RESEARCH NOTES

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Effects of bilateral vestibular loss on podokinetic after-rotation

Abstract We asked what the role of the vestibular system is in adaptive control of locomotor trajectory in response to walking on a rotating disc. Subjects with bilateral vestibular loss (BVL) were compared to age- and gender-matched controls (CTRL). Subjects walked in place on the surface of a rotating disc for 15 min and then attempted to step in place without vision on a stationary surface for 30 min. CTRL subjects demonstrated podokinetic after-rotation (PKAR), involuntarily and unknowingly turning themselves in circles while attempting to step in place. PKAR in CTRLs was characterized by a rapid rise in turning velocity over the first 1–2 min, followed by a gradual decay over the remaining 28 min. Subjects with BVL also demonstrated PKAR and had no knowledge of their turning. However, PKAR in BVLs was characterized by an extremely rapid, essentially instantaneous rise. Subjects with BVL immediately turned at maximum velocity and exhibited a gradual decay throughout the entire 30 min period. Despite this difference in the initial portion of PKAR in BVLs, their responses were not significantly different from CTRLs during minutes 2 to 30 of the response. These results suggest that vestibular inputs normally suppress PKAR velocity over the first 1–2 min of the response, but do not greatly influence PKAR decay. PKAR is therefore a process mediated primarily by somatosensory information and vestibular inputs are not required for its expression. Additionally, the absence of vestibular inputs does not result in increased somatosensory sensitivity that alters podokinetic intensity or decay time constants.

Keywords Vestibular system · Somatosensory system · Adaptation · Locomotion

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Introduction

When walking in everyday environments, one must change walking direction frequently to round corners and avoid obstacles. In fact, walking a straight line is the exception, rather than the norm. Several sensory modalities, including vision, vestibular sensation, and somatosensation, play a role in locomotor trajectory control. We have developed a paradigm that uses a rotating circular treadmill to examine the contributions of vestibular and somatosensory information to the adaptive control of locomotor direction.

Following stepping in place on a rotating disc, healthy subjects will turn in circles when asked to step in place on a stationary surface in the absence of vision (Gordon et al. 1995). This response, called podokinetic after-rotation (PKAR), likely represents a somatosensory-mediated remodeling of the rotational relationship between the trunk and the feet (Weber et al. 1998). There is evidence, however, that the vestibular system may also play a role in shaping the PKAR response. At the start of PKAR, healthy subjects show a characteristic rise in rotational velocity over the first 1–2 min, followed by a gradual decay in velocity from mins 2–30. Weber et al. (1998) hypothesized that the initial rise of PKAR was the result of vestibular-somatosensory interactions. The initial rotation of PKAR would stimulate the semicircular canals and produce a vestibular signal that opposes the podokinetic somatosensory drive. Given the semicircular canal time constant of roughly 15 s (Wilson and Melvill Jones 1979), and the PKAR time constant of 6–12 min, it is hypothesized that the vestibular system does not influence PKAR beyond the first min. However, previous studies showed that there was no change in the initial 1–2 min of PKAR in subjects with unilateral vestibular loss, as compared to healthy controls (Weber et al. 2002). Thus, we conducted a study of subjects with bilateral vestibular loss (BVL) to determine whether the vestibular system plays a role in suppression of PKAR over the initial 1–2 min and whether the decaying portion of PKAR is indeed free of vestibular influence. We hypothesized that

subjects with bilateral vestibular loss would demonstrate PKAR that lacked the initial rise and had higher maximum velocity. We also expected BVL subjects to demonstrate longer-lasting PKAR, as the weighting of somatosensory information increases in individuals with bilateral vestibular loss (Nashner et al. 1982; Bles and Roos 1991; Peterka 2002).

