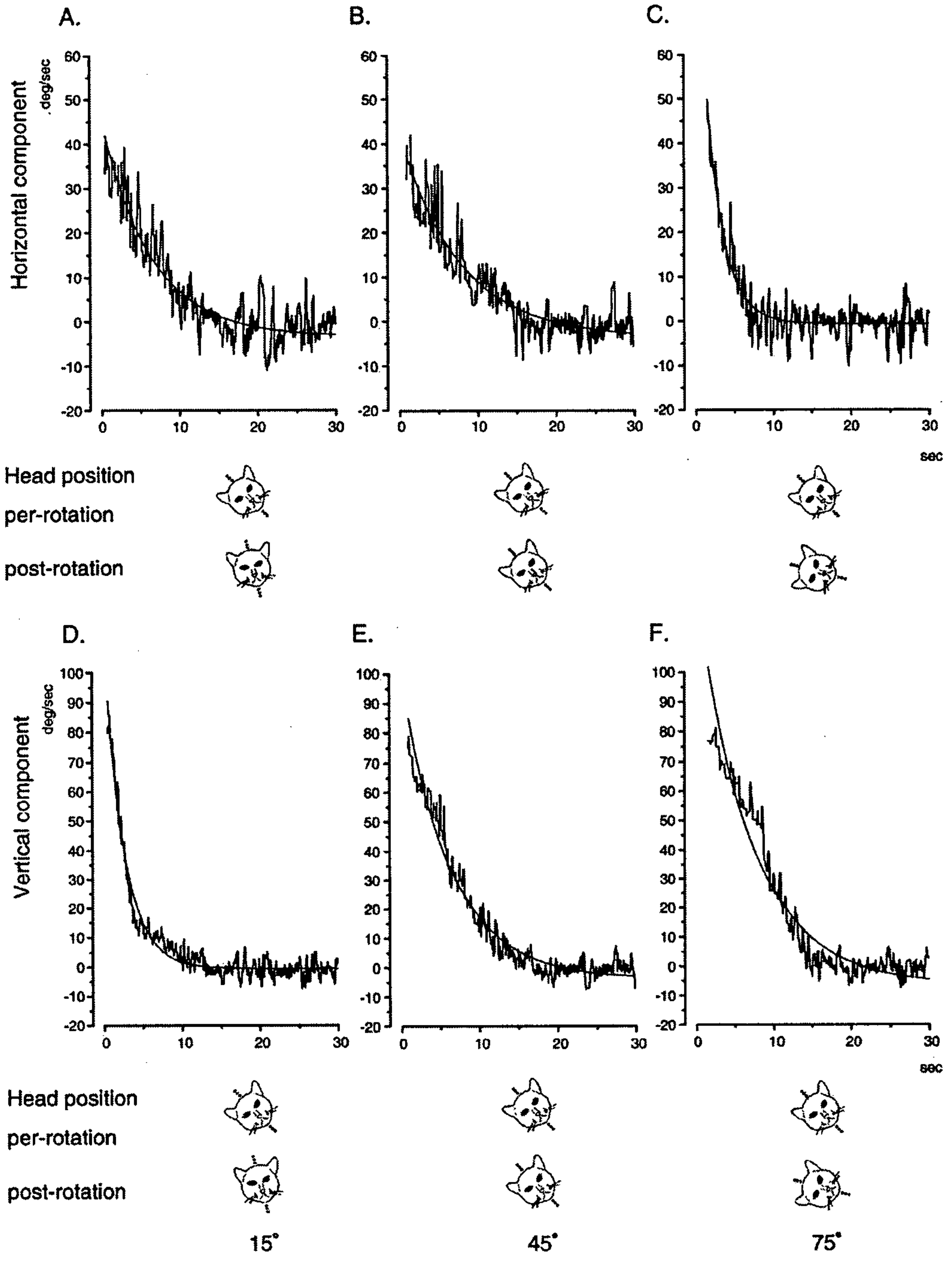

Fig. 1 – Examples of the time course of head-horizontal and-vertical SPV of PRN evoked from a cat by the EVA rotation such that the EVA was aligned with a diagonal axis in the head-roll plane. (B, E) PRN is illustrated in a cat maintained at a head-roll angle of 45° with respect to gravity. PRN decayed gradually with the slow phases of the right eye directed rightward and upward. (A, D) Immediately after the EVA rotation was stopped, the same animal was roll-tilted by 30° toward the upright. (C, F) Immediately after the EVA rotation was stopped, the same animal was roll-tilted by 30° toward the side down. "0 s" indicates the termination of the EVA rotation.

Rather, vertical PRN tended to be longer when the head orientation moved away from the EVA.

