JAMA Otolaryngology–Head & Neck Surgery | Original Investigation

Characterization of Head-Trunk Coordination Deficits After Unilateral Vestibular Hypofunction Using Wearable Sensors

Serene S. Paul, PhD, BAppSc(Phty)(Hons); Leland E. Dibble, PhD, DPT; Raymond G. Walther, MSME; Clough Shelton, MD; Richard Klaus Gurgel, MD; Mark E. Lester, PhD, DPT

IMPORTANCE Individuals with vestibular hypofunction acutely restrict head motion to reduce symptoms of dizziness and nausea. This restriction results in abnormal decoupling of head motion from trunk motion, but the character, magnitude, and persistence of these deficits are unclear.

OBJECTIVE To use wearable inertial sensors to quantify the extent of head and trunk kinematic abnormalities in the subacute stage after resection of vestibular schwannoma (VS) and the particular areas of deficit in head-trunk motion.

DESIGN, SETTING, AND PARTICIPANTS This cross-sectional observational study included a convenience sample of 20 healthy adults without vestibular impairment and a referred sample of 14 adults 4 to 8 weeks after resection of a unilateral VS at a university and a university hospital outpatient clinic. Data were collected from November 12, 2015, through November 17, 2016.

EXPOSURES Functional gait activities requiring angular head movements, including items from the Functional Gait Assessment (FGA; range, 1-30, with higher scores indicating better performance), the Timed Up & Go test (TUG; measured in seconds), and a 2-minute walk test (2MWT; measured in meters).

MAIN OUTCOMES AND MEASURES Primary outcomes included peak head rotation amplitude (in degrees), peak head rotation velocity (in degrees per second), and percentage of head-trunk coupling. Secondary outcomes were activity and participation measures including gait speed, FGA score, TUG time, 2MWT distance, and the Dizziness Handicap Inventory score (range, 0-100, with higher scores indicating worse performance).

RESULTS A total of 34 participants (14 men and 20 women; mean [SD] age, 39.3 [13.6] years) were included. Compared with the 20 healthy participants, the 14 individuals with vestibular hypofunction demonstrated mean (SD) reduced head turn amplitude (84.1° [15.5°] vs 113.2° [24.4°] for FGA-3), reduced head turn velocities (195.0°/s [75.9°/s] vs 358.9°/s [112.5°/s] for FGA-3), and increased head-trunk coupling (15.1% [6.5%] vs 5.9% [5.8%] for FGA-3) during gait tasks requiring angular head movements. Secondary outcomes were also worse in individuals after VS resection compared with healthy individuals, including gait speed (1.09 [0.27] m/s vs 1.47 [0.22] m/s), FGA score (20.5 [3.6] vs 30.0 [0.2]), TUG time (10.9 [1.7] s vs 7.1 [0.8] s), 2MWT (164.8 [37.6] m vs 222.6 [26.8] m), and Dizziness Handicap Inventory score (35.4 [20.7] vs 0.1 [0.4]).

CONCLUSIONS AND RELEVANCE With use of wearable sensors, deficits in head-trunk kinematics were characterized along with a spectrum of disability in individuals in the subacute stage after VS surgery compared with healthy individuals. Future research is needed to fully understand how patterns of exposure to head-on-trunk movements influence the trajectory of recovery of head-trunk coordination during community mobility.

JAMA Otolaryngol Head Neck Surg. doi:10.1001/jamaoto.2017.1443
Published online August 31, 2017.

Author Affiliations: Department of Physical Therapy and Athletic Training, University of Utah, Salt Lake City (Paul, Dibble, Walther); Faculty of Health Sciences, The University of Sydney, Sydney, Australia (Paul); Otolaryngology Division, School of Medicine, University of Utah, Salt Lake City (Shelton, Gurgel); Doctoral Program in Physical Therapy, US Army–Baylor University, Ft Sam Houston, Texas (Lester).

Corresponding Author: Leland E. Dibble, PhD, DPT, Department of Physical Therapy and Athletic Training, University of Utah, 520 Wakara Way, Salt Lake City, UT 84108 (lee.dibble@hsc.utah.edu).

Vestibular schwannomas (VSs) are benign, slow-growing tumors with an estimated incidence in the United States of approximately 1.09 per 100 000 persons.[1] The VS and treatment of the tumor with microsurgical removal or stereotactic radiation therapy result in unilateral vestibular hypofunction.[2] The resultant disruption of unilateral afferent stimuli to the vestibular centers of the central nervous system produces a well-characterized constellation of signs and symptoms and a negative effect on quality of life.[3] These vestibular effects include but are not limited to gaze instability (oscillopsia, especially if bilateral vestibular dysfunction occurs), vertigo, and postural and gait instability.[4]

