

## Comparison of head- and body-velocity trajectories during locomotion among healthy and vestibulopathic subjects

James T. Cavanaugh, PT, PhD, NCS;<sup>1-2\*</sup> Dov Goldvasser, MSc, ME;<sup>3</sup> Chris A. McGibbon, PhD;<sup>4-5</sup>  
David E. Krebs, DPT, PhD<sup>3,6-8</sup>

<sup>1</sup>Geriatric Research, Education and Clinical Center, Department of Veterans Affairs Medical Center, Durham, NC;

<sup>2</sup>Department of Physical and Occupational Therapy, Duke University Medical Center, Durham, NC; <sup>3</sup>Biomotion Laboratory, Massachusetts General Hospital (MGH), Boston, MA; <sup>4</sup>Department of Kinesiology, University of New Brunswick, Fredericton, New Brunswick, Canada; <sup>5</sup>Institute of Biomedical Engineering, University of New Brunswick, Fredericton, New Brunswick, Canada; <sup>6</sup>MGH Institute of Health Professions, Boston, MA; <sup>7</sup>Department of Mechanical Engineering, Massachusetts Institute of Technology, Cambridge, MA; <sup>8</sup>Department of Orthopaedics, Harvard Medical School, Boston, MA

**Abstract**—The optimal strategies for improving locomotor stability in people with vestibulopathy remain unclear. To help identify likely targets for intervention, we sought to determine whether vestibulopathic postural control impairment during locomotor activity was more localized to either the head or the whole body. We used high curvature analysis (HCA) to quantify the smoothness of head- and body-velocity trajectories during repeated stepping in 18 vestibulopathic and 17 healthy subjects. We employed a mixed-model repeated measures analysis of variance to compare differences in head- and body-trajectory HCA scores. Pearson coefficients were used to describe relationships between head- and body-trajectory HCA scores within each group. The results revealed that neither head- nor body-velocity trajectories were relatively more impaired in subjects with vestibulopathy. Importantly, however, the smoothness of head and body trajectories was more strongly related in subjects with vestibulopathy compared with healthy subjects, suggesting that the fundamental motor control impairment produced by vestibulopathy may be related to an abnormal coupling of head and body motion. We discuss implications for locomotor training in patients with vestibulopathy.

**Key words:** bilateral vestibular hypofunction, equilibrium, high curvature analysis, locomotion, locomotor control, measurement, postural control, rehabilitation, unilateral vestibular hypofunction, vestibulocollic, vestibulopathy.

## INTRODUCTION

Gait deviations and complaints of disequilibrium are common findings in vestibulopathic subjects [1–3], and fall risk increases with the severity of vestibular loss [4].

**Abbreviations:** aVCR = angular vestibulocollic reflex, BVH = bilateral vestibular hypofunction, CG = center of gravity, HCA = high curvature analysis, IVCR = linear vestibulocollic reflex, MGH = Massachusetts General Hospital, SCC = semi-circular canal, SD = standard deviation, SVAR = sinusoidal vertical axis rotation, 3-D = three-dimensional, UVH = unilateral vestibular hypofunction, VOR = vestibular ocular reflex, VR = vestibular rehabilitation.

**This material was based on work supported in part by a promotion of doctoral studies scholarship awarded to Dr. Cavanaugh by the Foundation for Physical Therapy, Inc., and by the National Institutes of Health (R01AG11255 and R21AT00553).**

\*Address all correspondence to J. T. Cavanaugh, Department of Veterans Affairs Medical Center, GRECC (182), 508 Fulton St., Durham, NC 27705; 919-286-6932; fax: 919-286-6823. Email: jimcavanaugh@nc.rr.com

DOI: 10.1682/JRRD.2004.01.0005

Vestibular rehabilitation (VR), a well-described form of exercise intervention, is known to improve postural stability in patients with vestibular disorders [5–6] and may help to reduce fall risk [7]. VR, however, does not necessarily improve steadiness during locomotion in all patients [8]. The limited reports of VR outcomes highlight the lack of information available to clinicians regarding the most effective therapeutic strategies for improving locomotor control.

