Published in final edited form as:

Otol Neurotol. 2017 March ; 38(3): 373–378. doi:10.1097/MAO.0000000000001301.

Compensatory Saccades are Associated with Physical Performance in Older Adults: Data from the Baltimore Longitudinal Study of Aging

YanJun Xie, B.A.¹, Eric R. Anson, P.T., Ph.D.¹, Eleanor M. Simonsick, Ph.D.², Stephanie A. Studenski, M.D., M.P.H.², and Yuri Agrawal, M.D., M.P.H.¹

¹Department of Otolaryngology—Head and Neck Surgery, Johns Hopkins University School of Medicine, 601 North Caroline Street, 6th Floor, Baltimore, Maryland 21287 United States

²Longitudinal Studies Section, Translational Gerontology Branch, National Institute on Aging, National Institute of Health, 251 Bayview Boulevard Suite 100, Baltimore, Maryland 21224 United States

Abstract

Objective—To determine whether compensatory saccade metrics observed in the video head impulse test, specifically saccade amplitude and latency, predict physical performance.

Study Design—Cross-sectional analysis of the Baltimore Longitudinal Study of Aging (BLSA), a prospective cohort study.

Setting—National Institute on Aging Intramural Research Program Clinical Research Unit in Baltimore, Maryland

Patients—Community-dwelling older adults

Intervention(s)—Video head impulse testing was performed, and compensatory saccades and horizontal vestibulo-ocular reflex (VOR) gain were measured. Physical performance was assessed using the Short Physical Performance Battery (SPPB), which included the feet side-by-side, semi-tandem, tandem, and single-leg stance; repeated chair stands; and usual gait speed measurements.

Main Outcome Measure(s)—Compensatory saccade amplitude and latency, VOR gain, and SPPB performance.

Results—In 183 participants who underwent vestibular and SPPB testing (mean age 71.8 years; 53% females), both higher mean saccade amplitude (odds ratio [OR] =1.62, p=0.010) and shorter mean saccade latency (OR=0.88, p=0.004) were associated with a higher odds of failing the tandem stand task. In contrast, VOR gain was not associated with any physical performance measure.

Corresponding Author: YanJun Xie, B.A., Department of Otolaryngology—Head and Neck Surgery, Johns Hopkins University School of Medicine, 601 North Caroline Street 6th Floor, Baltimore, Maryland 21287 United States, johnny@jhmi.edu, Phone: 410-614-9825, Fax: 410-955-0035.

Conflicts of Interest:

For the remaining authors none were declared.

Conclusions—We observed in a cohort of healthy older adults that compensatory saccade amplitude and latency were associated with tandem stance performance. Compensatory saccade metrics may provide insights into capturing the impact of vestibular loss on physical function in older adults.

Introduction

Vestibular receptors of the inner ear detect head movements, and equal and opposite eye movements are generated through the vestibulo-ocular reflex (VOR) to keep gaze fixed in space. The VOR is an evolutionarily ancient mechanism that allows for maintenance of stable gaze during locomotion (1). When the vestibular response is deficient, a class of eye movements called compensatory saccades occurs to rapidly move the eyes back to a visual target of interest. These saccades form the basis of the clinical head impulse test: their presence signals vestibular impairment (2).

New head and eye movement tracking technology has enabled quantitative measurement of these compensatory saccades. Both the magnitude of the saccadic response as well as its precise latency can be computed. At present, the functional significance of these compensatory saccade measures has not been fully established. It has been shown that shorter saccade latencies are associated with improved dynamic visual acuity (3). However, it is not known whether the magnitude or latency of saccades is reflected in functional measures such as postural control or gait speed. Moreover, it is not known whether compensatory saccade metrics independently predict functional performance beyond more standard measures of vestibular function such as the VOR gain.

In this study, we evaluate the relationship between VOR function, compensatory saccades, and physical performance using data from healthy older adults in the Baltimore Longitudinal Study of Aging. We have previously documented an association between vestibular loss and aging in this population (4, 5). In this study, we specifically examined whether VOR gain, compensatory saccade amplitude, and latency were associated with performance on the Short Physical Performance Battery (SPPB). We also compared VOR gain vs. saccade metrics as predictors of physical performance.

