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Reduced vestibular function is associated with longer, slower steps in healthy adults during normal speed walking

E Anson, PT, PhD[a,b], **K Pineault**[a], **W Bair, PhD**[c], **S Studenski, MD, MPH**[c], and **Y Agrawal, MD, MPH**[a]

[a]Department of Otolaryngology - Head & Neck Surgery, Johns Hopkins University School of Medicine, Baltimore, MD, USA

[b]Department of Otolaryngology, University of Rochester, Rochester, NY, USA

[c]Longitudinal Studies Section, National Institute on Aging, Baltimore, MD, USA

Abstract

Background: Vestibular signals contribute to balance and walking. With aging, vestibular function declines and gait speed decreases. Vestibular loss contributes to decreasing gait speed, but this influence could be linked to spatial and/or temporal aspects of gait. We investigated the relationship between vestibular function (semicircular canal and otolith function) and spatial and temporal gait parameters in a cohort of adults.

Methods: 113 community-dwelling healthy adults (mean age 72.2 (14.6) years) participating in the Baltimore Longitudinal Study of Aging were tested.

Horizontal semicircular canal (SCC) function was evaluated using quantitative vestibulo- ocular reflex gain. Otolith function was measured with cervical and ocular vestibular evoked myogenic potentials. Gait kinematics were collected during normal speed walking. Multiple linear regressions examined the association between spatial and temporal gait parameters and SCC and otolith function separately, controlling for age, gender, height, and either cadence (for spatial gait outcomes) or stride length (for temporal gait outcomes) to account for gait speed effects.

Results: Vestibular SCC function was significantly associated with both spatial and temporal gait parameters. Every 0.1 decrease in SCC function resulted in longer stride length ($\beta = -.04$ meters, $p = 0.004$), longer stance time ($\beta = 15.8$ ms, $p < 0.003$), and a slower cadence ($\beta = -2.1$ steps/minute, $p < 0.001$). Otolith function was not associated with any gait parameter.

Correspondence: Eric Anson, Department of Otolaryngology, 601 Elmwood Ave, Box 629, Rochester, NY 14642, Phone: 585-276-5719, eric_anson@urmc.rochester.edu.

Presentation

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Disclosure of Interest

All authors have no conflict of interest.

Conclusions: Reduced horizontal SCC function was associated with longer, slower steps in a cohort of healthy adults. These results indicate that vestibular signals contribute to specific spatial and temporal aspects of gait thought to contribute to upright balance.

INTRODUCTION

The vestibular system plays a critical role in the modulation of gait [1]. Previous research indicates that the otolith organs -- the saccule and utricle -- contribute linear acceleration cues to control gait speed [2] and to maintain upright posture during gait [3]. Semicircular canal input from vertical head rotation also contributes to walking balance and horizontal head rotation contributes to navigation [4]. The semicircular canals further provide important angular velocity input during walking which is necessary for gaze stability [5]. When there is damage to these vestibular organs and subsequent loss of sensory input during motion, normal gait patterns are compromised [6]. Recent studies have shown a link between vestibular impairment and gait abnormalities in patients with vestibular disorders such as vestibular neuritis and vestibular schwannoma, including decreased gait speed and increased variability in stance and swing time [7,8].

There is conflicting evidence however regarding relationships between measures of vestibular function and gait. The gaze stabilization test (GST) evaluates how fast the head can rotate and still read a letter clearly [9]. Both horizontal and vertical GST scores have been associated with clinical measures of walking balance [10,11], suggesting that vestibulo-ocular reflex (VOR) function is related to walking balance ability. Recently, change in horizontal semicircular canal VOR gain was associated with change in walking balance measured using the Dynamic Gait Index [12]. In contrast, horizontal semicircular canal VOR gain measured with video head impulses and rotational chair tests was not significantly associated with measures of trunk sway during walking [13]. Taken together these studies suggest a role for horizontal semicircular canal function in mediating walking balance, possibly related to foot placement [14]. Otolith function was not reported in those studies; therefore, the contributions of the saccule and utricle to dynamic balance in those studies is unknown.