Vestibular loss results in abnormal movement synergies for head and trunk control, suggesting a top-down influence of the vestibulospinal system on postural control [9]. Whether therapeutic gait and balance training should be directed toward improving control of the head, trunk, or whole body remains unclear. Wall et al., for example, attempted to improve postural control by directing exercise and neuroprosthetic interventions toward the trunk with mixed results [10]. Patten et al. described improvements in head-trunk coordination patterns after VR but did not determine whether greater coordination resulted from improved trunk control, head control, or both [11]. As a necessary step toward improving the efficacy of VR, we sought to compare the degree of impairment in head and body control in subjects with vestibulopathy. In principle, more profound impairments make more likely targets for exercise intervention.

Quantifying impaired locomotor control presents two distinct challenges. First, locomotion over a walkway or on a treadmill in a gait laboratory typically does not provide a sufficient challenge to the stability of vestibulopathic subjects [12]. Indeed, if gait pace or speed is controlled, vestibular subjects evince few kinematic abnormalities compared with healthy controls [11,13]. Second, the amount of data that can be recorded during challenging yet relatively discrete dynamic balance tasks is often insufficient to allow aggregate dynamic analysis [14]. In response to these challenges, we determined that a repeated bench-stepping paradigm (step up forward/down backward) provided a sufficient challenge to subjects' balance while enabling multiple cycles of kinematic information to be acquired during a single trial [14–17].

Defining what constitutes “locomotor control” is an important theoretical issue. Some researchers have defined control in biomechanical terms as the amount of variability in head and trunk displacements [18–19]. In this framework, larger center of gravity (CG) translations or angular deviations are thought to reflect a lack of preci-

sion in movement control. Other investigators have defined locomotor control in terms of the temporal coordination of head and trunk segments [11]. We recently determined that high curvature analysis (HCA), a method adopted from image analysis, is a relatively simple yet useful tool for quantifying the smoothness, i.e., stability, of movement trajectories generated over multiple cycles [14,20]. HCA was originally designed to detect sharp corners, such as structure edges, within an image [21]. One can successively identify sharp angles subtended by discrete triplets of points within a given curve using the cosine law. We believe that adapting the procedure to identify the number of dramatic changes (sharp angles) in movement trajectories makes HCA useful for distinguishing between the smooth, sinusoidal displacements of normal individuals and the more irregular movement patterns of individuals with vestibulopathy. Indeed, we have previously determined that HCA distinguished between the whole-body CG velocity trajectories of healthy and vestibulopathic subjects during a repeated stepping task and was a sensitive measure of improvements in body-CG dynamic control following rehabilitation [14].

This investigation compared head- and body-movement trajectory smoothness in subjects with vestibulopathy during repeated stepping. We expanded upon previous research [14] by including multiplanar body-CG velocity trajectory analysis and by including subjects with both bilateral and unilateral vestibulopathy. To provide a frame of reference, we also compared head and body trajectories in a group of healthy subjects. Based on the work of other investigators [22–24], we used velocity trajectories of head CG motion and head angular motion to indicate head control during whole-body movement.

## METHODS

### Subjects

We recruited individuals with vestibulopathy seeking treatment for unsteady locomotion at Massachusetts General Hospital (MGH) and from the surrounding community in Boston. We analyzed head and body data from 7 subjects with bilateral vestibular hypofunction (BVH) (4 male, 3 female, average age  $67.5 \pm 8.7$ , range 56.5–78.5), 11 subjects with unilateral vestibular hypofunction (UVH) (3 male, 8 female, average age  $50.1 \pm 19.2$ , range 20.3–72.9), and 17 healthy comparison subjects (3 male, 14 female, average age  $35.4 \pm 17.9$ , range 20.2–74.5). No

subject had uncorrected visual impairments or musculoskeletal disorders that might have affected balance or gait; all subjects walked without assistive devices and were able to negotiate a 7.6 cm step without assistance. Healthy subjects had no history or clinical evidence of vestibulopathy or other nervous system disorders upon physical examination. Subjects with vestibulopathy had no other disorders of the central or peripheral nervous system. Vestibulopathy was classified as BVH based on bilaterally reduced caloric responses and vestibular ocular reflex (VOR) gains  $>2.5$  standard deviations (SDs) below normal during whole-body sinusoidal vertical axis rotation (SVAR) tests at a frequency range of 0.01 to 1.0 Hz. Subjects with UVH had  $>30$  percent unilateral abnormality as determined by either reduced caloric response to cool (27 °C) and warm (44 °C) water stimulation or abnormal symmetry or phase leads during SVAR testing. Among the subjects with vestibulopathy, etiologies that produced impairment included aminoglycoside ototoxicity, acoustic neuroma, vestibular labyrinthitis or neuritis, ear surgery, and trauma. For most BVH subjects, the vestibulopathy etiology was idiopathic. MGH's Institutional Review Board approved the study protocol, and all subjects gave their informed, signed consent in accordance with institutional policy on human research.