Material and Methods

Study participants

The Baltimore Longitudinal Study of Aging (BLSA) is an ongoing prospective cohort study initiated in 1958 and currently supported by the Intramural Research Program (IRP) of the National Institute on Aging (NIA). The study assesses the health, cognitive, and functional status of a cohort of over 1300 community-dwelling individuals every 1–4 years to evaluate the normative aging process. All participants in this study provided written informed consent, and the BLSA protocol was approved by the Institutional Review Board of the National Institute of Environmental Health Sciences in Research Triangle Park, North Carolina. The study takes place at the NIA IRP Clinical Research Unit in Baltimore, Maryland. We used cross-sectional data on a sample of 201 individuals seen between

February 2013 and December 2014 who underwent vHIT and physical performance assessment.

Demographic data, smoking history, and cardiovascular risk factors were obtained. Race/ethnicity was categorized as “white”, “black”, and “other.” Education was categorized as “less than college”, “college”, and “more than college.” Smoking status was assessed by asking participants “Have you smoked at least 100 cigarettes over your entire life,” “Have you smoked at least 50 cigars over your entire life,” and “Have you smoked at least 3 packages of pipe tobacco over your entire life?” Participants were categorized as “never smoker” or “smoked 100 cigarettes or equivalent.” A history of hypertension and diabetes was obtained based on physician diagnosis in a medical interview.

Vision and proprioception were quantified and included in the analysis. Visual acuity was measured using the CSV-1000 Early Treatment Diabetic Retinopathy Study chart (VectorVision, Greenville, OH) and recorded in logMAR (logarithm of minimal angle of resolution) units. Standard visual acuity measures of 20/10, 20/20, 20/40, and 20/200 correspond to logMAR scores of -0.3, 0, 0.3, and 1.0. Proprioception was measured using customized equipment consisting of two pedals: one controlled by a motor operating at 0.3 degrees/second and another controlled freely by the participant (BALDOR, Fort Smith, AZ). For testing, participants were blindfolded and asked to place their bare or stocking feet on the pedals that were set at a natural ankle angle of 100 degrees at baseline. Over four trials of pre-set motor patterns, the pedals measured participant’s angle deviation from baseline using potentiometers. The smallest angular displacement (degrees) of ankle deviation that was detected by the participant in the motorized foot was defined as the proprioceptive threshold (6).

Video head impulse testing

VHIT assesses the angular VOR and was previously validated in older adults (7–8). Prior to testing, participants removed their corrective spectacles and sat 1.25 meters from a visual fixation target. They wore the EyeSeeCam video-oculography system consisting of a light-weight goggle frame with a built-in camera to record eye movements and an accelerometer to record head movements (Interacoustics USA, Eden Prairie, MN). In our testing, we applied the goggles as tightly as we thought possible around the participants’ head, while maintaining their comfort. Right eye position was calibrated using projected targets from a glasses-mounted laser. A trained examiner tilted the subject’s head 30 degrees below the horizon to bring the horizontal semicircular canal into the rotational plane. Then the examiner applied 10 to 15 low amplitude (15–20 degrees) horizontal head impulses to each side with unpredictable direction and timing. Subjects were excluded if they were blind, had poor neck range of motion, cervical spine instability, or a history of vascular surgery in the neck. Certified examiners evaluated vHIT tracings using custom software (MATLAB, MathWorks) and rejected tests with pupil tracking artifact or incorrect performance (i.e., low peak velocity, excessive head recoil, or overshoot saccades that exceeded 50 degree/second) (9). Peak head velocity ranged from 150–200 degrees/second, and eye velocity was measured in the right eye from a two-point differentiator. VOR gain was defined as the ratio

of the area under the eye velocity curve to the head velocity curve from HIT onset until the head velocity returned to zero (10).