Materials and methods

Subjects

Seven subjects with bilateral vestibular loss (BVL) and 7 age- and gender-matched control (CTRL) subjects took part in this study. The mean age of the BVL group was 58±3 years and the mean age of the CTRL group was 56±5 years. Test results confirmed that all BVL subjects demonstrated reduced yaw VOR gain at 0.05, 0.2, and 0.8 Hz. Refer to Table 1 for a summary of the BVL subject information. All subjects were free of any musculoskeletal, neurological or neuromuscular disorder. All subjects provided informed consent prior to participation in the study. The study was approved by the institutional review board of Oregon Health & Science University.

Protocol

Prior to podokinetic (PK) stimulation, all subjects underwent tests of light touch using Semmes-Weinstein Monofilaments, position sensation, and vibration to ensure normal somatosensory functioning in the feet. The sensory tests were within normal limits in all of the subjects. Each subject was then exposed to 15 min of PK stimulation. A duration of 15 min was chosen based on the work of Weber et al. (1998), to ensure robust PKAR responses with a minimal duration of treadmill stimulation. Subjects stepped in place in the center of the rotating treadmill while it turned in the counterclockwise direction at 60 deg/s. All subjects stepped at a cadence of 2 Hz, maintained by a metronome attached to the trunk. Subjects performed this period of treadmill walking with eyes open, as Jurgens et al. (1999) showed that PKAR does not result from a conflict between visual input and somatosensory input, and that elimination of visual inputs during stimulation does not affect the after-rotation. Subjects wore a safety harness and held an overhead low-friction wheel to maintain stability. Post-adaptation responses were assessed following PK stimulation. The treadmill was stopped and subjects were blindfolded and given earplugs. Subjects were then instructed to step in place for 30 min at the 2 Hz cadence established by the metronome, while holding the overhead wheel.

Data analysis

Angular velocity of the trunk was measured relative to the ground using a Motion Analysis Corporation system (Santa Rosa, CA). To do this, three markers were placed on the trunk at the acromion processes and to the left of the sternum, and two markers were placed on the ground around the perimeter of the treadmill. Motion Analysis output the angle between a line drawn between the two acromion markers and a line drawn between the two ground markers (as seen from an aerial view) at a frequency of 60 Hz. As a subject turned, these angle data created a sawtooth pattern that increased up to 180 degrees and then decreased back to zero. These angle data were used to calculate a running slope at each 1 s interval. Some slope values of the sawtooth waveform were negative, despite the fact that all subjects turned clockwise during the entire PKAR period. To remove this artifact from the data, angular velocities were taken as the absolute value of the running slope. The angular velocities were grouped into 5-s bins and plotted over the 30-min adaptive trial using Sigmaplot 5.0 (SPSS, Richmond, CA). For each subject, the PKAR response was divided into two parts. The first 2 min of the response were fitted with an exponential growth function to determine maximum velocity and rise time constant, while the following 28 min were fitted with a three-parameter exponential decay function that yielded values for initial velocity, decay time constant and final asymptote. [Data from one BVL subject (subject 3 in Table 1) could not be satisfactorily fit with the decay function, so decay curve fit parameter averages presented in Table 2 are from only six BVL subjects and their six matched controls.] We also measured maximum velocity, time at which this maximum velocity was reached, and average angular velocity across minutes 15–30 for each subject. Statistical comparisons between the groups were performed using independent t-tests ($P=0.05$). The rates of acceleration over the first min of the response in CTRLs were also calculated.

Results

All subjects completed the task without difficulty or loss of balance. All BVL and CTRL subjects demonstrated continuous, clockwise PKAR when instructed to step in place following 15 min of counterclockwise PK stimulation. This effect was robust and long-lasting, with all

Table 1 BVL subject information

ID	Age	Gender	Length of injury (years)	Cause of BVL	Yaw VOR gain at 0.05 Hz	Yaw VOR gain at 0.2 Hz	Yaw VOR gain at 0.8 Hz
1	57	F	11	Idiopathic	0.005	0.007	0.11
2	61	M	9	Ototoxicity	0.05	0.13	0.18
3	73	M	7	Ototoxicity	0.02	0.16	0.42
4	53	M	6	Ramsey-Hunt syndrome	0.26	0.04	0.12
5	64	M	2	Ototoxicity	0.03	0.53	0.57
6	45	F	6	Ototoxicity	0.03	0.08	0.16
7	56	F	5	Idiopathic	<0.39[b]	<0.40[b]	<0.59[b]