Numerous basic, clinical and epidemiologic studies have demonstrated a clear loss of vestibular function associated with normal aging [15,16]. Important changes in peripheral vestibular signaling occur with advancing age including: a decrease in number of sensory hair cells [17], degeneration of the vestibular ganglion and nerve [18], and degeneration and fragmentation of otoconia [19]. Age-related vestibular physiologic deficits have been shown to result from the degeneration of these anatomical structures including abnormal angular vestibulo-ocular reflex (VOR) function [20], and diminished otolith responses as measured by reduced amplitude and increased latency of vestibular-evoked myogenic potentials (VEMPs) [21,22]. Interestingly, the overall prevalence of semicircular canal dysfunction has been determined to be higher compared to otolith dysfunction in the older population [21].

With age, gait speed decreases and the gait pattern becomes more variable [23]. Although aging has been shown to have a profound impact on both vestibular function and gait over time, few studies have investigated the changes in spatial and temporal aspects of gait

associated with age-related vestibular loss. Investigation into how vestibular loss influences components of gait, such as stride length and cadence, independent of age and gait speed could be utilized to inform clinical practice to improve gait function and perhaps reduce fall risk. The purpose of this study was to identify a vestibular specific impact on spatial and temporal aspects of the gait cycle independent of age in a cohort of healthy older adults. We quantify the relationships between semicircular canal, saccular, and utricular function on spatial (stride length, step width) and temporal (cadence, swing time, stance time) gait parameters in a cohort of healthy adults.

METHODS

The Baltimore Longitudinal Study of Aging (BLSA) is an ongoing prospective cohort study initiated in 1958 and currently conducted by the Intramural Research Program of the National Institute on Aging (IRP-NIA). Subjects are community-dwelling participants age 20 and older who undergo a standardized array of tests over 3 days every 1–4 years at the Clinical Research Unit of the IRP-NIA in Baltimore, MD. This study includes a cross-sectional sample of all BLSA participants seen between January 2013 and April 2017. During this time period 113 participants underwent both vestibular and gait testing. The only exclusion criteria were with respect to vestibular testing as detailed below. All participants provided written informed consent. The BLSA study protocol was approved by the National Institute of Environmental Health Sciences Institutional Review Board.

Vestibular Function Tests

Individuals participating in the BLSA who consent to participate in vestibular testing undergo tests for both otolith function (cervical and ocular vestibular evoked myogenic potentials (VEMP)) and horizontal semicircular canal (SCC) function (video head impulse test (vHIT)). Detailed methods to measure horizontal SCC function using vHIT, cervical VEMPs and ocular VEMPs in this population have been published previously and are briefly described below [24]. Not all participants experienced all vestibular tests primarily due to criteria for exclusion from vHIT testing.

Video Head Impulse Testing

Participants wore either ICS Impulse 3-D video head impulse (vHIT) system (GN Otometrics, Schaumburg, Il) or the EyeSeeCam vHIT system (Interacoustics USA, Eden Prairie, MN). Each vHIT system consists of a lightweight goggle frame with a built in inertial measurement unit to record head velocity and a camera to record eye movements [25,26]. Participants received 10–15 small amplitude (15–20^{0}) head impulses to the right and left with their head tilted 30 degrees down from trained examiners while viewing a visual fixation target 1.25 meters away. Peak head impulse velocity was 150 to 250 degrees per second. Horizontal SCC VOR gain was calculated according to the manufacturers specifications [25,26]. Participants were excluded from head impulse testing if they had restricted neck rotation or pain with neck rotation. Vertical SCC testing was not performed due to testing restrictions at the BLSA.

Vestibular evoked myogenic potential (VEMP) recording conditions

VEMP tests were performed to measure otolith function. The cervical VEMP (cVEMP) is considered a test of saccular function, while the ocular VEMP (oVEMP) is considered a measure of utricular function. A commercial electromyographic (EMG) system (Carefusion Synergy, software version 14.1, Dublin, OH, USA) was used to record EMG signals with disposable, self-adhesive, pregelled, Ag/AgCl electrodes with 40-inch safety leadwires from GN Otometrics (Schaumburg, IL, USA). EMG signals were amplified 2500x and band-pass filtered, 20–2000 Hz for cervical VEMPs [27]. Subjects were reclined with their upper bodies elevated at 30 degrees from horizontal for all VEMP testing.