Compensatory saccade analysis

In post-hoc analyses, saccades were identified by an algorithm based on eye accelerations >4000 degrees/second² and verified by visual inspection. In this study, we analyzed the first compensatory saccade that was generated in the same direction as the VOR response (see Figure 1 for example). Of 201 individuals who underwent vHIT, 183 subjects generated at least one compensatory saccade. Mean saccade amplitude (degrees) and latency (milliseconds, [ms]) were calculated for all head impulses that generated a compensatory saccade for each individual. To exclude volitional responses (i.e., saccades not made reflexively to compensate for a deficient VOR), we analyzed saccades that occurred between 25 and 503ms after vHIT onset. We chose 25ms based on previously reported ranges for active and passive HIT (11). To determine the latest time for non-volitional saccades, we estimated the population average vHIT duration in the study cohort with bootstrapping. We estimated 1000 new populations allowing for re-sampling, with 500 nested re-samples for variance estimates. This resulted in a population average vHIT duration of 253ms. We used 503ms as the maximal latency after which saccades could no longer be reliably attributed to HIT stimulus, consistent with reported values for volitional saccade latencies in older adults (12,13). Artifacts in video head impulse testing had been reported by Mantokoudis et al., including a 23% rate of overshoot or “bounce” saccades (14). In this study, we rejected tracings with overshoot that exceeded 50 degree/second to exclude “bounce” saccades and included only impulses that were correctly applied.

Physical performance measurement

Physical performance was assessed using the Short Physical Performance Battery (SPPB) which consisted of standing balance tests, repeated chair stands, and a 6-meter walk. We administered SPPB testing on the 183 subjects who generated at least one compensatory saccade. To assess standing balance, participants were asked to sequentially stand with their feet side-by-side, in semi-tandem, in full tandem, and on one leg for 10 seconds each. Whether or not participants could complete the balance task for 10 seconds was recorded. For the chair stands, participants were asked to fold their arms across their chest and stand up once from a chair. If successful they were instructed to stand up and sit down five times as quickly as possible, and the time to completion was recorded. Finally, a usual pace 6-meter walk was timed from a standing start. Participants walked twice, and gait speed (meters/second) was calculated. Subjects were assigned a performance score based on a previously published scoring system with a maximum score of 12 (15–16). Individuals who scored less than 10 were shown to have a higher risk of developing disability related to mobility and activities of daily living (16). Based on this finding, performance score in our cohort was categorized into <10 and 10–12 groups.

Analysis

We evaluated demographic and health characteristics and SPPB performance using descriptive statistics. We used multivariate logistic regression to evaluate the association between VOR gain and compensatory saccade metrics and physical performance after

adjusting for demographic, health-related variables (age, sex, race, education level, smoking status, a history of hypertension, diabetes, visual acuity, proprioceptive threshold), and VOR gain. Vision and proprioception are potential confounders of performance on balance testing and were adjusted for in the models. We also included VOR gain in all models in order to assess whether saccade metrics independently predicted physical performance. A *p*-value less than 0.05 was considered statistically significant. The STATA 13 statistical software was used for all analyses (StataCorp, College Station, TX).

Results

From February 2013 to December 2014, 183 participants (mean age 71.8 years, standard deviation [SD]: 13.2; 53% females) underwent concurrent vHIT and SPPB testing in the BLSA (Table 1). Mean VOR gain in the study cohort was 1.0 (SD: 0.1). Mean compensatory saccade amplitude was 2.2 degrees (SD: 0.8) and mean latency was 276 ms (SD: 71.1). With regard to physical performance, 99.5% and 97.3% of participants completed the feet side-by-side and semi-tandem stands respectively. However, only 75% of participants were able to hold the tandem stand for 10 seconds, and 49% of participants were able to stand on one leg for 10 seconds. The average time to complete the five repeat chair stands was 9.3 seconds (SD: 3.0). The mean gait speed was 1.1 meters/second (SD: 0.2). The summary performance scores in the study cohort ranged from 3 to 12, and the mean performance score was 11.5 (SD: 1.5).

We evaluated the association between VOR gain, saccade amplitude, saccade latency and SPPB performance using multivariate logistic regression (Table 2). We observed that each degree increase in mean saccade amplitude was associated with a 62% higher odds of failure on the tandem stand task (odds ratio [OR] =1.62, *p*=0.010, 95% confidence interval [CI]: 1.12, 2.35. Additional data are given in the Appendix). Further, we observed that each 10ms increase in mean saccade latency was associated with a 12% lower odds of failing the tandem stand (OR=0.88, *p*=0.004, 95% CI: 0.81, 0.96). We did not observe a significant association between saccade amplitude or latency and performance on other components of the standing balance tests (i.e., feet side-by-side, semi-tandem, and single leg stance), the repeat chair stands, usual gait speed, score category, or the overall performance score. Additionally, mean VOR gain was not associated with any of the physical performance measures.