[a] Normal yaw VOR gains (95% confidence intervals) = 0.39–1.02 for 0.05 Hz, 0.40–1.02 for 0.2 Hz, and 0.59–1.07 for 0.8 Hz (Peterka et al. 1990)

[b] Subject unable to tolerate standard yaw VOR testing protocol, but was clearly well below normal range at all frequencies according to the EOG recordings obtained

Table 2 BVL vs. CTRL values (mean ± SE)

Parameter	BVL	CTRL
Asymptote (deg/s)	1.3±1.1	3.0±0.5
Initial velocity (deg/s)	8.4±0.7	8.5±1.6
Decay time constant (min)	9.6±1.4	12.6±4.9
R^2 2–30 min	0.7±0.1	0.7±0.1
R^2 0–2 min	0.0±0.0[a]	0.5±0.1
Maximum velocity (deg/s)	16.2±1.7	12.5±1.9
Time of peak velocity (min)	0.7±0.3[a]	1.4±0.1
Average velocity 15–30 min (deg/s)	4.6±0.7	2.9±0.8

[a]Significant difference between BVL and CTRL groups, $p<.05$

subjects continuously turning clockwise throughout the entire 30-min period of data collection. All subjects, regardless of vestibular status, reported no sensation of turning during PKAR.

Figure 1A shows the first 2 min of PKAR for a BVL subject (filled circles) and a matched CTRL subject (open circles). Note the striking difference in the initial portions of the two curves. The CTRL subject has an initial rise in PKAR velocity over the first 1–2 min of the response. All

CTRL subjects demonstrated this pattern of initial rapid rise in rotational velocity. The average acceleration from 0–1 min in CTRLs was $1.93±0.28$ deg/s^2 (mean ± SE). The BVL subject, on the other hand, showed an extremely rapid, nearly instantaneous initial rise. He immediately began turning at maximum velocity and showed a progressive decline over the entire 30-min period of PKAR. All seven BVL subjects lacked the initial rising phase of the PKAR response. We were unable to fit the first 2 min of the BVL subjects' responses with an exponential rise-to-maximum function, given the extremely rapid initial rise in PKAR velocity among this group. Thus, we measured the time at which maximum velocity was reached. Maximum velocity was reached significantly earlier in BVL subjects as compared to CTRL subjects (Table 2).

There was also a tendency for BVL maximum velocity values to be higher than those of CTRL subjects (Table 2, Fig. 2A). In six of the seven pairs of age-matched subjects, the maximum velocity was higher for the BVL than for the CTRL subject. However, the difference between groups was not significant due to high intersubject variability.