### Instrumentation

We collected bilateral three-dimensional (3-D) kinematic data using a SELSPOT II optoelectric system (Selective Electronics, Partille, Sweden). Light-emitting diode arrays were secured to 11 body segments: head, trunk, pelvis, thighs, shanks, feet, and upper arms. We sampled data at 150 Hz and digitally filtered the results using a low-pass (6 Hz) Butterworth filter. The 3-D global position and rotation of each segment array was calculated with TRACK<sup>®</sup> software (Massachusetts Institute of Technology, Cambridge, MA). We calculated segment masses and CG locations using individually scaled anthropometric data [25]. We used these data, combined with the measured kinematics, to calculate the location of the whole-body CG in space [26].

### Procedures

#### *Experimental Protocol*

Subjects were positioned in the center of the 2 m  $\times$  2 m  $\times$  2 m viewing volume, directly in front of a step measuring 29.4 cm wide  $\times$  22.9 cm deep  $\times$  7.6 cm high.

We set a metronome at 120 b/min, and instructed subjects to step repeatedly on and off the step (right foot up, left foot up, right foot down, left foot down) to the beat of the metronome with arms swinging naturally. Data collection began after subjects had reached a steady state. Because of changes in the experimental protocol made during the data collection period, trial duration ranged from 10 to 30 s; no significant differences between durations were found. Five subjects with BVH (62.5%) and four subjects with UVH (41.7%) were unable to step at 120 steps/min without loss of balance. Consequently, to provide a sufficient but not excessive challenge, we tested these subjects at 100 steps/min. The slower-stepping subjects were not demographically different from the subjects who stepped at 120 steps/min, nor were other differences found between the two groups, including symptom severity or use of a different biomechanical strategy. Two trials were recorded for analysis.