To account for multiplicity of SPPB testing, we considered the Bonferroni correction. Applying the most conservative correction, the adjusted *p*-value for 6 tests would be *p*<0.0083. However, for several tests in the SPPB, the likelihood of finding a significant difference was low due to a lack of variability in performance. For example, only 5/183 participants (2.7%) failed the semi-tandem stance; similarly, 13/183 participants (7.1%) scored below 10 in the SPPB performance score. In contrast, a larger proportion of individuals (*n*=44, 24.0%) failed the full tandem stance where we observed a significant association between saccade properties and performance. Recognizing the above limitations and the hypothesis-generating nature of this work, we did not apply the Bonferroni correction to our data.

Discussion

In this study, we observed a significant association between compensatory saccade amplitude, latency, and SPPB performance in a cohort of healthy older adults. Specifically, we observed that a higher mean saccade amplitude and a shorter mean saccade latency were independently associated with inability to perform the 10-second tandem stand. Although VOR gain is typically used to assess VOR function, it was not significantly associated with any physical performance measure in these data. The current study suggests that mean compensatory saccade amplitude and latency are associated with full tandem stance performance and may reflect the functional impact of partial loss of vestibular function due to aging.

Compensatory saccades are an oculomotor gaze-stabilizing strategy that compensates for a deficient VOR (17). Prior studies have demonstrated a relationship between compensatory saccades and VOR function. One study of nine labyrinthine-defective patients observed that poorer VOR function (defined by greater gaze position error) was associated with higher amplitude and longer latency of compensatory saccades (18). Another case report of a single patient with unilateral vestibular loss reported a reduction in saccade amplitude and shortening of saccade latency as the patient's VOR gain returned to baseline (3). These previous studies suggest that higher saccade amplitude and longer saccade latency indicate poorer VOR function.

In our study, we also observed that higher saccade amplitude was associated with inability to perform the tandem stand. Presumably, poorer vestibular function results in greater retinal slip, which triggers compensatory saccades of larger amplitudes (17). However, in contrast to previous studies, we observed that shorter saccade latency was associated with poorer physical performance. Prior studies among patients with recent vestibular deafferentation observed that vestibular compensation was associated with shorter saccade latencies (14). It is possible that in our cohort of healthy older individuals with partial vestibular loss, vestibular compensation has already occurred. Thus in this study, saccade latency appears to be an indicator of underlying vestibular function rather than vestibular compensation. Poorer VOR function and greater levels of retinal slip may have triggered earlier compensatory saccades. It is possible that in other cohorts with greater levels of vestibular impairment or with more recent-onset vestibular losses, saccade latency more clearly reflects the ability to compensate for vestibular loss rather than underlying vestibular function.

In this study, we found that in contrast to the association between saccade properties and full tandem performance, VOR gain was not associated with any physical performance metrics in older adults. VOR gain deficits have been previously associated with physical and functional impairment in patients with vestibulopathies (19–20). However, among older adults with partial vestibular losses due to aging, VOR gain may be a less sensitive measure to distinguish lower levels of VOR impairment compared to compensatory saccade amplitude and latency (21).

We only noted an association between compensatory saccade metrics and the tandem stand test of the SPPB. In this cohort of healthy adults, most individuals were able to complete the

side-by-side and semi-tandem stance. As such, insufficient variability in the outcome measure may have accounted for the lack of significant association with vestibular measures. The narrow base of support in tandem stance results in reduced mechanical stability that necessitates an increased weighting of sensory input (22–23). During this challenging postural task, the individual may rely more heavily on vestibular inputs to counteract the tendency to sway and maintain an upright body orientation (24). Indeed, a tandem walking test has been shown to be useful in screening older adults for vestibular disorders (25). Our findings corroborate that the tandem stance test is one of the more specific indicators of vestibular function.

We did not observe a significant association between saccade metrics and the single leg stance. The single leg stance is also a challenging balance task, but it may be more dependent on lower extremity muscle strength and endurance in older adults rather than sensory postural adjusting systems (26). Further, we did not observe a significant association between saccade metrics or VOR gain and gait speed. It has been shown that vestibular inputs are more salient during gait transitions (e.g. moving from stance to walking or sitting to standing) (27). As such, a timed walk on a straight path may not have captured moments when vestibular inputs are most critical, and thus may be less informative about underlying vestibular function.