Despite differences in the initial portion of the PKAR response that made the use of an exponential rise to
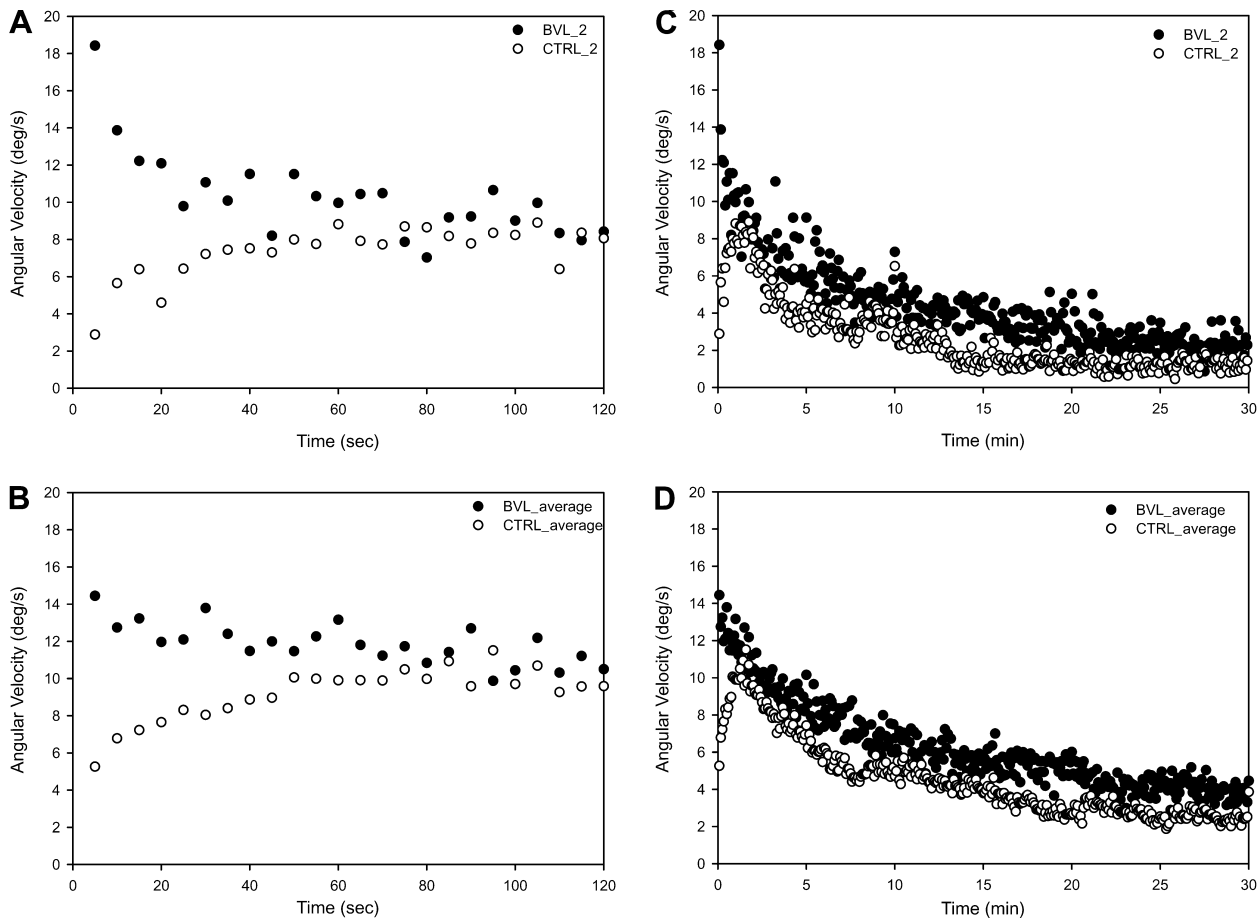

Fig. 1 Plots of the first 2 min of the PKAR response for a BVL subject and a matched CTRL **(A)**, and average responses of the BVL and CTRL groups **(B)**. Note the absence of an initial rise in the BVL group, as compared to the CTRL group. Despite this difference in initial rise, there were no differences in the decaying portion of the curves for the BVL or CTRL groups. The entire 30-min PKAR response is shown for a BVL subject and matched CTRL **(C)**, as are average responses of the BVL and CTRL groups **(D)**