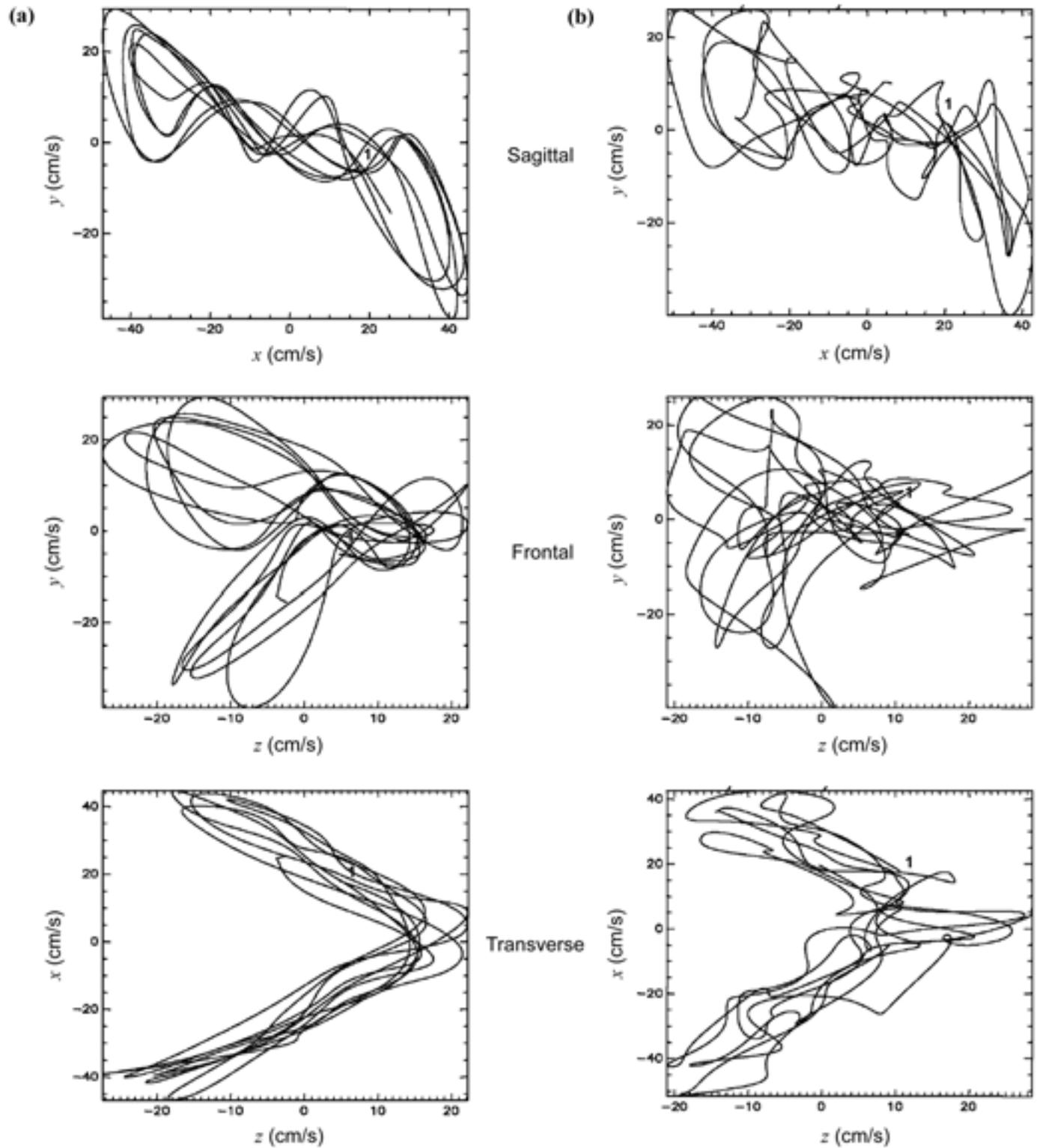
#### *High Curvature Analysis*

We analyzed four sets of 3-D kinematic data: head-CG translations, whole-body CG translations, head-CG relative to body-CG translations, and head rotations. For CG translations, we created two-dimensional (2-D) linear velocity trajectory plots in the sagittal, transverse, and frontal planes (**Figure**). Using the head-rotation data, we created 2-D angular velocity trajectories by plotting pitch versus yaw, pitch versus roll, and roll versus yaw. Thus, for each subject, a total of 12 velocity trajectories (4 kinematic sets  $\times$  3 velocity trajectory plots per set) were available from each trial.

The HCA procedure has been detailed elsewhere [14,20–21]. Briefly, for each velocity trajectory, we used the cosine law to calculate the central angle in successive triplets of points. Sharp curves were defined as central angles of less than 100°. We normalized the number of sharp curves in a velocity trajectory to the number of complete stepping cycles to produce a single HCA score. Higher HCA values indicated more irregular velocity trajectories consistent with reduced dynamic movement control.

#### *Data Analysis*

We analyzed our data with Statistical Package for the Social Sciences (SPSS) software version 9.0 (SPSS Inc., Chicago, IL). We pooled data within each group and calculated a group mean HCA score for each kinematic set using the trial average scores from each trajectory plot.

**Figure.**

Representative whole-body center of gravity velocity trajectories for (a) healthy and (b) vestibulopathic (bilateral vestibular hypofunction) subjects; the latter have more sudden changes (high curvatures) than healthy trajectories ( $x$  = anteroposterior axis,  $y$  = vertical axis, and  $z$  = mediolateral axis).

We used a  $3 \times 4$  (group  $\times$  kinematic set) mixed model analysis of variance with repeated measures on the second factor to compare group mean HCA difference scores from head- and body-velocity trajectories ( $\alpha = 0.05$ ). We were specifically interested in identifying the nature of any differences between head trajectory HCA scores and the body-CG trajectory HCA score in subjects with vestibulopathy compared to healthy subjects. To avoid violations of the sphericity assumption, we relied on the more conservative Greenhouse-Geisser  $F$ -test. We used Pearson product-moment correlation coefficients to determine the strength of the relationship between head and body HCA scores within each group.