During gait, individuals without vestibular deficits actively dissociate their head movement from their trunk.[5] In contrast, individuals with unilateral peripheral vestibular deficits acutely constrain their head movements relative to their trunk to reduce symptoms of oscillopsia, dizziness, and nausea. Such symptoms may lead to alterations in head movements and/or the loss of normal decoupling of head motion from trunk motion while walking, with the extent of loss varying with the severity of hypofunction.[6,7] Nevertheless, compared with individuals with normal vestibular function, people with vestibular deficits reorganize their head movements differently and sometimes less efficiently when performing gait and postural activities. In addition, these alterations in movements are not limited to the head and trunk.[6] Previous research has documented reduced gait speed, increased body sway, and slower performance of functional movement tasks.[7-10]

Basic science research[11] and current vestibular rehabilitation meta-analyses[12] and clinical guidelines[13] emphasize the critical nature of adequately dosed rehabilitation demands on gaze stability through head movement to enhance vestibular adaptation in individuals with acute vestibular hypofunction. For this reason, a better understanding of the nature of head movement alterations after unilateral vestibular hypofunction is needed. The relatively recent availability of wearable inertial sensors provides a previously unavailable opportunity to examine head and trunk movements in less constrained settings during challenging gait activities.[14] The identification of the extent and characteristics of head movement kinematic changes during the recovery from VS surgery could help to provide clarity regarding the sufficient dosage (frequency and intensity; ie, velocity) of head movements that might be necessary for rehabilitation to facilitate recovery toward premorbid levels of function.

We sought to examine and compare the character and magnitude of alterations in head kinematics and head-trunk coordination in patients with surgically induced unilateral vestibular hypofunction and in healthy individuals without such deficits. To quantify multiple domains of disability, we used wearable sensors during dynamic gait activities that required head movement and standardized clinical measures of gait function and dizziness. Our primary hypothesis was that individuals at 6 weeks after VS surgery would demonstrate altered head kinematics and head-trunk coupling compared with neurologically healthy individuals. Our secondary hypothesis was that patients with VS would also have deficits in gait speed and dynamic stability during gait and increased dizziness compared with healthy individuals.

Key Points

Question What is the extent of head movement and head-trunk coordination deficits in individuals 4 to 8 weeks after surgical resection of a vestibular schwannoma?

Findings In this cross-sectional study, 14 individuals with vestibular hypofunction demonstrated significantly reduced head turn amplitude, reduced head turn velocities, and increased head-trunk coupling during gait tasks requiring angular head movements compared with 20 neurologically healthy individuals.

Meaning At 4 to 8 weeks after vestibular schwannoma resection, patients demonstrated incomplete recovery of gait, dynamic stability, head movement, and head-trunk coordination, suggesting that early referral for vestibular rehabilitation for these individuals may be beneficial.

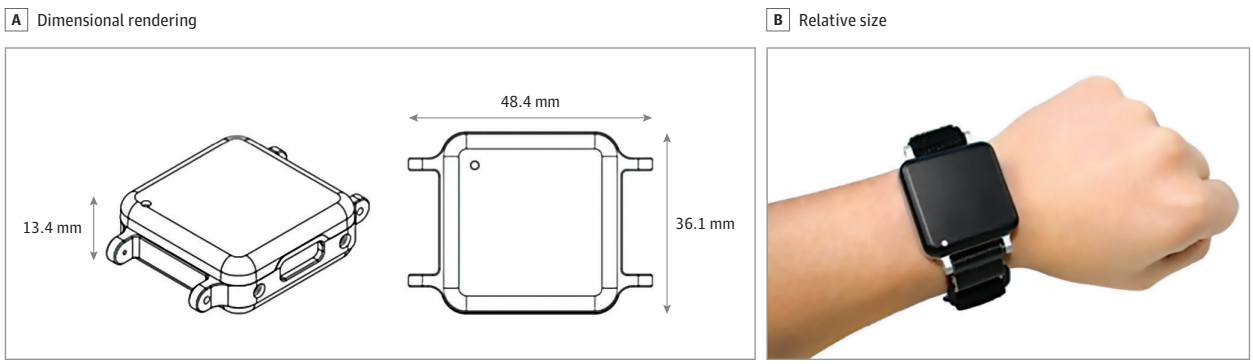
Methods

We used a cross-sectional design in this observational study. Individuals who had undergone surgical resection of a VS (via retrosigmoid, translabyrinthine, or middle fossa approaches) and healthy individuals without vestibular deficits were recruited for this study from November 12, 2015, through November 17, 2016. Participants were eligible if they were aged 18 to 70 years, were able to walk unaided, and had no surgery or injury of the lower extremity within the past 12 months. Individuals with vestibular hypofunction were included if they had undergone resection of VS within the past 2 months. Individuals using vestibular suppressants or who had unstable medical conditions that would interfere with the individual's ability to participate in the study procedures were excluded. Healthy individuals were also eligible if they had no central or peripheral nervous system disease and no history of central or peripheral nervous system vestibular disease.