We note several limitations in this study. First, the data we analyzed are cross-sectional and cannot support casual inferences between saccade characteristics and physical performance. It is possible that poorer physical performance resulted in poorer vestibular function, perhaps through reduced levels of physical activity. It is also possible that unmeasured confounders account for the observed associations in our study. Future studies will be needed to establish causality and better delineate how compensatory saccades may predict physical performance. Second, we rejected tracings with more than 50 degree/second overshoot to exclude “bounce” saccades. However, the roles of these overshoots, as well as their impact on the amplitude and latency of overt saccades, are not well understood and should be addressed in future studies. Moreover, we evaluated multiple associations for this exploratory study. Although we observed a consistent trend related to compensatory saccade metrics and tandem stance, further studies based on the hypotheses generated by this study will be needed to confirm these findings. Another limitation relates to the healthy older cohort that we studied. The association between saccade metrics and physical performance may be different among older individuals with greater levels of vestibular impairment. Our study was also limited by the SPPB tests performed in the BLSA. A more challenging and vestibular-specific dynamic balance task such as the Functional Gait Assessment may demonstrate a stronger relationship with vestibular function (28). Finally, we did observe some cases where the eye movement preceded the head movement, although this was far from the majority of cases. Goggle slippage had been suggested to explain the “eye-before-head” phenomenon (29), and in our testing, we applied the goggles as tightly as we thought possible around the participants’ head, while maintaining their comfort. Nevertheless, goggle slippage may have occurred. Other potential explanations for “eye-before-head” tracing have also been suggested such as cerebellar dysfunction (30). Given the lack of consensus about the nature of these findings, we chose to include all impulses in our analysis as might be encountered in a clinical setting. We also only considered horizontal VOR

function and its associated compensatory saccades, given the time and technical limitations of testing vertical canal VOR.

In summary, we observed that compensatory saccade amplitude and latency were significantly associated with performance of the tandem stance in a cohort of healthy older adults. Compensatory saccade characteristics from vHIT provided insights into the gradual loss of VOR function and its associated functional impairment in older adults. These data suggest the importance of quantifying compensatory saccade metrics to capture the impact of vestibular impairment on physical function in healthy older adults. Future studies are needed to determine if this relationship is causal and understand how VOR deficit affects the various dimensions of physical performance. Screening for vestibular impairment using compensatory saccade analysis in older adults and intervening with vestibular therapy may be beneficial in stabilizing the trajectory of functional decline.

Acknowledgments

Source of Funding:

Author Eric Anson receives funding support from the National Institute of Health (NIH) grant T32-DC000023.
Author Yuri Agrawal receives funding support from the NIH grant NIDCD K23-DC013056.

References

1. Walls GL. The evolutionary history of eye movements. *Vision Res.* 1962; 2(1–4):69–80.
2. Halmagyi GM, Curthoys IS. A clinical sign of canal paresis. *Arch Neurol.* 1988; 45(7):737–739. [PubMed: 3390028]
3. Schubert MC, Migliaccio AA, Della Santina CC. Modification of compensatory saccades after aVOR gain recovery. *J Vestib Res.* 2006; 16(6):285–291. [PubMed: 17726282]
4. Li C, Layman AJ, Carey JP, Agrawal Y. Epidemiology of vestibular evoked myogenic potentials: Data from the Baltimore Longitudinal Study of Aging. *Clin Neurophysiol.* 2015 Jan.
5. Li C, Layman AJ, Geary R, et al. Epidemiology of Vestibulo-Ocular Reflex Function. *Otol Neurotol.* 2015; 36(2):267–272. [PubMed: 25275869]
6. Ko S-U, Simonsick E, Deshpande N, Ferrucci L. Sex-specific age associations of ankle proprioception test performance in older adults: results from the Baltimore Longitudinal Study of Aging. *Age Ageing.* 2015; 44(3):485–490. [PubMed: 25637144]
7. Bartl K, Lehnen N, Kohlbecher S, Schneider E. Head impulse testing using video-oculography. *Ann N Y Acad Sci.* 2009; 1164:331–333. [PubMed: 19645921]
8. Agrawal Y, Schubert MC, Migliaccio AA, et al. Evaluation of quantitative head impulse testing using search coils versus video-oculography in older individuals. *Otol Neurotol.* 2014; 35(2):283–288. [PubMed: 24080977]
9. Mantokoudis G, Saber Tehrani AS, Kattah JC, et al. Quantifying the vestibulo-ocular reflex with video-oculography: nature and frequency of artifacts. *Audiol Neurotol.* 2015; 20(1):39–50. [PubMed: 25501133]
10. MacDougall HG, McGarvie LA, Halmagyi GM, Curthoys IS, Weber KP. The video Head Impulse Test (vHIT) detects vertical semicircular canal dysfunction. *PLoS One.* 2013; 8(4):e61488. [PubMed: 23630593]
11. Tian J-R, Crane BT, Ishiyama A, Demer JL. Three dimensional kinematics of rapid compensatory eye movements in humans with unilateral vestibular deafferentation. *Exp brain Res.* 2007; 182(2): 143–155. [PubMed: 17549461]
12. Abel LA, Troost BT, Dell'Osso LF. The effects of age on normal saccadic characteristics and their variability. *Vision Res.* 1983; 23(1):33–37. [PubMed: 6868379]

