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RESEARCH NOTE

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Podokinetic after-rotation in patients with compensated unilateral vestibular ablation

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Abstract Previous studies showed that after stepping-in-place on a rotating turntable, blindfolded subjects cannot step-in-place on firm ground. Instead they involuntarily turn themselves relative to space in the same direction as they were turning relative to the rotating turntable. This phenomenon has been termed podokinetic after-rotation (PKAR). PKAR comprises a brief exponentially rising phase of response during the first 2 min followed by a prolonged second phase of slow exponential decline during the next 28 min. Here we ask whether PKAR is modified in patients with compensated unilateral vestibular loss. Eleven patients who had previous vestibular ablation underwent (1) a Fukuda-like control stepping test, (2) podokinetic adaptation to 30 min of stepping in place on the centre of a turntable rotating at 45 deg/s and (3) PKAR. Control tests showed that the blindfolded patients had no significant rotational bias while stepping-in-place on the ground for 1 min. After 30 min of adaptation, the 2-min rising phase of PKAR was indistinguishable from normal. In contrast, the subsequent 28-min phase of exponential decline showed a lesion-dependent asymmetry. PKAR had significantly higher mean velocities toward the side of the lesion than away from the lesion. The observed PKAR asymmetry may signify occult residual static vestibular imbalance.

Keywords Unilateral vestibular loss · Podokinetic after-rotation · Vestibular compensation

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Introduction

Acute unilateral loss of vestibular function causes signs of static vestibular imbalance, such as spontaneous nystagmus and postural imbalance (Curthoys and Halmagyi 1996; Ito et al. 1995; Katsarkas et al. 1995). These signs gradually disappear over days to weeks but postural responses during locomotion often show incomplete recovery of function (Ito et al. 1995). Recent studies indicate that behavioural recovery following unilateral vestibular loss may occur via parametric changes in the vestibular commissural system (Graham and Dutia 2001) as well as in non-vestibular afferent systems (Fetter and Zee 1988; Cartwright et al. 1999). The present study examines the effect of compensated unilateral vestibular loss on podokinetic adaptation.

Podokinetic adaptation is an experimental paradigm whereby, after prolonged walking in place on the periphery of a rotating treadmill, blindfolded subjects involuntarily walk around a circular path without being aware of the curved trajectory (Gordon et al. 1995a; Jürgens et al. 1999; Earhart et al. 2001a). A similar phenomenon occurs after prolonged stepping in place on the centre of a rotating turntable. When subjects then try to step in place on the stationary turntable they involuntarily rotate themselves relative to space in the same direction as they had been turning relative to the rotating turntable. We term this phenomenon podokinetic after-rotation (PKAR) (Weber et al. 1998; Earhart et al. 2001b).

The normal pattern of PKAR comprises an initial phase of accelerating rotation lasting 1–2 min followed by a phase of exponentially decaying rotation lasting about 28 min. At the end of the 30-min period, there is a steady asymptotic rotation of 4–5 deg/s (Weber et al. 1998).

Materials and methods

Subjects

Eleven subjects (median age 44 years, range 36–59 years) who had complete unilateral vestibular ablation due to translabyrinthine surgical removal of acoustic neuroma were studied after giving informed consent. Seven subjects had right-sided ablation and four had left-sided ablation. The median interval from surgery to testing was 18 months (range 12–60 months). At the time of these experiments none of the patients reported vestibular symptoms and all demonstrated normal visual acuity, eye movement, and sensations of vibration and joint position. All patients had deficient VOR in response to rapid rotational head impulses toward the operated side (Halmagyi and Curthoys 1988). The University of Calgary Office of Medical Bioethics approved the study.

Experimental equipment and procedures

Each subject completed two experimental sessions separated by at least 2 days. The podokinetic system was adaptively modified as described previously (Weber et al. 1998). Briefly, the adaptive stimulus consisted of 30 min of stepping-in-place over the centre of a circular horizontally rotating disc of 76 cm diameter mounted on a servo-driven turntable (Neurokinetics Model 80R). The disc rotated at 45 deg/s. Subjects stepped in the light at a rate of two steps per second by matching their cadence to a metronome affixed to the trunk. Post-adaptive PKAR consisted of 30 min of attempted stepping-in-place on the axis of the stationary disc while blindfolded in the dark. Auditory cues were minimized by means of earplugs. To aid stability and enable measurement of rotation, subjects grasped a low-friction overhead wheel, which incorporated a potentiometer to record angular position of the body relative to space. Each subject was exposed to one direction of adaptive stimulus during the first session and the opposite direction during the second session. Thus, PKAR toward and away from the side of their vestibular loss was recorded.

Exposure to the adaptive stimulus was preceded in each experimental session by a control test adapted from Fukuda (1959). For this test, subjects wore blindfolds and earplugs and stepped in-place on a stationary disc for 1 min while their rotation was recorded.