Ocular VEMPs—Participants maintained a 20 degree upward gaze during ocular VEMP (oVEMP) stimulation and recording. The first electrode was placed 2 cm below and centered on the pupil, with the second located 0.5 cm below and just lateral to the first electrode. 50 midline head taps were delivered at Fz with an Aesculap model ACO12C reflex hammer fitted with an inertial microswitch trigger. The oVEMP waveform consists of a negative peak (n10), identified as the first distinctive peak in the waveform, followed by a positive peak (p16), identified as the first distinctive trough in the waveform. Individuals with EMG recordings lacking definable n10 waves were defined as having an absent oVEMP response. oVEMP function was dichotomized as present (response in one or both ears) or bilaterally absent. Participants were excluded from the oVEMP test if they could not see the fixation point.

Cervical VEMPs—Participants lifted their heads up and rotated to the side to provide tonic background sternocleidomastoid (SCM) activity during stimulation and recording. Electrodes were placed at the SCM belly and the sternoclavicular junction and the ground electrode on the sternum. Air-conducted sound stimuli consisted of 500 Hz, 125 dB SPL tone bursts of positive polarity, with a linear envelope (1 ms rise/- fall time, 2 ms plateau), at a repetition rate of 5 Hz. Sound stimuli were delivered monaurally through Audiocups noise-excluding headset enclosures (Amplivox, Eden Prairie, MN). The cVEMP waveform consists of a positive peak (p13), identified as the first distinctive trough in the waveform, followed by a negative peak (n23), identified as the first distinctive peak in the waveform. Subjects with EMG recordings lacking definable p13 waves were defined as having an absent cVEMP response. cVEMP function was dichotomized as present (in one or both ears) or bilaterally absent. Participants were excluded from the cVEMP test if they had pain while turning their head fully to the side.

Spatial and Temporal Gait Parameters

Participants were asked to walk along a 10-meter walkway at their self-selected usual walking speed. The first two and last two steps in each walking trial were excluded to eliminate acceleration and deceleration phases from the analysis. For each participant a total of 6 trials, each with at least 5 useable steps, were assessed. Gait kinematics were recorded by a 10-camera Vicon motion analysis system (Oxford Metrics Ltd., UK) with a standard Plug-in-Gait markers. Kinematic data were sampled at 60 frame/sec. Spatial (stride length, stride length SD, step width, step width SD) and temporal (cadence, swing time, swing time SD, stance time, and stance time SD) gait parameters were calculated for each stride based

on right heel strikes and the average and SD of those gait parameters were used in statistical analyses.

Data Analysis

Separate multivariate linear regressions were used to determine the relationship between mean and variability (standard deviation (SD)) for each spatial and temporal gait parameter (stride length, stride length SD, step width, step width SD, cadence, swing time, swing time SD, stance time, and stance time SD) and 1) horizontal SCC VOR gain, or 2) cVEMP function, or 3) oVEMP function while controlling for height, age and gender. Gait speed is directly related to the dependent variables; therefore, we adjusted for either the temporal or spatial aspect of gait speed as follows. In models with spatial gait outcome variables, we adjusted for cadence (as a measure of temporal gait function) to evaluate the independent association between vestibular function and spatial gait parameters. Similarly, in analyses with temporal gait outcome variables, we adjusted for stride length (as a measure of spatial gait function) to evaluate the independent association between vestibular function and temporal gait parameters. STATA 14 (College Station, TX, USA) was used for all analyses. Associations with each measure of vestibular function (horizontal SCC VOR gain, cVEMP function [present/absent], and oVEMP function [present/absent]) were considered to be independent research questions. Nine independent regressions were performed for each vestibular variable; therefore, after dividing 0.05 by 9, an $\alpha = 0.0056$ was used for each regression analysis to adjust for multiple comparisons.