This study conforms to STROBE reporting guidelines[15] and was approved by the institutional review board of the University of Utah, Salt Lake City. All participants gave written informed consent before data collection, and demographic data and all outcomes were deidentified and input into an electronic spreadsheet that was used for analysis.

Although previous research has documented deficits after VS surgery, studies have often used retrospective cohorts[16-18] or have been limited in the spectrum of outcomes that they have measured.[19-21] To address this gap, in this study we characterized disability using the 3 domains of disablement outlined by the World Health Organization's International Classification of Function[22] (ie, body structure and function, activity, and participation). The body structure and function domain was characterized by our primary outcome of head-trunk coordination using kinematics. The activity domain was characterized by 1 set of secondary outcomes (ie, clinical gait and postural abilities). The participation domain was characterized by the remaining secondary outcome (ie, dizziness). Demographic data, including age, body mass index, angular vestibular ocular reflex gain obtained from head impulse testing, and dynamic visual acuity were gathered for all participants.

Figure. Wearable Sensors



Images of Opal sensors are adapted and used with permission of APDM, Inc.

Participants performed a series of standardized dynamic gait tasks in a corridor of a university building or university hospital while wearing a wireless inertial sensor (Opal sensors; APDM, Inc) (**Figure**) on their forehead, sternum, and waist. The task specifics and head-trunk movement requirements of the Functional Gait Assessment (FGA; range, 1-30, with higher scores indicating better performance),[23] the Timed Up & Go test (TUG; measured in seconds),[24] and the 2-minute walk test (2MWT; measured in meters) are summarized in **Table 1**. Each wearable sensor weighed less than 25 g and contained an accelerometer, gyroscope, and magnetometer. The monitors wirelessly synchronized with each other, sampled at 128 Hz, and had 8 GB of on-monitor storage. These capabilities allowed us to collect data from all gait tasks without the need to remove the monitors. The International Classification of Function body structure and function outcomes of interest (ie, primary outcomes) included peak head rotation amplitude (in degrees), peak head rotation velocity (in degrees per second),[25] and percentage of head-trunk coupling for the 4 specific tasks.

A custom-written Matlab algorithm (Mathworks) was used to derive the primary outcomes from sensor data (S.S.P., R.G.W., Ethan A. Beseris, L.E.D., amd M.E.L.; unpublished data; study completed October 31, 2016). Raw data were filtered using a 6-Hz low-pass filter. Mean head and trunk rotational velocity signals from normal walking trials (FGA-1) of healthy participants were calculated to determine a noise value for each signal, which was used to identify the beginning and end of head turns and of trunk turns greater than the usual degree of trunk rotations during gait. Turns for all participants commenced at the point where the rotational velocity exceeded 3 times the relevant noise value, whereas turns ended at the point where the rotational velocity decreased below the relevant noise value or where the rotational velocity changed direction, whichever occurred earlier. The amplitude of each turn was then determined by numerical integration. Turns were excluded if the total duration of the turn was less than one-sixth of a second. When 2 subsequent turns in the same direction occurred within one-third of a second of each other, if both turns had amplitudes of at least 5°, a single turn was calculated by summing the amplitudes, whereas if either turn had an amplitude of less than 5°, only the larger turn was included for analysis. Peak head rotation amplitude of valid turns was determined by the area under the curve (AUC) of the signal[26] obtained from the head sensor from head turn commencement to head turn completion for each turn. Peak head rotation velocity was determined as the maximum velocity value obtained from the head sensor between the start and end of turns in each direction. An equivalent AUC from the trunk sensor was also calculated for the period between head turn commencement and completion; the AUC from the trunk sensor was divided by the head sensor's AUC to arrive at the amount of head-trunk coupling. Head-trunk coupling percentages close to 1% indicated that the head moved independently of the trunk, percentages close to 100% indicated en bloc turns, and percentages greater than 100% indicated that the participant rotated their trunk to a greater extent than their head. All tasks except FGA-5 involved multiple turns; thus, the mean value for each individual for each task was used in analysis.

Secondary outcomes in the International Classification of Function activity domain included gait speed (obtained from FGA-1), the overall FGA score, TUG time, and 2MWT distance. Faster gait speeds, higher FGA scores, and longer 2MWT distances indicate better performance, whereas longer TUG times indicate poor performance. The secondary outcome in the International Classification of Function participation domain was the Dizziness Handicap Inventory,[27] for which higher scores indicate a greater degree of handicap (range, 0-100).

Statistical Analysis

A sample size of 13 per group was determined to be sufficient to detect a 23% between-group difference in head-trunk coordination, assuming an SD of 0.38 in healthy participants, 10% equipment malfunction rate, α of .05, and power of 0.80.[6] Differences in outcomes between individuals with and without vestibular hypofunction were compared using separate independent-samples *t* tests or Mann-Whitney tests for outcomes with nonnormal distributions or when the assumption of homogeneity of variance was violated. The Bonferroni-adjusted critical α was no greater than .003. Effect sizes were reported using Cohen *d*, with absolute values of 0 to 0.2 indicating a small effect; 0.3 to 0.5, a medium effect; 0.6 to 0.8, a large effect; and 0.9 or greater, a very large effect.[28] Missing data were reported as such.