Data acquisition and analysis

Turntable velocity and body rotational position were recorded at a sampling rate of 200 Hz and then reduced by averaging to 2 Hz. Body rotational velocity was calculated from the slopes between successive positional data points at 0.5-s intervals and correcting for control bias. Control bias (deg/s) for each session was calculated as the change in angular position over the 1-min control test prior to adaptation.

Velocity data from the first 2 min of PKAR (the rising phase) were fitted by least squares regression to a first-order exponential equation rising to a maximum (asymptotic) value, yielding parameters of a rising time constant and maximum velocity. Velocity data from the subsequent 28 min of response decline were fitted with exponential decay curves, yielding parameters of initial velocity, decay time constant and final asymptotic value (Tables 1, 2). Group averages for PKAR toward and away from the side of the lesion were also obtained by binning all subjects' data at 10-s intervals. Each of the resulting data sets was plotted at the midpoint of the interval and fitted with exponential decay curves (e.g. Fig. 1A, B). SigmaPlot7 software was used for plotting and curve fitting and Student's *t*-test was used for statistical analysis. Matlab and Simulink software was used for modeling.

Table 1 Values of rising time constant and maximum velocity of PKAR, calculated from each subject's best-fit exponential curves during the first (2-min) phase of rising velocities in the ipsilateral and contralateral directions

	Rising time constant (s)		Max vel. (deg/s)	
Patient	Ipsilateral	Contralateral	Ipsilateral	Contralateral
1	27.0	24.0	20.5	19.3
2	13.0	16.6	22.2	17.9
3	4.7	24.4	11.3	10.9
4	9.7	11.2	31.3	31.3
5	6.1	10.0	30.4	22.1
6	16.3	6.8	19.2	23.0
7	8.6	28.2	17.7	15.7
8	19.1	8.8	17.3	17.9
9	5.0	15.9	17.0	17.8
10	5.2	12.5	18.2	17.9
11	7.3	16.7	14.4	19.1
Mean	11.1	15.9	19.9	19.4
SE	2.2	2.1	1.9	1.5
P value	0.14		0.59	

Results

All subjects successfully completed two sessions (one for each direction) comprising a control stepping test followed by 30 min of adaptation and 30 min of podokinetic after-rotation (PKAR).

The control stepping tests revealed no significant directional bias in the subject population. Five of the 11 subjects rotated toward the side of the lesion, two subjects rotated away from the side of the lesion, and four subjects rotated in different directions in the two control stepping tests. The range of average velocities was 0.03–2.7 deg/s and the median was 0.18 deg/s.

Table 1 shows the values of rising time constant and maximum velocity of PKAR, calculated from each subject's best-fit exponential curves during the first (2-min) phase of rising velocities in the ipsilateral and contralateral directions. Statistical analysis revealed no significant differences between the two data sets. Figure 1A illustrates the averaged group responses (± SE) over the first 2-min rise of the PKAR response. Continuous lines show the best-fit exponential curves for the two data sets, neither of which differs significantly from data of normal subjects (Weber et al. 1996).

Table 2 shows the values of initial velocity, decay time constant and asymptotic velocity of PKAR, calculated from each subject's best-fit exponential curves during the next 28-min phase of decreasing velocities in the two directions. The estimates of initial velocity in the first column give the values of the calculated exponential curves at commencement of the second phase of response (2 min after the onset of stepping on the stationary turntable). The time constants of the two sets were indistinguishable from one another ($P>0.1$). The initial velocities and the final asymptotes were significantly greater for PKAR in the ipsilateral direction [mean initial velocities (± SE): 18.9±1.8 vs 16.1±1.6 deg/s, $P<0.05$;

Table 2 Estimates of initial velocity give values of the fitted decaying exponential curves at commencement of the second phase of response (i.e., 2 min after the onset of PKAR)

Patient	Initial vel. (deg/s)		Decay time const. (min)		Asymptote (deg/s)	
	Ipsi.	Contra.	Ipsi.	Contra.	Ipsi.	Contra.
1	18.1	12.1	7.7	10.0	7.0	0.4
2	18.1	15.5	8.3	11.1	4.6	3.0
3	10.8	8.5	6.3	9.1	2.8	1.7
4	29.0	26.2	11.1	7.7	8.6	4.4
5	30.8	20.4	10.0	6.3	13.0	1.6
6	18.1	23.4	33.3	14.3	5.7	3.7
7	18.6	14.7	3.3	7.1	2.1	0.4
8	15.9	14.3	33.3	12.5	3.0	2.5
9	20.1	16.0	8.3	7.1	8.2	3.1
10	15.1	12.8	5.0	5.0	4.0	2.1
11	12.8	12.9	10.0	6.7	3.0	3.0
Mean	18.9	16.1	12.4	8.8	5.6	2.4
SE	1.8	1.6	3.2	0.9	1.0	0.4
P value	0.04		0.19		0.009	