## RESULTS

Head- and body-CG velocity trajectories were generally more smooth (lower HCA scores) in healthy subjects and subjects with UVH (Table). Scores for head angular velocity trajectories were markedly larger than head- and body-CG trajectories for all subjects ( $F_{2,2, 71.1} = 191.9$ ,  $p < 0.001$ ). No significant interaction was found, however, between group and kinematic set, suggesting that the relative difference in the smoothness of head- and body-CG velocity trajectories was similar among subjects with vestibulopathy and healthy subjects.

For the CG translations, we calculated correlations among HCA scores for the head and body within each plane of analysis. The strength and direction of the relationship between head and whole-body HCA scores depended on group membership. For the CG translations of healthy subjects, little or no association was found between head and body HCA scores in either the sagittal, transverse, or frontal planes ( $-0.16 < r < 0.49$ ). For subjects with UVH, moderate to strong positive relationships were found between HCA scores for head-CG translation

relative to the trunk- and body-CG translation in the transverse ( $r = 0.93$ ) and frontal ( $r = 0.64$ ) planes, as well as for head- and body-CG translations in the sagittal plane ( $r = 0.74$ ). For subjects with BVH, moderate to strong positive relationships between head and body HCA scores were found for all CG comparisons ( $0.73 < r < 0.94$ ).

Because of the incongruities inherent in precisely matching specific head-rotation planes of analysis with body-CG translations, we elected to calculate correlations among all head-rotation and body-CG translation HCA scores. For healthy subjects and those with UVH, little or no association was found between the smoothness of velocity trajectories for angular head motion and whole-body CG motion. For subjects with BVH, however, the smoothness of angular head velocity trajectories and whole-body CG trajectories was consistently and strongly related ( $0.86 < r < 0.95$ ).

## DISCUSSION

This study describes relative differences in head and body control in subjects with vestibulopathy performing a locomotor activity. Our primary goal was to provide a theoretically defensible basis for targeting exercise interventions in this population. The results revealed that neither head- nor body-motion control, when measured in terms of movement irregularity, was relatively more impaired in subjects with vestibulopathy. The finding may explain why previous investigations that have examined the effect of intervention directed exclusively at the trunk were inconclusive [10].

Our data also revealed that in the presence of vestibulopathy, control of head and body segments is strongly linked. In particular, subjects with BVH who had more irregular head-velocity trajectories were more likely to have unstable whole-body velocity trajectories. These data

### Table.

Group mean high curvature analysis scores ( $\pm$  standard deviation [SD]) of velocity trajectories for body center of gravity (CG), head CG, head-body CG, and head angular. Group size and grand mean (standard error [SE]) scores are also indicated.

Group	Body CG (Mean $\pm$ SD)	Head CG (Mean $\pm$ SD)	Head-Body CG (Mean $\pm$ SD)	Head Angular Velocity (Mean $\pm$ SD)
Healthy ( $n = 17$ )	$2.96 \pm 1.60$	$2.40 \pm 0.48$	$2.05 \pm 0.38$	$8.52 \pm 1.65$
UVH ( $n = 11$ )	$3.08 \pm 1.72$	$3.18 \pm 1.33$	$2.52 \pm 0.92$	$8.89 \pm 1.89$
BVH ( $n = 7$ )	$4.89 \pm 4.48$	$3.57 \pm 1.46$	$3.30 \pm 2.07$	$12.14 \pm 4.60$
Grand Mean (SE)	$3.64 \pm 0.44$	$3.05 \pm 0.19$	$2.62 \pm 0.19$	$9.85 \pm 0.46$

BVH = bilateral vestibular hypofunction and UVH = unilateral vestibular hypofunction.

provide indirect support for the idea that subjects with BVH demonstrate impaired top-down vestibulospinal control of body motion during tasks in which the upper body must be decoupled from the motion of the legs [9]. The absence of head-trunk coupling among healthy subjects implies that normal postural control mechanisms do not require head-motion stabilization to produce smooth body-movement trajectories during locomotion. From a systems motor control perspective, such a hypothesis is consistent with the tenet that healthy, developmentally mature physiologic states are associated with a greater number of control-system degrees of freedom that can be independently regulated [27–30].