Table 1. Dynamic Gait Tasks and the Head and Trunk Requirements Inherent to Each Task

Gait Task	Task Specifics	Head and Trunk Movements Required
Forward gait with horizontal head turns (FGA-3)	Forward walking at self-selected pace for a 6-m path with horizontal head turns alternating to right and then left every 3 steps	Horizontal (yaw plane) angular rotation of the head; decoupling of head and trunk rotation to continue forward walking
Forward gait and pivot turn (FGA-5)	Forward walking at self-selected pace; rapid 180° turn and stop on verbal command while maintaining stability	Horizontal (yaw plane) angular rotation of the head and trunk; coupling of head and trunk rotation to accomplish 180° body turn
Timed Up & Go test	Stand up from sitting, walk out 3 m at self-selected pace, turn 180°, walk back to chair, turn 180°, and sit down	Horizontal (yaw plane) angular rotation of the head and trunk; coupling of head and trunk rotation to accomplish each 180° body turn
2-min Walk test	Forward walking on 17-m path with 180° turns at each end; objective is to walk as far as possible in 2 min	Horizontal (yaw plane) angular rotation of the head and trunk; coupling of head and trunk rotation to accomplish each 180° body turn

Abbreviation: FGA, Functional Gait Assessment.

Table 2. Participant Characteristics by Groups

Characteristic	Study Group[a]	
	Patients With Vestibular Hypofunction (n = 14)	Healthy Individuals (n = 20)
Age, y	47.2 (13.1) [23 to 66]	33.7 (11.6) [17 to 65]
Sex, No. (%)		
Male	5 (36)	9 (45)
Female	9 (64)	11 (55)
BMI	28.9 (5.1) [19.6 to 38.6]	22.9 (2.7) [19.4 to 28.3]
DVA, line difference	−4.1 (1.9) [−8 to −1]	−1.7 (1.5) [−6 to 1]
aVOR gain, ratio[b]		
L/affected horizontal	0.34 (0.11) [0.18 to 0.56]	0.92 (0.14) [0.74 to 1.43]
R/unaffected horizontal	0.85 (0.06) [0.76 to 0.93]	0.99 (0.11) [0.79 to 1.34]
L/affected anterior[c,d]	0.36 (0.19) [0.13 to 0.82]	0.90 (0.14) [0.64 to 1.10]
R/unaffected anterior[e]	0.83 (0.15) [0.54 to 1.13]	0.98 (0.16) [0.62 to 1.22]
L/affected posterior[f]	0.42 (0.17) [0.17 to 0.74]	0.90 (0.11) [0.68 to 1.09]
R/unaffected posterior[g]	0.73 (0.13) [0.47 to 0.95]	0.85 (0.11) [0.65 to 1.04]

Abbreviations: aVOR, angular vestibulo-ocular reflex (normal values, 0.8- 1.0); BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); DVA, dynamic visual acuity; L, left side; R, right side.

[a] Unlesss otherwise indicated, data are expressed as mean (SD) [range]. Missing values are attributable to removal of noisy data after visual inspection of head impulse test results.

[b] Described by L or R for healthy individuals and by affected (ie, with a lesion) or unaffected sides based on surgery.

[c] Includes 16 healthy individuals.

[d] Includes 13 patients with vestibular hypofunction.

[e] Includes 18 healthy individuals.

[f] Includes 19 healthy individuals.

[g] Includes 17 healthy individuals.

Results

We recruited 20 healthy individuals and 17 individuals with VS. One individual with VS had prion disease and was excluded, whereas 2 individuals with VS developed postoperative complications and withdrew before data collection. The final sample included 20 healthy individuals and 14 individuals with unilateral vestibular hypofunction (14 men and 20 women; mean [SD] age, 39.3 [13.6] years). Participant characteristics are described in **Table 2**. Individuals with vestibular hypofunction underwent VS resection a mean (SD) of 44 (6) days after resection of VS and had a range of tumor sizes (mean [SD], 15.1 [8.4] mm). Nine individuals (64%) had a left VS resection, whereas 5 (36%) had a right VS resection.