13. Warren DE, Thurtell MJ, Carroll JN, Wall M. Perimetric evaluation of saccadic latency, saccadic accuracy, and visual threshold for peripheral visual stimuli in young compared with older adults. *Invest Ophthalmol Vis Sci.* 2013; 54(8):5778–5787. [PubMed: 23882693]
14. Mantokoudis G, Schubert MC, Tehrani ASS, Wong AL, Agrawal Y. Early adaptation and compensation of clinical vestibular responses after unilateral vestibular deafferentation surgery. *Otol Neurotol.* 2014; 35(1):148–154. [PubMed: 23965525]
15. Guralnik JM, Simonsick EM, Ferrucci L, et al. A short physical performance battery assessing lower extremity function: association with self-reported disability and prediction of mortality and nursing home admission. *J Gerontol.* 1994; 49(2):M85–M94. [PubMed: 8126356]
16. Guralnik JM, Ferrucci L, Pieper CF, et al. Lower extremity function and subsequent disability: consistency across studies, predictive models, and value of gait speed alone compared with the short physical performance battery. *J Gerontol A Biol Sci Med Sci.* 2000; 55(4):M221–M231. [PubMed: 10811152]
17. MacDougall HG, Curthoys IS. Plasticity during Vestibular Compensation: The Role of Saccades. *Front Neurol.* 2012; 3(February):1–9. [PubMed: 22279441]
18. Tian J, Crane BT, Demer JL. Vestibular catch-up saccades in labyrinthine deficiency. *Exp brain Res.* 2000; 131(4):448–457. [PubMed: 10803413]
19. Herdman SJ, Blatt P, Schubert MC, Tusa RJ. Falls in patients with vestibular deficits. *Am J Otol.* 2000; 21(6):847–851. [PubMed: 11078074]
20. Iwasaki S, Yamasoba T. Dizziness and Imbalance in the Elderly: Age-related Decline in the Vestibular System. *Aging Dis.* 2015; 6(1):38–47. [PubMed: 25657851]
21. Paige GD. Senescence of human visual-vestibular interactions. 1. Vestibulo-ocular reflex and adaptive plasticity with aging. *J Vestib Res.* 1992; 2(2):133–151. [PubMed: 1342388]
22. Winter DA, Patla AE, Frank JS. Assessment of balance control in humans. *Med Prog Technol.* 1990; 16(1–2):31–51. [PubMed: 2138696]
23. O'Connor SM, Kuo AD. Direction-Dependent Control of Balance During Walking and Standing. *J Neurophysiol.* 2009; 102(3):1411–1419. [PubMed: 19553493]
24. Cohen HS, Mulavara AP, Peters BT, et al. Sharpening the Tandem Walking Test for Screening Peripheral Neuropathy. *South Med J.* 2013; 106(10):565–569. [PubMed: 24096950]
25. Cohen HS, Sangi-Haghpeykar H, Ricci NA, Kampangkaew J, Williamson RA. Utility of Stepping, Walking, and Head Impulses for Screening Patients for Vestibular Impairments. *Otolaryngol Head Neck Surg.* 2014; 151(1):131–136. [PubMed: 24664545]
26. Jonsson E, Seiger A, Hirschfeld H. One-leg stance in healthy young and elderly adults: a measure of postural steadiness? *Clin Biomech (Bristol, Avon).* 2004; 19(7):688–694.
27. Bent LR, Inglis JT, McFadyen BJ. When is vestibular information important during walking? *J Neurophysiol.* 2004; 92(3):1269–1275. [PubMed: 15102904]
28. Wrisley DM, Marchetti GF, Kuharsky DK, Whitney SL. Reliability, internal consistency, and validity of data obtained with the functional gait assessment. *Phys Ther.* 2004; 84(10):906–918. [PubMed: 15449976]
29. Collewijn H, Smeets JB. Early components of the human vestibulo-ocular response to head rotation: latency and gain. *J Neurophysiol.* 2000; 84(1):376–389. [PubMed: 10899212]
30. Crane BT, Tian JR, Demer JL. Initial vestibulo-ocular reflex during transient angular and linear acceleration in human cerebellar dysfunction. *Exp brain Res.* 2000; 130(4):486–496. [PubMed: 10717790]