Subjects with BVH had generally higher HCA scores than healthy or UVH subjects. Importantly, subjects with BVH also were older and consequently may have had other subtle comorbidities or age-related physiologic decline that affected balance control [31]. Nonetheless, the presence of profound vestibular loss was likely to have influenced angular head-movement and body control. Angular head-movement control during whole-body movement has been implicated in the control of gaze direction [22,32–35], locomotor navigation [36], postural control during whole-body function movements [37], and voluntary reaching activities [38]. Angular head-movement control during locomotor activity may allow the vestibular organs to serve as an inertial guidance system that contributes to an internal frame of reference necessary for postural orientation [35,39]. Among subjects with BVH, both semicircular canal (SCC) and otolith dysfunction may have contributed to impaired head-movement control. In this study, we did not analyze coordination patterns of head-trunk rotation or head translation/head rotation as has been done previously [32–34]; consequently, we did not directly assess the contributions of each angular vestibulocollic reflex (aVCR) and linear vestibulocollic reflex (IVCR) to head control. The BVH subjects, however, all had profound angular VOR deficiencies on SVAR testing, indicating the presence of significant SCC dysfunction. As a result, head-stabilizing aVCR mechanisms were probably impaired. In addition, we believe that the 3-D head translations, which dominated the stepping task and were performed at 2 steps/min, served as a sufficient challenge to head- and postural-control mechanisms mediated by the otolith system. We suggest, therefore, that IVCR dysfunction may also have contributed to the relatively large amount of head and whole-body dyscontrol in BVH subjects.

Not to be confused with traditional biomechanical analyses, HCA is fundamentally a graphical analysis technique useful for understanding the temporal dynamics of human movement. The HCA scores of body-CG velocity trajectories generally were consistent with our previous data, which demonstrated significant whole-body dyscontrol in vestibulopathic subjects prior to VR [14]. For HCA to be useful for identifying movement impairment, subjects must be engaged in a focused and vigorous physical activity that presents a sufficient challenge to postural control. In our study, trials were well tolerated by all participants. Some subjects, however, lost their balance during testing (an indication of sufficient challenge) and were unable to complete trials stepping at the prescribed rate of 120 steps/min. The slower rate (100 steps/min) allowed inclusion of these subjects, but did not affect our results, because we were primarily interested in within-subject relationships between head and body control.

These data suggest that effective gait-training strategies in this population may depend on a patient's potential for eventually decoupling the control of head and body segments. Individuals with less severe vestibulopathy may be more likely to achieve this goal. Accordingly, gait training should include multitask scenarios in which the individual is required to maintain control of postural stability during locomotion while performing secondary tasks involving head orientations or gaze directions that are incongruent with the path of navigation. Examples include walking (forward, backward, or to the side) while performing a visual search of the environment, walking forward while looking over one's shoulder, stepping backward while sweeping a floor, and descending stairs while looking upward. Individuals with more severe vestibulopathy (e.g., acute unilateral hypofunction or complete bilateral hypofunction) might be trained to stabilize head and body segments through a variety of compensatory strategies that allow for coupled motion of the head and trunk. Although such strategies might occur spontaneously during attempts to cope with severe disequilibrium, some individuals require specific training. For subjects with BVH, techniques that promote angular head-motion stabilization, either through voluntary or cervicocollic control mechanisms [40], hypothetically would improve locomotor control.

Our study was limited in several respects. First, ours was a convenience sample and consequently may not have represented the population of individuals with vestibulopathy. Second, although previous reports support the

top-down influence of the vestibulospinal system on postural control [9,39,41], the strong correlations between head- and body-motion smoothness among vestibulopathic subjects in our study do not imply causality. Third, age dissimilarities and unequal sample sizes among the groups limited our ability to draw conclusions regarding the severity of vestibular loss and locomotor control.

## CONCLUSION

In conclusion, our findings suggest that head- and whole-body motion control appear to be similarly impaired by vestibulopathy. Neither the head nor the trunk, therefore, make a more likely exclusive target for gait-training intervention designed to resolve signs and symptoms of disequilibrium. The data also support the idea that head and body motion during walking may be abnormally coupled, especially for individuals with more severe impairment. We recommend that optimal gait-training strategies incorporate a patient's potential for restoring a healthy decoupling of head and body control during locomotion.

## ACKNOWLEDGMENTS

We thank Donna M. Scarborough, MS, PT, and Lara Asmundson, MS, PT, for help in acquiring data.