Primary Outcomes

Compared with healthy individuals, individuals with vestibular hypofunction demonstrated reduced mean (SD) head turn amplitude (84.1° [15.5°] vs 113.2° [24.4°]; Cohen *d*, 1.4; 95% CI, 0.6-2.1) and increased head-trunk coupling (15.1% [6.5%] vs 5.9% [5.8%]; Cohen *d*, −1.5; 95% CI, −2.3 to −0.7) during gait tasks requiring head turns (FGA-3), with very large effect sizes. Compared with healthy individuals, those with vestibular hypofunction also had slower head rotation velocities for FGA-3 (195.0°/s [75.9°/s] vs 358.9°/s [112.5°/s]; Cohen *d*, 1.7; 95% CI, 0.8-2.4), FGA-5 (232.2°/s [64.8°/s] vs 348.5°/s [98.8°/s]; Cohen *d*, 1.3; 95% CI, 0.6-2.1), TUG (155.8°/s [34.6°/s] vs 242.6°/s [47.2°/s]; Cohen *d*, 2.0; 95% CI, 1.2-2.9), and 2MWT (168.7°/s [36.9°/s] vs 220.4°/s [52.2°/s]; Cohen *d*, 1.1; 95% CI, 0.4-1.8), with very large effect sizes. No other body and structure outcome was statistically significant, although effect sizes for head turn amplitude were large in the TUG test (Cohen *d*, 0.9; 95% CI, 0.2-1.7) but small for FGA-5 (Cohen *d*, −0.02; 95% CI, −0.7 to 0.7) and the 2MWT (Cohen *d*, 0.1; 95% CI, −0.6 to 0.8). Head-trunk coupling of all other tasks except for FGA-3 had medium effect sizes ranging from 0.3 (95% CI, −0.4 to 1.0) for the 2MWT to 0.4 for the TUG test (95% CI, −0.2 to 1.1) and FGA-5 (95% CI, −0.3 to 1.0) (**Table 3**).

Secondary Outcomes

For activity domain outcomes, individuals with vestibular hypofunction demonstrated reduced gait speed (FGA-1) (1.09 [0.27] m/s vs 1.47 [0.22] m/s; Cohen *d*, 1.6; 95% CI, 0.8-2.4), reduced walking distance (2MWT) (164.8 [37.6] m vs 222.6 [26.8] m; Cohen *d*, 1.8; 95% CI, 1.0-2.6), and impaired stabil-

Table 3. Head Kinematics and Head-Trunk Coordination in Healthy Individuals and Patients With Unilateral Vestibular Hypofunction

Outcome	Study Group, Mean (SD)		Between-Group Effect Size, Mean Cohen *d* (95% CI)[a]
	Patients With Vestibular Hypofunction (n = 14)	Healthy Individuals (n = 20)	
Body and Structure Domain			
FGA-3			
Head rotation amplitude, °	84.1 (15.5)	113.2 (24.4)	1.4 (0.6 to 2.1)[b]
Head rotation velocity, °/s	195.0 (75.9)	358.9 (112.5)	1.7 (0.8 to 2.4)[b]
Head-trunk coupling, %	15.1 (6.5)	5.9 (5.8)	−1.5 (−2.3 to −0.7)[b,c]
FGA-5			
Head rotation amplitude, °	155.2 (12.3)	154.9 (19.1)	−0.02 (−0.7 to 0.7)[c]
Head rotation velocity, °/s	232.2 (64.8)	348.5 (98.8)	1.3 (0.6 to 2.1)[b,c]
Head-trunk coupling, %	104.5 (12.2)	109.5 (15.5)	0.4 (−0.3 to 1.0)
TUG			
Head rotation amplitude, °	156.1 (11.3)	166.5 (10.8)	0.9 (0.2 to 1.7)[c]
Head rotation velocity, °/s	155.8 (34.6)	242.6 (47.2)	2.0 (1.2 to 2.9)[b]
Head-trunk coupling, %	98.8 (9.6)	102.5 (6.8)	0.4 (−0.2 to 1.1)[c]
2MWT			
Head rotation amplitude, °	156.2 (11.1)	157.3 (12.4)	0.1 (−0.6 to 0.8)[c]
Head rotation velocity, °/s	168.7 (36.9)	220.4 (52.2)	1.1 (0.4 to 1.8)[b]
Head-trunk coupling, %	103.5 (11.9)	106.5 (7.3)	0.3 (−0.4 to 1.0)[c]
Activity Domain			
Gait speed, m/s[d]	1.09 (0.27)	1.47 (0.22)	1.6 (0.8 to 2.4)[b]
FGA[e]	20.5 (3.6)	30.0 (0.2)	4.1 (2.4 to 5.7)[b,c]
TUG, s[f,g]	10.9 (1.7)	7.1 (0.8)	−3.0 (−4.2 to −1.8)[b,c]
2MWT, m	164.8 (37.6)	222.6 (26.8)	1.8 (1.0 to 2.6)[b]
Participation Domain			
DHI[h]	35.4 (20.7)	0.1 (0.4)	−2.7 (−3.9 to −1.4)[b,c]

Abbreviations: DHI, Dizziness Handicap Inventory; FGA, Functional Gait Assessment; TUG, Timed Up & Go test; 2MWT, 2-minute walk test.

[a] Negative Cohen *d* values indicate that the vestibular group had higher scores than the healthy group.

[b] Statistically significant between-group difference.

[c] Determined by the Mann-Whitney test owing to nonnormal distribution and/or lack of homogeneity of variance.