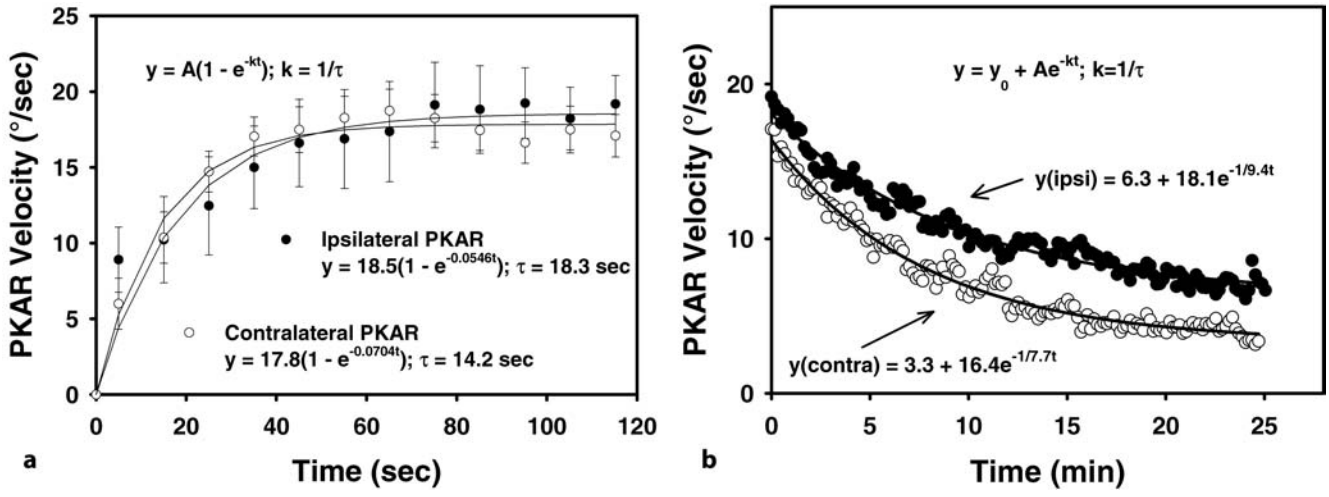

Fig. 1 **A** Plot of PKAR velocity vs time during the initial phase (initial 2 min) of ipsilateral (*closed circles*) and contralateral (*open circles*) PKAR. Data points represent group average velocities (± SE) binned at 10-s intervals. The points are fitted with first-order exponentially rising curves to reach maximum asymptotic values. **B** Plot of PKAR velocity vs time during the last 28 min of ipsilateral (*closed circles*) and contralateral (*open circles*) PKAR. Data points represent group average velocities (± SE) binned at 10-s intervals. The points are fitted with first-order exponential decay curves, which reach a positive asymptote

mean asymptotes (± SE): 5.6±1.0 vs 2.4±0.4 deg/s, $P<0.01$].

Figure 1B shows the corresponding averaged group responses. Over the whole data set all ipsilateral plotted points (filled circles) lie above corresponding contralateral points. Nine of the 11 subjects had initial velocities that were greater in the ipsilateral direction and 10 had greater ipsilateral asymptotic velocities (Table 2).

Discussion

Previous studies of podokinetic after-rotation (PKAR) in normal subjects revealed a robust unidirectional locomotor after-response following exposure to a prolonged unidirectional podokinetic stimulus (Gordon et al. 1995a; Earhart et al. 2001a, 2001b; Weber et al. 1998; Jürgens et al. 1999). PKAR typically accelerates over the first 2 min to reach a maximum velocity of about one-third of the velocity of the adapting stimulus, followed over the next 28 min by a subsequent slow exponential decay of response velocity toward a positive asymptotic value.

In this study, patients with compensated unilateral vestibular loss performed in a generally similar way to normal subjects. The Fukuda-like control stepping test yielded patterns of rotation that were not significantly different from the normal results of Gordon et al. (1995b), including the absence of any significant directional bias. During the initial acceleratory phase of the PKAR response, neither the rising time constants nor maximum velocities (Table 1) were significantly different from corresponding values obtained from previous studies on normal subjects (Weber et al. 1996, 1998). There were also no significant differences during this phase in the rising time constants or maximum velocities of PKAR directed ipsi- and contralateral to the side of the lesion (Fig. 1A).