Fig. 2 Maximum PKAR velocity (**A**) and average velocity from minutes 15–30 (**B**) for each individual in the BVL and CTRL groups. Note the tendency, in all but one pair, for the BVL subject to have a higher maximum velocity and higher average velocity from 15–30 min than their age-matched CTRL subject. **C** is a schematic and highly simplified illustration, showing how vestibular inputs may normally summate with somatosensory inputs during the initial portion of the PKAR response but likely have no effect later in the response. Note the similarity of the predicted response in the absence of vestibular input to the actual responses depicted in Fig. 1
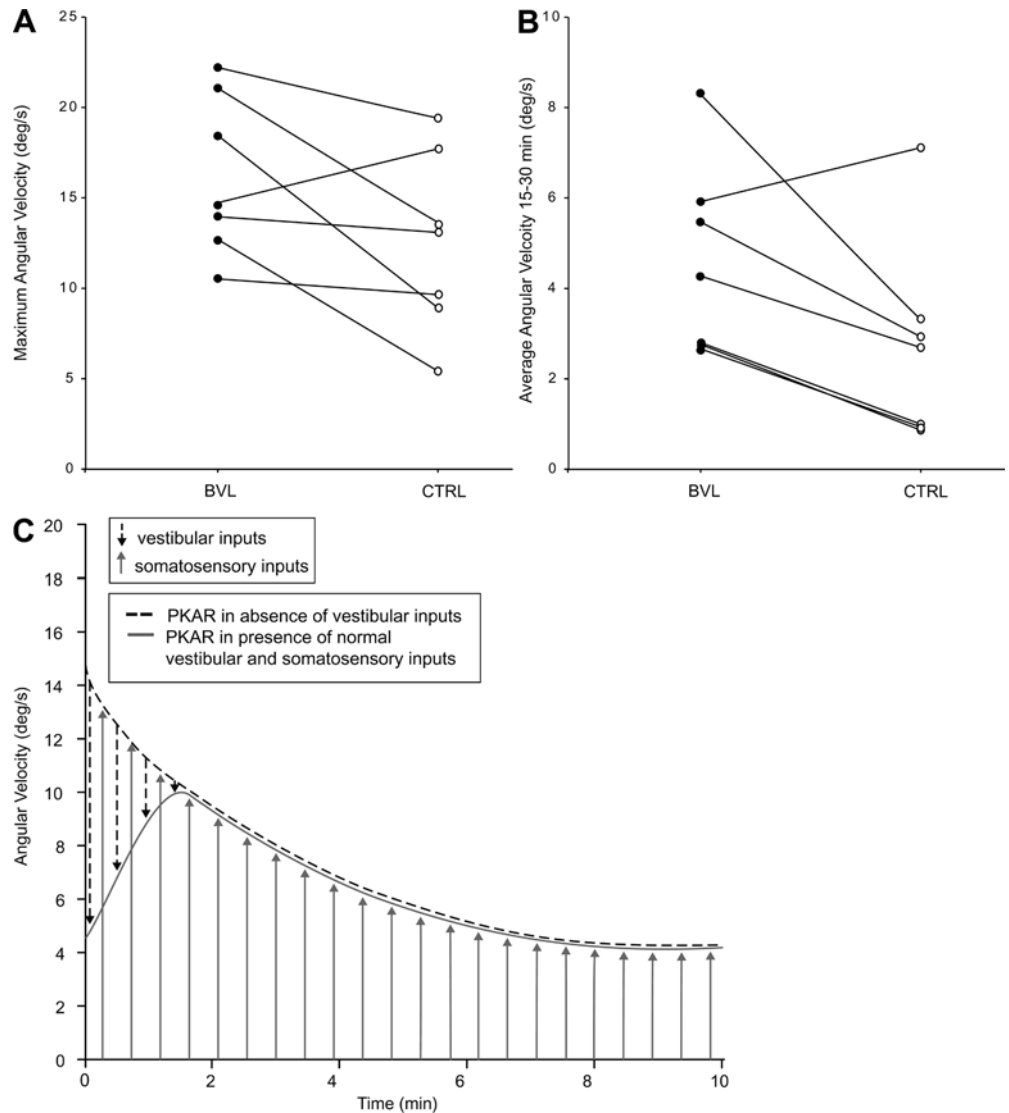
maximum curve fit inappropriate, an exponential decay curve fit could still be successfully and validly applied to six of the seven BVL and seven CTRL subject responses during the final 28 min. The single BVL subject's data that could not be fit with an exponential decay showed a very slow rate of decay and highly variable velocities throughout the response.