Results

The mean (SD) age of the participants was 72.2 (14.6) and 58% were male (Table 1). The average (SD) gait speed for this sample was 1.16 (0.21) meters/sec. Not all participants experienced all vestibular testing due to exclusion criteria resulting in a sample of 90 participants with both horizontal SCC VOR gain and gait testing, and 113 participants with oVEMP and cVEMP measures and gait testing.

VOR Gain and Gait Parameters

There were significant relationships between horizontal SCC VOR gain and both spatial and temporal aspects of gait (Table 2). Stride length (m) increased as horizontal SCC VOR gain decreased ($\beta = -.379$ meters, $p = 0.004$) after controlling for cadence, age, gender, and height. Both stance time ($\beta = -158.4$ ms, $p = 0.003$) and swing time ($\beta = -68.4$ ms, $p = 0.009$) increased as horizontal SCC VOR gain decreased after controlling for stride length, age, gender, and height, although the association was only significant for stance time. Cadence decreased as horizontal SCC VOR gain decreased ($\beta = 20.5$ steps/min, $p < 0.001$) after controlling for stride length, age, gender, and height. The same analyses were conducted controlling for gait speed rather than stride length or cadence and there was no substantive difference in the results (data not shown).

Otolith function and Gait Parameters

There were no significant relationships between saccular function as measured by cVEMP responses (present/absent) and any of the spatial or temporal aspects of gait examined in

these analyses (Table 3). There were no significant relationships between utricular function as measured by oVEMP responses (present/absent) and either spatial or temporal aspects of gait after controlling for cadence (stride length), age, gender, and height (Table 4). As a sensitivity analysis, we performed the same regression analyses using cVEMP and oVEMP amplitudes (data not shown). This reduced the sample size to 91 participants and there were no significant associations between any measure of otolith function and the reported gait parameters. The same analyses were conducted controlling for gait speed rather than stride length or cadence and there was no substantive difference in the results (data not shown).

Discussion

Semicircular canal function as measured by horizontal SCC VOR gain during head impulse testing was significantly associated with both spatial and temporal aspects of gait during normal speed walking. Reduced vestibular function was associated with a longer stride length, more time in stance, and a slower cadence. Previous investigations have demonstrated that gait speed, cadence, and step length are all slower (shorter) for individuals with vestibular disease compared to healthy controls [20,28]. However, gait speed, step timing, and step length are all inter-related [29]. Previous findings may have all been the effect of a single gait parameter and it was not clear whether the spatial (stride length), temporal (step timing), or composite (gait speed), are responsible for the previous results. To avoid this potential confound we included cadence or stride length as proxy variables representing average gait behavior in each model to independently evaluate the relationship between vestibular function and spatial and temporal gait variables. This allowed us to demonstrate that the relationships between horizontal SCC VOR gain and stride length, stance time, and cadence were present even after controlling for the relationship between those spatial and temporal gait parameters and a proxy variable representing average gait behavior.

Horizontal SCC function was associated with mean spatial and temporal aspects of gait. Mamoto and colleagues reported that when stride length was constrained individuals with peripheral vestibular disease had slower gait speed and slower cadence [30]. This is consistent with our observation that cadence and stance timing were slower in individuals with age-related vestibular loss after controlling for stride length. Other studies using galvanic electrical vestibular stimulation demonstrated a modulation of the temporal regularity of walking and an interaction between the magnitude of the muscle response to the electrical stimulation and gait speed [1,31]. Using electrical stimulation has an advantage of specifically stimulating vestibular afference, but the disadvantage is lack of end organ selectivity. Here we separately investigated whether there was a relationship between the average function of the vestibular system based on vestibular end-organ specific diagnostic testing and average spatial and temporal components of gait. We observed that semicircular canal function appears to play a predominant role in the vestibular regulation of gait. However, we note that the current analyses do not prove a causal relationship between semicircular canal function and dynamic gait modulation. Additionally, these results should not be interpreted as dynamic gait cycle phase dependent responses to vestibular stimulation as has been demonstrated by others since the vestibular stimulation was not applied during walking [1,31]. The present results rather demonstrate that in adults with age-related