An interesting implication of the apparent normality of the rising response phase stems from the fact that the blindfolded subjects were instructed to "step in place without turning" on the stationary disc. Consequently, the actual rate of rise probably depended on a systematic interaction of the tendency to turn (PKAR) and its conscious suppression due to suprathreshold vestibular sensation during rotation (Weber et al. 1998). Simulation of the likely profile of vestibular stimulation throughout PKAR, based on a vestibular canal signal acting as a "leaky" integrator with time constant in the range of 5–15 s, indicates that there would be vestibular stimulation above the presumed sensory threshold of 2 deg/s during the initial rising phase of PKAR. This inference is also supported by earlier data showing that the rise-time in normal subjects is substantially shortened when vestibular stimulation is nullified by servo-stabilizing the body during PKAR (Weber et al. 1996). The normality of results in the initial rising phase of PKAR (Table 1, Fig. 1A) may therefore stem from the likely normality of a weak *dynamic* vestibular influence at these low velocities, in line with previous reports of approximately balanced vestibulo-ocular responses at low velocities in fully compensated human patients (Katsarkas et al. 1995; Paige 1989).

In contrast there can be no relevant vestibular controlling factor during the subsequent slowly decaying phase of response due to the wide separation between time constants of the podokinetic (~600 s; Table 2, Fig. 1B) and vestibular (equal to or less than ~15 s; Dai et al. 1999) system. For the vestibular system it is as though the declining phase of PKAR is of zero angular velocity relative to space. Presumably therefore the observed ipsilateral bias of this phase reflects a *static* imbalance in the vestibulospinal system, a feature which is not directionally significant in the weak or absent dark-tested spontaneous ocular nystagmus of the fully compensated patient (Katsarkas et al. 1995; Paige 1989).

Why should PKAR reveal a static vestibulospinal imbalance whereas the Fukuda like control tests did not? Perhaps, as during PKAR, it takes longer than 1 min for the podokinetic system to manifest a weak static imbalance. Alternatively there may have been some form of adaptive alteration in vestibulospinal control of the podokinetic system itself, although it is hard to rationalize the observed bias in terms of a behavioral advantage during locomotion of the patient. Whatever the mechanism, the results suggest that PKAR may reveal an otherwise occult static imbalance in the vestibulospinal system of the compensated patient with unilateral vestibular ablation.

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References

Cartwright AD, Curthoys IS, Gilchrist DP (1999) Testable predictions from realistic neural network simulations of vestibular compensation: integrating the behavioural and physiological data. Biol Cybern 81:73–87

Curthoys IS, Halmagyi GM (1996) How does the brain compensate for vestibular lesions? In: Baloh RW, Halmagyi GM (eds) Disorders of the vestibular system. Oxford University Press, New York, pp 145–154

Dai M, Cohen B, Raphan T (1999) Model-based study of the human cupular time constant. J Vestib Res 9:293–301

Earhart GM, Melvill Jones G, Horak FB, Block EW, Weber KD, Fletcher WA (2001a) Forward vs. backward walking: transfer of podokinetic adaptation. J Neurophysiol 86:1666–1670

Earhart GM, Melvill Jones G, Horak FB, Block EW, Weber KD, Fletcher WA (2001b) Podokinetic after-rotation following unilateral and bilateral podokinetic stimulation. J Neurophysiol 87:1138–1141

Fetter M, Zee DS (1988) Recovery from unilateral labyrinthectomy in rhesus monkey. J Neurophysiol 59:370–393

Fukuda T (1959) The stepping test. Two phases of the labyrinthine reflex. Acta Otolaryngol (Stockh) 50:95–108

Gordon CR, Fletcher WA, Melvill Jones G, Block EW (1995a) Adaptive plasticity in the control of locomotor trajectory. Exp Brain Res 102:540–554

Gordon CR, Fletcher WA, Melvill Jones G, Block EW (1995b) Is the stepping test a specific indicator of vestibulo-spinal function? Neurology 45:2035–2037

Graham BP, Dutia MB (2001) Cellular basis of vestibular compensation: analysis and modelling of the role of the commissural inhibitory system. Exp Brain Res 137:387–396

Halmagyi GM, Curthoys IS (1988) A clinical sign of canal paresis. Arch Neurol 45:737–739

Ito S, Odahara S, Hiraki M, Idate M (1995) Evaluation of imbalance of the vestibulo-spinal reflex by "the circular walking test". Acta Otolaryngol Suppl (Stockh) 19:124–126

Jürgens R, Bob T, Becker W (1999) Podokinetic after-rotation does not depend on sensory conflict. Exp Brain Res 128:563–567

Katsarkas A, Galiana HL, Smith HL (1995) Vestibulo-ocular reflex (VOR) biases in normal subjects and patients with compensated vestibular loss. Acta Otolaryngol (Stockh) 115:476–483

Paige GD (1989) Nonlinearity and asymmetry in the human vestibulo-ocular reflex. Acta Otolaryngol 108:1–8

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