[d] Obtained from FGA-1.

[e] Scores range from 0 to 30, with higher scores indicating better performance.

[f] Higher scores indicate worse performance.

[g] Includes 19 healthy individuals (missing data from 1 participant owing to sensor malfunction on this test).

[h] Scores range from 0 to 100, with higher scores indicating worse dizziness.

ity during dynamic gait tasks, including the overall FGA score (20.5 [3.6] vs 30.0 [0.2]; Cohen *d*, 4.1; 95% CI, 2.4 to 5.7) and TUG time (10.9 [1.7] s vs 7.1 [0.8] s; Cohen *d*, −3.0; 95% CI, −4.2 to −1.8), compared with healthy individuals, with very large effect sizes. For the participation outcome, individuals with vestibular hypofunction demonstrated a substantially greater mean (SD) Dizziness Handicap Inventory score compared with healthy individuals, with a very large effect size (35.4 [20.7] vs 0.1 [04]; Cohen *d*, −2.7; 95% CI, −3.9 to −1.4) (Table 3).

Discussion

Head and trunk coordination and dynamic stability during activities of daily living are not constrained in the context of normal vestibular function. In contrast, acute deficits in gaze and postural stability and en bloc movement of the head and trunk are well recognized in the acute period after surgically induced unilateral peripheral vestibular hypofunction.[9,29] To examine the disability induced by unilateral vestibular hypofunction during the subacute period after VS surgery, we used wearable sensors to examine head movement kinematics and head-trunk coordination. In support of our a priori hypotheses, participants who had undergone VS surgery moved their head significantly more slowly, dissociated their head and trunk less, walked more slowly, were less stable during gait, and had greater dizziness compared with neurologically healthy individuals.

Is Habitual Self-Selected Activity Enough for Recovery?

Basic science research suggests that exposure to head movements is critical for recovery of gaze and postural stability.[11] Although standard postsurgical treatment of individuals who have undergone VS surgery consists of education about head movement, gait, and exercise without regular postsurgical vestibular rehabilitation,[7] our findings indicate that individuals at 6 weeks after surgery are not performing head movements and head-trunk decoupling in a manner similar to that of healthy individuals without vestibular dysfunction. Mijovic et al[25] used wearable sensors to examine head movements during gait and postural tasks and showed that individuals at more than 6 months after VS surgery generated lower angular pitch plane velocities during gait tasks. Together, these results raise the question as to whether individuals who have undergone VS surgery independently expose themselves to sufficient frequency, intensity, and velocity of head movements to induce the appropriate error signals (retinal slip, losses of center of mass stability) necessary to drive vestibulo-ocular and vestibulospinal adaptation toward premorbid levels of function.[30]

Persistence of Disability and Potential Targets for Rehabilitation

Acute unilateral vestibular hypofunction after surgery may create substantial mobility limitations and dizziness. When examined longitudinally, the natural history of these deficits appears to be a gradual reduction with a concomitant return to daily activities.[4] At a mean of 6 weeks after surgery, our participants with VS demonstrated reductions in gait speed and dynamic stability compared with healthy participants, suggesting that recovery at this point is incomplete. Our results are consistent with and add to the body of evidence indicating that alterations in voluntary movement strategies persist after peripheral vestibular hypofunction.[8,12,13,25] In the few prospective longitudinal studies that have examined the progression of the domains of disability after VS surgery,[29,31-33] the spectrum of outcome measures has been limited. Regardless, these studies demonstrate that deficits in static balance, gaze stabilization, and dizziness persist after VS surgery in a substantial percentage of individuals, although recovery of a certain proportion of premorbid functional levels can be expected.[13] Our demographic data showing reduced vestibular ocular reflex gain in response to passive head movements toward the affected side and reduced dynamic visual acuity are consistent with these previous studies. These measures of gaze stability, in addition to head-trunk kinematics, dynamic stability during gait, and the Dizziness Handicap Inventory, characterize the disability present in the VS group in our study at 6 weeks after surgery. The spectrum of disability present herein represents potential targets for vestibular rehabilitation.

Limitations

This study used a cross-sectional design and a spectrum of measures to quantify disability 6 weeks after VS surgery in a relatively small cohort who underwent VS resection using a variety of surgical approaches. Owing to previous research being equivocal regarding the influence of tumor size on postoperative signs and symptoms,[4] no control was exerted over tumor size. Although randomized clinical trials have characterized the efficacy of high dosages of gaze stabilization exercises,[12,13] clinicians generally have not had an objective means to measure the frequency, intensity, and duration of patients' movements during gait tasks. This study was, to our knowledge, the first to use a suite of wearable sensors to quantify the spatial components of head movements and head-trunk decoupling during dynamic gait tasks requiring head movement. Future studies should investigate the temporal aspects of head-trunk coordination in individuals with unilateral vestibular hypofunction. To increase participant recruitment and to constrain data analysis, we limited the tasks studied. In addition, our spectrum of outcome measures covered all 3 domains of disability as defined by the World Health Organization.[22] Future research should use such a spectrum of outcome measures and examine a broader range of community mobility tasks with longer data-gathering periods to determine the extent of head movement constraints over the course of hours or days. Ideally, participants would undergo testing from symptom onset before surgery, in the acute period after surgery, and for a longer postoperative follow-up.