vestibular loss, worse function of the semicircular canals preferentially lengthens the duration of the stance phase of the gait cycle, lengthens stride length, and slows cadence. The relationship between horizontal SCC VOR gain and gait parameters is particularly relevant as a previous study demonstrated that all older adults displayed SCC abnormalities [21]. The adaptations to the spatial and temporal aspects of gait observed here with aging may represent early changes that contribute to later fall risk as individuals with vestibular dysfunction have increased risk for falling [32,33]. Future studies are needed to determine whether there is a differential influence on dynamic gait modulation from semicircular canals and otoliths using techniques to stimulate specific end organs in isolation during walking.

Somewhat surprisingly, after correcting for multiple comparisons, we did not find any significant relationships between otolith function and spatial or temporal gait parameters. The current results do not imply that otolith sensory afference does not contribute to gait, in fact research in monkeys has demonstrated that the irregular fibers of the otoliths are ideally tuned to detect the frequencies of natural head motion [34]. Additionally, altered head positions during gait have also been shown to modulate muscle activity during gait in healthy adults [3]. Therefore, otolith inputs would facilitate head on body [35] and trunk on legs [36] stabilization for balance control during walking in addition to contributing to gaze stabilization [5]. Head position was not systematically altered in this study which could explain why there was not a significant relationship between otolith function and the gait parameters investigated here. Additionally, only about 10% of individuals had absent otolith function as measured by cVEMP and oVEMPs which may have contributed to our results. However, when VEMP amplitudes was used in a sensitivity analysis there were also no significant relationships between otolith function and gait parameters (data not shown). Future studies investigating the causal nature of otolith specific contributions to gait in humans should focus on head and/or trunk orientation rather than lower limb kinematics.

Interestingly, variability in stride length and step width increased significantly with age but not with any measure of vestibular function. In a previous study, vestibular loss was associated with increased gait variability [37], but the increased variability was dependent on gait speed. Individuals with vestibular loss have less gait variability when walking at their preferred speed. Individuals with vestibular diseases including vestibular neuritis, superior canal dehiscence, and vestibular schwannoma were all found to have greater variability in center of pressure trajectory during walking relative to healthy individuals [8]. Additionally, spatiotemporal variability but not average spatial and temporal gait parameters was modulated by noisy galvanic vestibular stimulation in individuals with bilateral vestibular loss [38]. However, the majority of differences reported by Angunsri et al. and Wuehr et al. were only observed during eyes closed walking [8,38]. The participants in the current study were older adults with age-related vestibular loss which may not have as profound an impact on gait variability as disease related vestibular loss. They also walked at their preferred gait speed with their eyes open which could also explain the lack of a significant relationship between vestibular function and gait variability observed in this study.

Limitations

This was a cross-sectional study and as such does not support causal inferences. The vestibular function tests were performed at a different time from the gait testing during the participants visit at the BLSA and the results do not represent dynamic vestibular modulation of gait unlike studies that use electrical vestibular stimulation. We report only horizontal SCC VOR gain, using vertical SCC VOR gain may result in different results. Different VEMP stimulation protocols may result in relationships between otolith function and the gait parameters different than those reported here. Other factors not included in this analysis like vision and pain when walking may also influence the gait parameters described here in ways not captured by the present analysis. The observed associations may be different in a sample of individuals with more profound vestibular loss or greater balance/gait problems.

Conclusion

Reduced vestibular function was associated with longer slower steps during walking at normal speed with eyes open in a cohort of older adults. This suggests more careful control over foot placement, trading typical gait cycle timing for postural control as vestibular function declines. These results suggest that vestibular signals contribute to specific spatial and temporal aspects of the gait cycle.

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Highlights

- Lower VOR gain was associated with longer, slower steps
- Otolith function was not associated with any gait parameter
- VOR gain decline may lead to a greater emphasis on balance during walking

Table 1.