## REFERENCES

- Gill-Body KM, Krebs DE. Locomotor stability problems associated with vestibulopathy: assessment and treatment. *Phys Ther Pract.* 1994;3(4):232–45.
- Borello-France DF, Whitney SL, Herdman SJ. Assessment of vestibular hypofunction. In: Herdman SJ, editor. *Vestibular rehabilitation.* Philadelphia (PA): F.A. Davis; 1994. p. 247–86.
- Krebs DE, Gill-Body KM, Riley PO, Parker SW. Double-blind, placebo-controlled trial of rehabilitation for bilateral vestibular hypofunction: preliminary report. *Otolaryngol Head Neck Surg.* 1993;109(4):735–41.
- Herdman SJ, Blatt P, Schubert MC, Tusa RJ. Falls in patients with vestibular deficits. *Am J Otol.* 2000;21(6):847–51.
- Herdman SJ. Exercise strategies for vestibular disorders. *Ear Nose Throat J.* 1989;68:961–64.
- Horak FB, Jones-Rycewicz C, Black FO, Shumway-Cook A. Effects of vestibular rehabilitation on dizziness and imbalance. *Otolaryngol Head Neck Surg.* 1992;106(2):175–80.
- Herdman SJ, Schubert MC, Tusa RJ. Strategies for balance rehabilitation: fall risk and treatment. *Ann N Y Acad Sci.* 2001;942:394–412.
- Krebs DE, Gill-Body KM, Parker SW, Ramirez JV, Wernick-Robinson M. Vestibular rehabilitation: useful but not universally so. *Otolaryngol Head Neck Surg.* 2003;128(2):240–50.
- Horak FB, Buchanan J, Creath R, Jeka J. Vestibulospinal control of posture. *Adv Exp Med Biol.* 2002;508:139–45.
- Wall C 3rd, Weinberg MS, Schmidt PB, Krebs DE. Balance prosthesis based on micromechanical sensors using vibrotactile feedback of tilt. *IEEE Trans Biomed Eng.* 2001;48(10):1153–61.
- Patten C, Horak FB, Krebs DE. Head and body center of gravity control strategies: adaptations following vestibular rehabilitation. *Acta Otolaryngol.* 2003;123(1):32–40.
- McDonald PV, Basdogan C, Bloomberg JJ, Layne CS. Lower limb kinematics during treadmill walking after space flight: implications for gaze stabilization. *Exp Brain Res.* 1996;112(2):325–34.
- Tucker CA, Ramirez JV, Krebs DE, Riley PO. Center of gravity dynamic stability in normal and vestibulopathic gait. *Gait Posture.* 1998;8(2):117–23.
- Goldvasser D, McGibbon CA, Krebs DE. Vestibular rehabilitation outcomes: velocity trajectory analysis of repeated bench stepping. *Clin Neurophysiol.* 2000;111(10): 1838–42.
- Hudson CC, Krebs DE. Frontal plane dynamic stability and coordination in subjects with cerebellar degeneration. *Exp Brain Res.* 2000;132(1):103–13.
- Krebs DE, Goldvasser D, Lockert JD, Portney LG, Gill-Body KM. Is base of support greater in unsteady gait? *Phys Ther.* 2002;82(2):138–47.
- McPartland DD, Krebs DE, Wall C. Quantifying ataxia: ideal trajectory analysis—a technical note. *J Rehabil Res Dev.* 2000;37(4):445–54.
- Mamoto Y, Yamamoto K, Imai T, Tamura M, Kubo T. Three-dimensional analysis of human locomotion in normal subjects and patients with vestibular deficiency. *Acta Otolaryngol.* 2002;122(5):495–500.
- Yamamoto K, Mamoto Y, Imai T, Hirasaki E, Kubo T. Studies of locomotor patterns in subjects with vestibular dysfunction using 3D analysis video system. In: Claussen CF, Haid CT, Hofferberth B, editors. *Equilibrium in research and equilibrium in modern treatment.* Amsterdam (NL): Elsevier; 1999. p. 261–68.
- Goldvasser D, McGibbon CA, Krebs DE. High curvature and jerk analyses of arm ataxia. *Biol Cybern.* 2001;84(2): 85–90.