Owing to this study's cross-sectional design and the comparison with healthy participants, no control was exerted over participants' activities in the first 6 weeks after surgery. The intergroup differences demonstrated in this study can be used to appropriately power future clinical trials. Such a clinical trial should randomly assign persons after VS surgery to a usual care group and an experimental group, testing participants immediately after VS surgery and following them up longitudinally until disability is minimized and their function plateaus.

Conclusions

A key component to recovery from peripheral vestibular deficits is the regular exposure to head movements that may induce gaze and postural stability errors and therefore facilitate recovery. The use of wearable sensors and clinical measures provided an objective means to document deficits across multiple domains of disability in individuals after surgically induced unilateral vestibular injury compared with healthy individuals. Future research is needed to fully understand the trajectory of recovery with and without vestibular rehabilitation and the potential benefits of gaze and postural stability exercises on head and trunk coordination during community mobility.

ARTICLE INFORMATION

Accepted for Publication: June 16, 2017.

Published Online: August 31, 2017. doi:10.1001/jamaoto.2017.1443

Author Contributions: Dr Paul had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.
Study concept and design: Paul, Dibble, Lester.
Acquisition, analysis, or interpretation of data: All authors.
Drafting of the manuscript: Paul, Dibble.
Critical revision of the manuscript for important intellectual content: All authors.
Statistical analysis: Paul, Dibble.
Obtained funding: Dibble, Lester.
Administrative, technical, or material support: All authors.
Study supervision: Dibble, Lester.

Conflict of Interest Disclosures: All authors have completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Dr Paul reports receiving grants from the American Parkinson Disease Association, Parkinson's NSW, and The University of Sydney Industry Engagement Scheme outside the submitted work. Dr Dibble reports receiving grants from the US Army Advanced Medical Technology Initiative and personal fees from the National Multiple Sclerosis Society during the conduct of the study. Dr Shelton reports receiving grants from Cochlear Americas and the Neurofibromatosis Program, US Department of Defense, outside the submitted work. Dr Lester reports receiving grants from the US Army Medical Department Advanced Medical Technology Initiative during the conduct of the study. No other disclosures were reported.

Funding/Support: This study was supported by an Advanced Medical Technology Initiative grant 2015-2016 from the US Army.

Role of the Funder/Sponsor: The sponsor had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; preparation, review, or approval of the manuscript; and decision to submit the manuscript for publication.

Additional Contributions: Sherry Hogge, School of Medicine, University of Utah, did not receive compensation for her assistance in recruiting individuals with vestibular schwannoma. Ethan Beseris, Alicia Dibble, Kirsten Gonski, Jane Saviers-Steiger, Jaclyn Hill, BS, and Blake Rowinski, DPT, University of Utah, received compensation as research assistants for assisting with data collection.

REFERENCES

1. Kshettry VR, Hsieh JK, Ostrom QT, Kruchko C, Barnholtz-Sloan JS. Incidence of vestibular schwannomas in the United States. *J Neurooncol*. 2015;124(2):223-228.

2. Ansari SF, Terry C, Cohen-Gadol AA. Surgery for vestibular schwannomas: a systematic review of complications by approach. *Neurosurg Focus*. 2012;33(3):E14.

3. Robinett ZN, Walz PC, Miles-Markley B, Moberly AC, Welling DB. Comparison of long-term quality-of-life outcomes in vestibular schwannoma patients. *Otolaryngol Head Neck Surg*. 2014;150(6):1024-1032.

4. Saman Y, Bamiou DE, Gleeson M. A contemporary review of balance dysfunction following vestibular schwannoma surgery. *Laryngoscope*. 2009;119(11):2085-2093.

5. 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.

6. Cavanaugh JT, Goldvasser D, McGibbon CA, Krebs DE. Comparison of head- and body-velocity trajectories during locomotion among healthy and vestibulopathic subjects. *J Rehabil Res Dev*. 2005;42(2):191-198.

7. Allum JH, Adkin AL. Improvements in trunk sway observed for stance and gait tasks during recovery from an acute unilateral peripheral vestibular deficit. *Audiol Neurootol*. 2003;8(5):286-302.

8. Borello-France DF, Gallagher JD, Redfern M, Furman JM, Carvell GE. Voluntary movement strategies of individuals with unilateral peripheral vestibular hypofunction. *J Vestib Res*. 1999;9(4):265-275.

9. Parietti-Winkler C, Gauchard GC, Simon C, Perrin PP. Sensorimotor postural rearrangement after unilateral vestibular deafferentation in patients with acoustic neuroma. *Neurosci Res*. 2006;55(2):171-181.