Cerebellar nodulus-uvula is the site responsible for controlling the velocity storage component of eye reflexive movement. The nodulus-uvula contributes to modification of the TC of PRN (Waespe et al., 1985) as well as to control of the spatial orientation of PRN (Wearne et al., 1998). Tilt-suppression of the horizontal PRN becomes void in patients with uvulo-nodular lesions (Wiest et al., 1999). However, the horizontal and vertical components of velocity storage seem

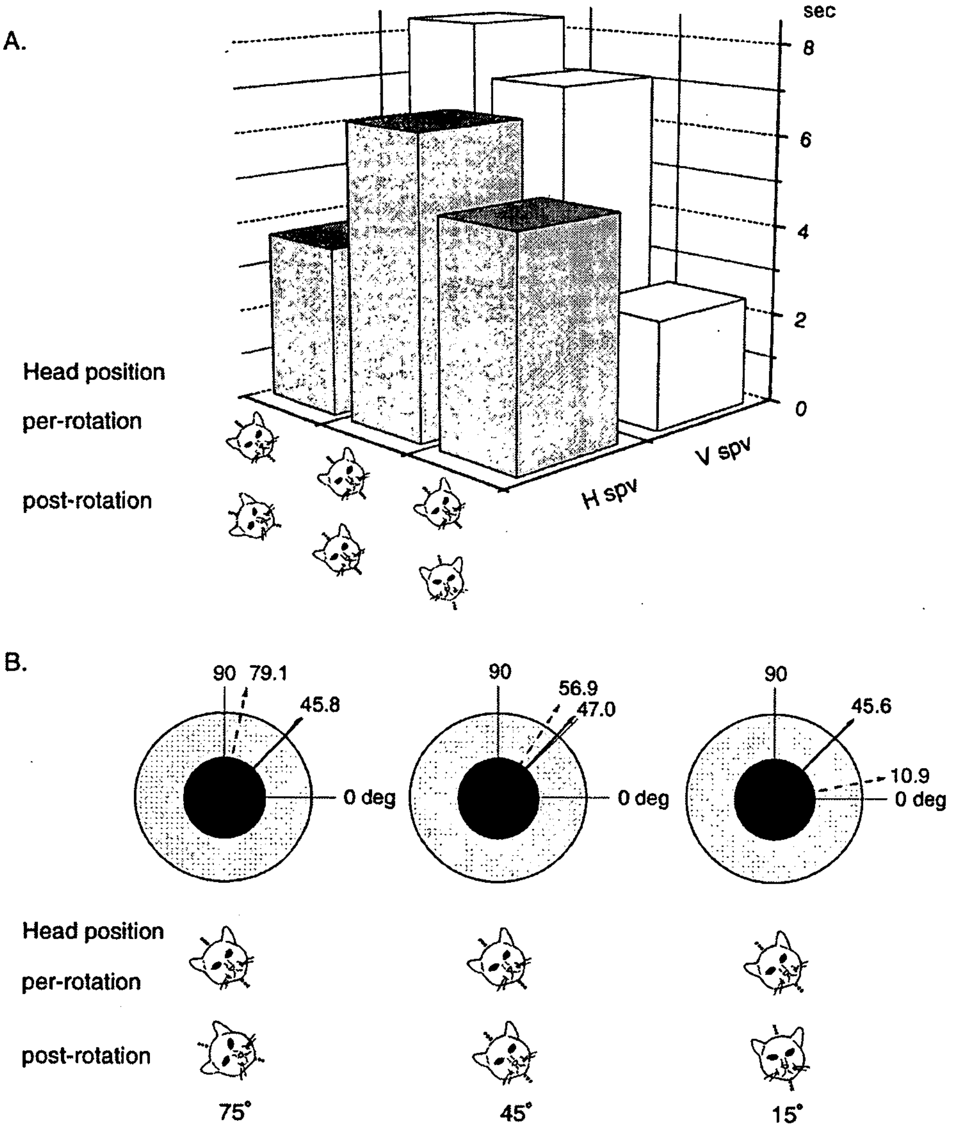

Fig. 2 – The effects of head-roll tilt on the PRN were examined in a group of four cats. (A) The average TCs for the horizontal and vertical SPV are shown. The effect was direction-specific, in that suppression occurred only following a stimulus. Significant tilt-suppression for the horizontal and vertical components was seen when nystagmus moved away from the EVA. The vertical TC tended to be longer for tilts toward the side down. (B) The average final angles of the PRN plane are shown. PRN was reoriented toward the earth-horizontal for the head tilt of 30° toward either the upright or the side down. The thick line and dashed line indicate initial and final angles of PRN plane, respectively. The angle "0°" corresponds to the orbital right, and the angle "90°" corresponds to the orbital up.

to be controlled separately. After ablation of the uvula-nodulus in the monkey, the vertical and roll TC were found to decrease, while horizontal TC increased (Wearne et al., 1998). The horizontal and vertical central vestibular systems differ in their contribution of otolith inputs to the semicircular canals within those circuits [Barmack, 2003 for review]. In the present study, TCs for the head-vertical component increased slightly when the head tilt moved toward the EVA. In other words, the vertical component tended to be longer as the animal was positioned toward the side down. Studies in monkeys showed that TC of vertical PRN decreased as the animals were tilted toward the upright position and increased as they were tilted toward the upside-down position (Angelaki and Hess, 1994a). For per-rotatory vertical VOR during off-axis rotation at a constant velocity, the duration of nystagmus not only decreases but also increases, according to the change in the linear force acting on the otolith organs (Angelaki et al., 1991). It is thus likely that otolith input contributes differently to horizontal VOR than to vertical VOR.

We also observed that PRN was reoriented toward gravity after the head tilt toward either the upright or the side down. However, the tilt-suppression and reorientation seem to not