Values for initial velocity, decay time constant, and asymptote were obtained (Table 2). There were no significant differences in curve fit values for the two groups. Figure 1B shows the average responses of the BVL and CTRL groups during the first 2 min of PKAR. Figure 1C shows the entire 30-min response of an individual BVL and a matched CTRL subject, while Fig. 1D shows group average results across the 30-min period of PKAR. Although six of seven BVL subjects had higher angular velocities than their matched controls during the second half of PKAR (Fig. 2B), there was no significant difference in average angular velocity from 15–30 min between the groups.

Discussion

As hypothesized, the vestibular system clearly plays a role in the initial portion of the PKAR response. Vestibular inputs suppress PKAR angular velocity over the first 1–2 min. This result is consistent with the very rapid initial rise in velocity noted when a servo-controlled turntable was used to allow PKAR to occur without stimulating the vestibular system in CTRL subjects (Weber et al. 1996). However, PKAR velocities are clearly above the vestibular sensory threshold of 1–2 deg/s (Mergner et al. 1993). Thus, although vestibular inputs appear to be suppressing PKAR velocity, this suppression is not sufficient to keep velocity below the normal perceptual threshold. Despite this fact, subjects have no perception of turning during PKAR. This suggests that vestibular perception may be suppressed in favor of somatosensory information, as has been noted in similar situations (Mergner et al. 1993). After 15 min of equating leg rotation on the trunk with body stability in space on the rotating surface, subjects appear to continue to consider the somatosensory

information from their rotating legs as an indication of body stability in space. Dietz et al. (2001) also provide evidence for somatosensory suppression of vestibulospinal drive, although this suppression is only partial and does not completely eliminate the vestibular influence during stepping in place.

After the initial 1–2 min of PKAR, there appears to be no substantial vestibular influence, as evidenced by the similar decay characteristics of PKAR in the BVL and CTRL groups (Fig. 2C). If PKAR resulted from the vestibular velocity storage mechanism receiving somatosensory signals during walking on the rotating surface, we would expect to see disrupted post-rotatory decay for a longer period in the BVL subjects. The apparent summation of vestibular and somatosensory inputs during the initial portion of PKAR is in keeping with studies of locomotion (Marlinsky 1992), where there is evidence for summation of these inputs at slow velocities (Jahn et al. 2000). Vestibular information is also integrated with somatosensory information during locomotion to remembered targets, with loss of vestibular input resulting in alterations in path curvature despite normal somatosensory inputs (Glasauer et al. 1994; Takei et al. 1996). Vestibular inputs are critical for self-turning to a target, during which the presence of vestibular information is a prerequisite for optimal use of podokinesthetic information (Becker et al. 2002). This interdependence of vestibular and proprioceptive inputs is also apparent in postural control tasks. Vestibular information is important for interpreting somatosensory information regarding support surface orientation (Mergner and Rosemeier 1998), as evidenced by an increased instability in people with BVL when standing on an inclined surface (Kluzik et al. 2002). In contrast to the tasks examined in these studies, however, PKAR is of very long duration and has no abrupt accelerations or decelerations after the initial rise typically seen during the first two minutes. As such, the vestibular signal is likely present to summate with the proprioceptive signal only during the initial portion of the response, but the properties of the vestibular system are such that it no longer detects the rotation of the head (and body) in space during the remainder of PKAR. Given the properties of the semicircular canals, and their time constant of approximately 15 s, it is not surprising that vestibular signals do not strongly influence the decaying portion of the curve.

Previous studies of PKAR have shown a closed loop relationship between the vestibular and podokinetic systems, demonstrating that vestibular stimulation alone can elicit a PK response (Melvill Jones et al. 2000). Subjects with unilateral vestibular loss showed asymmetrical, lesion-dependent asymmetries thought to reveal occult imbalance of the vestibulospinal drive to the podokinetic system (Weber et al. 2002). Furthermore, it is known that individuals with vestibular loss increase the emphasis placed on remaining sensory systems. For example, people with vestibular loss show an increased reliance on proprioceptive information during balance tasks (Nashner et al. 1982; Peterka and Benolken 1995; Peterka 2002). As such, we expected to see an increased