10. Wilhelmsen K, Nordahl SH, Moe-Nilssen R. Attenuation of trunk acceleration during walking in patients with unilateral vestibular deficiencies. *J Vestib Res*. 2010;20(6):439-446.

11. Igarashi M, Levy JK, O-Uchi T, Reschke MF. Further study of physical exercise and locomotor balance compensation after unilateral labyrinthectomy in squirrel monkeys. *Acta Otolaryngol*. 1981;92(1-6):101-105.

12. McDonnell MN, Hillier SL. Vestibular rehabilitation for unilateral peripheral vestibular dysfunction. *Cochrane Database Syst Rev*. 2015;1:CD005397.

13. Hall CD, Herdman SJ, Whitney SL, et al. Vestibular rehabilitation for peripheral vestibular hypofunction: an evidence-based clinical practice guideline from the American Physical Therapy Association Neurology Section. *J Neurol Phys Ther*. 2016;40(2):124-155.

14. Horak F, King L, Mancini M. Role of body-worn movement monitor technology for balance and gait rehabilitation. *Phys Ther*. 2015;95(3):461-470.

15. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP; STROBE Initiative. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. *PLoS Med*. 2007;4(10):e296.

16. Tufarelli D, Meli A, Labini FS, et al. Balance impairment after acoustic neuroma surgery. *Otol Neurotol*. 2007;28(6):814-821.

17. El-Kashlan HK, Shepard NT, Arts HA, Telian SA. Disability from vestibular symptoms after acoustic neuroma resection. *Am J Otol*. 1998;19(1):104-111.

18. Levo H, Blomstedt G, Pyykkö I. Postural stability after vestibular schwannoma surgery. *Ann Otol Rhinol Laryngol*. 2004;113(12):994-999.

19. Herdman SJ, Schubert MC, Das VE, Tusa RJ. Recovery of dynamic visual acuity in unilateral vestibular hypofunction. *Arch Otolaryngol Head Neck Surg*. 2003;129(8):819-824.

20. Enticott JC, O'Leary SJ, Briggs RJ. Effects of vestibulo-ocular reflex exercises on vestibular compensation after vestibular schwannoma surgery. *Otol Neurotol*. 2005;26(2):265-269.

21. Cohen HS, Kimball KT, Jenkins HA. Factors affecting recovery after acoustic neuroma resection. *Acta Otolaryngol*. 2002;122(8):841-850.

22. Jette AM. Toward a common language for function, disability, and health. *Phys Ther*. 2006;86(5):726-734.

23. 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.

24. Podsiadlo D, Richardson S. The timed "Up & Go": a test of basic functional mobility for frail elderly persons. *J Am Geriatr Soc*. 1991;39(2):142-148.

25. Mijovic T, Carriot J, Zeitouni A, Cullen KE. Head movements in patients with vestibular lesion: a novel approach to functional assessment in daily life setting. *Otol Neurotol*. 2014;35(10):e348-e357.

26. McGarvie LA, MacDougall HG, Halmagyi GM, Burgess AM, Weber KP, Curthoys IS. The Video Head Impulse Test (vHIT) of semicircular canal function: age-dependent normative values of VOR gain in healthy subjects. *Front Neurol*. 2015;6:154.

27. Jacobson GP, Newman CW. The development of the Dizziness Handicap Inventory. *Arch Otolaryngol Head Neck Surg*. 1990;116(4):424-427.

28. Sullivan GM, Feinn R. Using effect size—or why the *P* value is not enough. *J Grad Med Educ*. 2012;4(3):279-282.

29. Batuecas-Caletrio A, Rey-Martinez J, Trinidad-Ruiz G, et al. Vestibulo-ocular reflex stabilization after vestibular schwannoma surgery: a story told by saccades. *Front Neurol*. 2017;8:15.

30. Schubert MC, Della Santina CC, Shelhamer M. Incremental angular vestibulo-ocular reflex adaptation to active head rotation. *Exp Brain Res*. 2008;191(4):435-446.

31. Parietti-Winkler C, Gauchard GC, Simon C, Perrin PP. Long-term effects of vestibular compensation on balance control and sensory organisation after unilateral deafferentation due to vestibular schwannoma surgery. *J Neurol Neurosurg Psychiatry*. 2010;81(8):934-936.

32. Parietti-Winkler C, Lion A, Frère J, Perrin PP, Beurton R, Gauchard GC. Prediction of balance compensation after vestibular schwannoma surgery. *Neurorehabil Neural Repair*. 2016;30(5):395-401.

33. Batuecas-Caletrio A, Santacruz-Ruiz S, Muñoz-Herrera A, Sousa P, Otero A, Perez-Fernandez N. Vestibular compensation after vestibular schwannoma surgery: normalization of the subjective visual vertical and disability. *Acta Otolaryngol*. 2013;133(5):475-480.