Participant demographics

	Mean (SD) or N (%)
Age	72.2 (14.6), range 24–93
Gender	
Female	47 (41.6%)
Male	66 (58.4)
Height (m)	
Female	1.61 (0.72)
Male	1.74 (0.64)
Horizontal SCC VOR Gain[1]	0.96 (0.13)
cVEMP[2]	
Present	91
Absent	10
Normalized Amplitude	0.88 (0.48), n = 91
oVEMP[3]	
Present	91
Absent	14
Amplitude (μV)	11.1 (11.7), n = 91
Gait Speed (m/s)	1.16 (0.21)
Cadence (steps/min)	112.4 (10.1)
Stride Length (m)	1.23 (0.19)
Step Width (m)	0.08 (0.03)
Stance Time (ms)	667.3 (69.5)
Swing Time (ms)	409.9 (33.6)

[1] Sample size = 90

[2] Sample size = 91, amplitude normalized by baseline muscle activity

[3] Sample size = 91

Table 2.

Regression coefficients quantifying the relationship between spatial and temporal gait parameters and horizontal SCC VOR gain while controlling for age, cadence (stride length), height, and gender (relative to females as the reference value).

Spatial Variable	**VOR gain**	**Age**	**Cadence (steps/min)**	**Height (m)**	**Gender**
Stride Length (m)	β = −.379 * p = 0.004	β = −0.0036 * p = 0.002	β = 0.005 p = 0.006	β = 0.0071 p = 0.007	β = −0.0052 p = 0.914
Stride Length SD (m)	β = −0.018 p = 0.017	β = 0.0003 * p < 0.001	β = −0.00003 p = 0.757	β = −0.0001 p = 0.407	β = 0.006 p = 0.033
Step Width (m)	β = 0.0041 p = 0.873	β = −0.0002 p = 0.406	β = −0.0008 p = 0.027	β = −0.0003 p = 0.555	β = 0.0034 p = 0.724
Step Width SD (m)	β = −0.0065 p = 0.156	β = 0.0001 * p = 0.001	β = −0.00003 p = 0.693	β = 0.00005 p = 0.613	β = −0.0023 p = 0.176
Temporal Variables	**VOR gain**	**Age**	**Stride Length (m)**	**Height (m)**	**Gender**
Stance Time (ms)	β = −158.4 * p = 0.003	β = −0.42 p = 0.375	β = −159.2 * p < 0.001	β = 1.5 p = 0.166	β = 17.2 p = 0.377
Stance Time SD (ms)	β = −2.9 p = 0.554	β = 0.06 p = 0.215	β = −25.4 * p < 0.001	β = 0.05 p = 0.621	β = 3.4 p = 0.064
Swing Time (ms)	β = −68.4 p = 0.009	β = −0.02 p = 0.932	β = −17.2 p = 0.411	β = 1.5 p = 0.006	β = −0.67 p = 0.944
Swing Time SD (ms)	β = −2.7 p = 0.535	β = 0.08 p = 0.039	β = −23.6 * p < 0.001	β = 0.12 p = 0.162	β = −0.14 p = 0.933
Cadence	β = 22.4 * p = 0.003	β = 0.03 p = 0.611	β = 17.0 p = 0.006	β = −0.34 p = 0.029	β = −1.5 p = 0.599

* indicates significant relationships at $p < 0.0056$. $n = 90$.

Table 3.

Regression coefficients quantifying the relationship between spatial and temporal gait parameters and saccular function (*present* function as reference value) while controlling for age, cadence (stride length), height, and gender (relative to females as the reference value).