Figure 1.

Sample video head impulse test (vHIT) tracing from three participants (red line: eye movement, blue line: head movement). In this example, three individuals with a similar VOR gain (~ 0.95) had different compensatory saccade amplitudes and latencies

Table 1

Demographic, health characteristics, and performance on the Short Physical Performance Battery (SPPB), the Baltimore Longitudinal Study of Aging (BLSA)

Study variable	N	Mean (SD ^a)	Percentage
Mean age in years	183	71.8 (13.2)	100
Gender			
Male	86		47.0
Female	97		53.0
Race/ethnicity			
White	114		62.3
Black	53		29.0
Other	16		8.7
Education level			
Less than college	18		9.8
College	43		23.5
More than college	81		44.3
Smoking			
Never smoked	80		43.7
Smoked 100 cigarettes or equivalent	62		33.9
History of hypertension			
Yes	69		37.7
No	73		39.9
History of type II diabetes			
Yes	35		19.1
No	106		57.9
Visual acuity (logMAR ^b)	140	0.05 (0.1)	
Proprioceptive threshold (degree)	130	1.6 (1.9)	
Mean VOR gain	183	1.0 (0.1)	
Mean saccade amplitude (degree)	183	2.2 (1.7)	
Mean saccade latency (millisecond)	183	276 (71.1)	
Feet side by side stance			
Passed	182		99.5
Failed	1		0.5
Semi-tandem stance			
Passed	178		97.3

Study variable	N	Mean (SD ^a)	Percentage
Failed	5		2.7
Full tandem stance			
Passed	136		74.3
Failed	44		24.0
Single leg stance			
Passed	75		49.0
Failed	78		55.6
Time for repeated chair stands (second)	174	9.3 (3.0)	
Usual gait speed (meter/second)	176	1.1 (0.2)	
SPPB performance category			
Scores <10	13		7.1
Scores 10–12	170		92.9
Total SPPB performance score	181	11.5 (1.5)	

^aSD: standard deviation

^blogMAR: logarithm of the minimal angle of resolution. LogMAR scores of −0.3, 0, 0.3, and 1.0 correspond to standard visual acuity measures of 20/10, 20/20, 20/40, and 20/200.

Multivariate regression of saccade metrics and SPPB performance after adjusting for demographic and health covariates. Feet side-by-side stance was omitted due to the small number of participants who failed this condition (182 passed, 1 failed).

Table 2

vHIT measures	Semi-tandem stance		Full tandem stance		Single leg stance		Repeated chair stands time (second)		Usual gait speed (m/s)		SPPB performance score category		Total SPPB performance score	
	OR ^a	p	OR	p	OR	p	β	p	β	p	OR	p	β	p
Mean VOR gain	12.04	0.608	2.07	0.678	3.84	0.589	1.64	0.342	-0.08	0.540	0.05	0.204	-1.73	0.064
Mean saccade amplitude (degree)	1.08	0.768	1.62 [*]	0.010 [*]	1.38	0.112	-0.06	0.607	0.00	0.875	1.58	0.229	0.04	0.547
Mean saccade latency (per 10 ms)	0.96	0.545	0.88 [*]	0.004 [*]	0.94	0.141	-0.04	0.211	0.00	0.600	0.94	0.384	0.00	0.822

^{*} Statistically significant values (p<0.05); see appendix for detailed statistical values.

^a OR=odds ratio