21. Chetverikov D, Szabo Z. A simple and efficient algorithm for detection of high curvature points in planar curves. In: 23rd workshop of the Austrian Pattern Recognition Group; 1999. p. 175–84.
22. Pozzo T, Berthoz A, Lefort L, Vitte E. Head stabilization during various locomotor tasks in humans. II. Patients with bilateral peripheral vestibular deficits. *Exp Brain Res.* 1991; 85(1):208–17.
23. Reschke MF, Bloomberg JJ, Harm DL, Paloski WH. Space flight and neurovestibular adaptation. *J Clin Pharmacol.* 1994;34(6):609–17.
24. Holt KG, Ratcliffe R, Jeng SF. Head stability in walking in children with cerebral palsy and in children and adults without neurological impairment. *Phys Ther.* 1999;79(12): 1153–62.
25. McConville J, Churchill TD, Kaleps I, Clauser CE, Cuzzi J. Anthropometric relationship of body segment moments of inertia. Wright-Patterson Air Force Base (OH): Air Force Aerospace Medical Research Laboratory; 1980. Report No. AFAMRL-TR-80-119.
26. Riley PO, Mann RW, Hodge WA. Modelling of the biomechanics of posture and balance. *J Biomech.* 1990;23(5): 503–6.
27. Goldberger AL. Fractal variability versus pathologic periodicity: complexity loss and stereotypy in disease. *Perspect Biol Med.* 1997;40(4):543–61.
28. Lipsitz LA, Goldberger AL. Loss of ‘complexity’ and aging. Potential applications of fractals and chaos theory to senescence. *JAMA.* 1992;267(13):1806–9.
29. Newell K. Degrees of freedom and the development of postural center of pressure profiles. In: Newell KM MP, editor. *Applications of non-linear dynamics to developmental process modeling.* Mahwah (NJ): Lawrence Erlbaum Associates; 1998. p. 80–81.
30. Creath R, Kiemel T, Horak F, Jeka JJ. Limited control strategies with the loss of vestibular function. *Exp Brain Res.* 2002;145(3):323–33.
31. Dietz V, Baaken B, Colombo G. Proprioceptive input overrides vestibulospinal drive during human locomotion. *Neuroreport.* 2001;12(12):2743–46.
32. Bloomberg JJ, Peters BT, Smith SL, Huebner WP, Reschke MF. Locomotor head-trunk coordination strategies following space flight. *J Vestib Res.* 1997;7(2–3):161–77.
33. Imai T, Moore ST, Raphan T, Cohen B. Interaction of the body, head, and eyes during walking and turning. *Exp Brain Res.* 2001;136(1):1–18.
34. Hirasaki E, Moore ST, Raphan T, Cohen B. Effects of walking velocity on vertical head and body movements during locomotion. *Exp Brain Res.* 1999;127(2):117–30.
35. Berthoz A, Pozzo T. Intermittent head stabilization during postural and locomotory tasks in humans. In: Amblard B, Berthoz A, Clarac F, editors. *Posture and gait: development, adaptation, and modulation.* Amsterdam (NL): Elsevier; 1988. p. 189–98.
36. Glasauer S, Amorim MA, Vitte E, Berthoz A. Goal-directed linear locomotion in normal and labyrinthine-defective subjects. *Exp Brain Res.* 1994;98(2):323–35.
37. McGibbon CA, Krebs DE, Scarborough DM. Vestibulopathy and age effects on head stability during chair rise. *Acta Otolaryngol.* 2001;121(1):52–58.
38. Dagherstani L, Anderson JH, Flanders M. Coordination of a step with a reach. *J Vestib Res.* 2000;10(2):59–73.
39. Berthoz A. *The brain’s sense of movement.* Cambridge (MA): Harvard University Press; 2000.
40. Keshner EA, Cromwell RL, Peterson BW. Mechanisms controlling human head stabilization. II. Head-neck characteristics during random rotations in the vertical plane. *J Neurophysiol.* 1995;73(6):2302–12.
41. Allum JH, Honnegger F, Schicks H. The influence of a bilateral peripheral vestibular deficit on postural synergies. *J Vestib Res.* 1994;4(1):49–70.

Submitted for publication January 9, 2004. Accepted in revised form September 28, 2004.