weighting of PK information in the BVL group, which would manifest as a response with higher amplitude and slower decay throughout the 30-min period of PKAR. We noted a tendency toward higher PKAR maximum velocity and higher average velocity in the latter half of PKAR in the BVL subjects, which may indicate a slightly increased sensitivity to somatosensory information during the PKAR response. However, there was no difference in PKAR decay rate between the groups. This lack of substantial sensory reweighting during the major portion of PKAR may relate to the fact that PKAR in the healthy subject is relatively free of vestibular influence after the first 1–2 min. Thus, the absence of vestibular information may not be a critical factor in control of the decaying portion of PKAR, and as such substantial reweighting is not necessary.

References

Becker W, Nasios G, Raab S, Jurgens R (2002) Fusion of vestibular and podokinesthetic information during self-turning towards instructed targets. Exp Brain Res 144:458–474

Bles W, Roos JW (1991) The tilting room and posturography. Acta Otorhinolaryngol Belg 45:387–391

Dietz V, Baaken B, Colombo G (2001) Proprioceptive input overrides vestibulospinal drive during human locomotion. Neuroreport 12:2743–2746

Glasauer S, Amorim M-A, Vitte E, Berthoz A (1994) Goal-directed linear locomotion in normal and labyrinthine-defective subjects. Exp Brain Res 98:323–335

Gordon CR, Fletcher WA, Melvill Jones G, Block EW (1995) Adaptive plasticity in the control of locomotor trajectory. Exp Brain Res 102:540–545

Jahn K, Strupp M, Schneider E, Dieterich M, Brandt T (2000) Differential effects of vestibular stimulation on walking and running. Neuroreport 11:1745–1748

Kluzik J, Hlavacka F, Peterka RJ, Horak FB (2002) Vestibular information is important for postural control on inclined and tilting surfaces. Proc of the 3rd SIAMOC Congress Abstr, S213–214

Marlinsky VV (1992) Activity of lateral vestibular nucleus neurons during locomotion in the decerebrate guinea pig. Exp Brain Res 90:583–588

Melvill Jones G, Galiana HL, Weber KD, Fletcher WA, Block EW (2000) Complex podokinetic (PK) response to post-rotational vestibular stimulation. Arch Ital Biol 138:99–105

Mergner T, Rosemeier T (1998) Interaction of vestibular, somatosensory and visual signals for postural control and motion perception under terrestrial and microgravity conditions—a conceptual model. Brain Res Rev 28:118–135

Mergner T, Hlavacka F, Schweigart G (1993) Interaction of vestibular and proprioceptive inputs. J Vestib Res 3:41–57

Nashner LM, Black FO, Wall C 3rd (1982) Adaptation to altered support and visual conditions during stance: patients with vestibular deficits. J Neurosci 2:536–544

Peterka RJ (2002) Sensorimotor integration in human postural control. J Neurophysiol 88:1097–1118

Peterka RJ, Benolken MS (1995) Role of somatosensory and vestibular cues in attenuating visually induced human postural sway. Exp Brain Res 105:101–110

Peterka RJ, Black FO, Schoenhoff MB (1990) Age-related changes in human vestibulo-ocular reflexes: sinusoidal rotation and caloric tests. J Vestib Res 1:49–59

Weber KD, Fletcher WA, Melvill Jones G, Block EW (1996) A new method for measuring podokinetic after-effects without vestibular stimulation. Soc Neurosci Abstr 21:1107

Weber KD, Fletcher WA, Gordon CR, Melvill Jones G, Block EW (1998) Motor learning in the "podokinetic" system and its role in spatial orientation during locomotion. Exp Brain Res 120:377–385

Weber KD, Fletcher WA, Melvill Jones G, Block EW (2002) Podokinetic after-rotation in patients with compensated unilateral vestibular ablation. Exp Brain Res 147:554–557

Wilson VJ, Melvill Jones G (1979) Biophysics of the peripheral end organs. In: Mammalian vestibular physiology. Plenum Press, New York, pp 41–76