Spatial Variables	**Saccular Function**	**Age**	**Cadence (steps/min)**	**Height (m)**	**Gender**
Stride Length (m)	β = −0.0013 p = 0.811	β = −0.0040 p = 0.001	β = 0.004 p = 0.022	β = 0.0084 * p = 0.002	β = −0.0031 p = 0.533
Stride Length SD (m)	β = −0.0019 p = 0.536	β = 0.0003 * p < 0.001	β = −0.0001 p = 0.243	β = −0.0001 p = 0.483	β = 0.0055 p = 0.040
Step Width (m)	β = 0.0067 p = 0.507	β = 0.0002 p = 0.304	β = −0.0004 p = 0.174	β = 0.0003 p = 0.596	β = 0.0063 p = 0.474
Step Width SD (m)	β = 0.0008 p = 0.643	β = 0.00009 p = 0.022	β = −0.00009 p = 0.106	β = 0.00009 p = 0.313	β = −0.0034 p = 0.031
SD (m)	p = 0.643	p = 0.022	p = 0.106	p = 0.313	p = 0.031
Temporal Variables	**Saccular Function**	**Age**	**Stride Length (m)**	**Height (m)**	**Gender**
Stance Time (ms)	β = −4.1 p = 0.855	β = −0.19 p = 0.706	β = −144.1 * p < 0.001	β = 2.9 p = 0.008	β = −3.5 p = 0.860
Stance Time SD (ms)	β = 1.6 p = 0.408	β = 0.04 p = 0.358	β = −21.2 * p < 0.001	β = 0.10 p = 0.269	β = 1.9 p = 0.262
Swing Time (ms)	β = −1.3 p = 0.904	β = 0.05 p = 0.836	β = 4.06 p = 0.803	β = 1.94 * p < 0.001	β = 9.3 p = 0.320
Swing Time SD (ms)	β = 0.95 p = 0.591	β = 0.09 p = 0.034	β = −20.8 * p < 0.001	β = 0.16 p = 0.055	β = −0.69 p = 0.658
Cadence	β = 1.03 p = 0.748	β = 0.001 p = 0.979	β = 13.4 * p = 0.022	β = −0.53 * p = 0.001	β = 1.5 p = 0.609

* indicates significant relationships at p < 0.0056. n = 102.

Table 4.

Regression coefficients quantifying the relationship between spatial and temporal gait parameters and utricular function (*present* function as reference value) while controlling for age, cadence (stride length), height, and gender (relative to females as the reference value).

Spatial Variables	**Utricular Function**	**Age**	**Cadence (steps/min)**	**Height (m)**	**Gender**
Stride Length (m)	β = −0.088 p = 0.073	β = −0.0033 p = 0.007	β = 0.0041 p = 0.015	β = 0.0093 * p = 0.001	β = −0.034 p = 0.461
Stride Length SD (m)	β = −0.0003 p = 0.922	β = 0.0003 * p < 0.001	β = −0.0001 p = 0.232	β = −0.0001 p = 0.497	β = 0.0060 p = 0.029
Step Width (m)	β = 0.0001 p = 0.989	β = −0.0002 p = 0.391	β = −0.0004 p = 0.204	β = −0.0003 p = 0.505	β = 0.0079 p = 0.345
Step Width SD (m)	β = 0.0011 p = 0.500	β = 0.0001 p = 0.016	β = −0.0001 p = 0.060	β = 0.00004 p = 0.671	β = −0.0026 p = 0.095
Temporal Variables	**Utricular Function**	**Age**	**Stride Length (m)**	**Height (m)**	**Gender**
Stance Time (ms)	β = −36.4 p = 0.068	β = 0.11 p = 0.825	β = −150.3 * p < 0.001	β = 3.3 * p = 0.002	β = −5.0 p = 0.788
Stance Time SD (ms)	β = 0.09 p = 0.958	β = 0.06 p = 0.223	β = −21.1 * p < 0.001	β = 0.12 p = 0.217	β = 1.6 p = 0.341
Swing Time (ms)	β = −15.9 p = 0.093	β = 0.21 p = 0.378	β = 1.81 p = 0.923	β = 2.04 * p < 0.001	β = −8.1 p = 0.357
Swing Time SD (ms)	β = −2.4 p = 0.143	β = 0.11 p = 0.006	β = −21.3 * p < 0.001	β = 0.20 p = 0.019	β = −1.1 p = 0.486
Cadence	β = 4.9 p = 0.087	β = −0.04 p = 0.572	β = 14.1 p = 0.015	β = −0.57 * p < 0.001	β = 1.5 p = 0.584

* indicates significant relationships at p < 0.0056. n = 105.