

An Assessment of Gait and Balance Deficits After Traumatic Brain Injury

Jeffrey R. Basford, MD, PhD, Li-Shan Chou, PhD, Kenton R. Kaufman, PhD, PE, Robert H. Brey, PhD, Ann Walker, MS, James F. Malec, PhD, Anne M. Moessner, RN, MSN, Allen W. Brown, MD

ABSTRACT. Basford JR, Chou L-S, Kaufman KR, Brey RH, Walker A, Malec JF, Moessner AM, Brown AW. An assessment of gait and balance deficits after traumatic brain injury. *Arch Phys Med Rehabil* 2003;84:343-9.

Objective: To assess the sensations of instability that many patients report after traumatic brain injury (TBI).

Design: A controlled study.

Setting: A motion analysis and vestibular and balance laboratory.

Participants: Twenty subjects, 10 with TBI and complaints of instability, and 10 without TBI.

Interventions: Balance and gait analysis.

Main Outcome Measures: Dizziness Handicap Inventory (DHI), caloric irrigation, optokinetic testing, Dix-Hallpike Test, posturography, and center of mass (COM) movement.

Results: Subjects were well matched in terms of age, height, weight, and gender. DHI scores of those with and without TBI differed significantly (32.2 ± 23.0 vs 0.2 ± 0.63 , $P < .001$). Caloric and optokinetic circularvection testing were abnormal only in subjects with TBI (8/10 and 4/10, respectively). Benign paroxysmal positioning vertigo was present in only 3 subjects with TBI, and this either resolved spontaneously ($n=1$) or was successfully treated ($n=2$). Composite posturography scores of those with and without TBI differed significantly (69.6 ± 35.8 vs 79.5 ± 40.5 , $P = .02$). Gait parameters also differed significantly between the groups ($P = .05$), with the subjects with TBI having lower anterior and posterior and higher medial and lateral COM displacements and velocities.

Conclusion: Patients' complaints of instability after TBI may have objective correlates and may be rectifiable. Balance and gait testing in these patients is warranted.

Key Words: Balance; Brain injuries; Gait; Rehabilitation; Vestibular function tests.

© 2003 by the American Congress of Rehabilitation Medicine and the American Academy of Physical Medicine and Rehabilitation

TRAUMATIC BRAIN INJURY (TBI) is a major cause of morbidity and mortality.^{1,2} Population-based statistics have been difficult to gather,^{3,4} but it is estimated that more than 1.5 million people in the United States receive a TBI each

year⁴ and that more than 400,000 of these injuries are severe enough to require hospitalization.^{4,5} Although catastrophic injuries, such as those that result in prolonged loss of consciousness (LOC), receive the most attention, it is well known that cognitive, affective, and behavioral functions can persist for years after what may be regarded as a "mild" TBI.^{6,7}

Subjective complaints that occur in the absence of physical findings after TBI are frequently difficult to assess. Nonetheless, because evaluation methods have improved, organic causes are often detected.^{3,8,9} Symptoms of impaired balance and altered coordination have been particularly troublesome, with as many as 30% of patients complaining of these problems after TBI.^{3,8,10}

This incidence may not be surprising inasmuch as effective coordination of activities and balance involves a complex interaction of the sensory, motor-programming, and musculoskeletal systems. Even minor impairments in integrating this information can lead to significant disability. For example, the sensory system's monitoring of the location of the body's center of mass (COM) and orientation must be effectively coordinated with motor responses if movements are to be performed in the right direction with the appropriate forces and proper latencies.¹¹

Balance research with people who have had a TBI has, for the most part, been limited to assessing postural sway during quiet standing or during standing with altered sensory inputs.¹¹⁻¹⁹ Evaluation in more dynamic settings, or in association with other symptomatic and psychometric assessments, has been limited. The investigations that have been performed have found that persons with TBI often have an increased reliance on visual input and tend to sway more (in both the anteroposterior [AP] and mediolateral [ML] directions) than control subjects who are without neurologic dysfunction.¹¹⁻¹⁹ It also seems that people who have TBI may not use their vestibular systems to resolve conflicts between the inputs from their visual and somatosensory systems as effectively as do people who have not had a similar injury.¹⁶

In summary, many people with TBI complain of problems with balance and instability that may not be apparent on clinical examination. In addition, there are findings, primarily from studies with the subject in a fixed location, suggesting that persons with TBI exhibit increased sway while standing and have difficulty processing sensory information. A comprehensive evaluation of the interaction of cognitive processing, vestibular function, and motion analysis does not seem to have been done. Given the frequency of complaints and the potential effect of balance and gait problems in this population, a study that assessed these factors seemed warranted.

This study had 2 goals. The first was to quantitatively assess the gait and dynamic balance systems of individuals with complaints of instability or imbalance after TBI that did not have an obvious neuromuscular origin. The second was to investigate the relation between these symptoms and pathophysiological findings.

From the Departments of Physical Medicine and Rehabilitation (Basford, Moessner, Brown) and Otorhinolaryngology (Brey), Motion Analysis Laboratory, Department of Orthopedic Surgery (Kaufman, Walker), and Section of Neuropsychology and Rehabilitation Psychology (Malec), Mayo Clinic and Foundation, Rochester, MN; and the Department of Exercise and Movement Science, University of Oregon, Eugene, OR (Chou).

No commercial party having a direct financial interest in the results of the research supporting this article has or will confer a benefit upon the author(s) or upon any organization with which the author(s) is/are associated.

Correspondence to Jeffrey R. Basford, MD, PhD, Dept of Physical Medicine and Rehabilitation, 200 SW Second St, Mayo Clinic and Foundation, Rochester, MN 55905. Reprints are not available.

0003-9993/03/8403-343\$30.00/0
doi:10.1053/apmr.2003.50034

METHODS

Subject Recruitment

This study was reviewed by the Mayo Clinic Institutional Review Board. After the protocol was approved, 6 men and 4 women, ranging in age from 18 to 65 years, who had had a TBI documented by their history and medical records (eg, a decreased Glasgow Coma Scale [GCS] score within 24h after initial admission; documented LOC), were recruited by physician referral and from the Mayo Clinic Traumatic Brain Injury Model Systems center. These subjects were required to be at least 3 months postinjury, be living in the community, and to have had normal gait and balance before being injured. In addition, they had to have complaints of dizziness or unsteadiness when walking, and they were also required to have a normal neurologic and musculoskeletal examination (see below). Cognitive, medical, or behavioral problems that would interfere with their participation were exclusion criteria. Ten healthy persons without a history of TBI were recruited from our institution by advertisement to serve as control subjects matched for age (± 5 y), gender, height (± 7.5 cm), and weight (± 14 kg).

The hospital records and radiology reports of all participants (ie, those with or without TBI) were reviewed. All subjects were assessed with the Tinetti Balance Assessment Scale²⁰ and were required to have normal neurologic (ie, normal strength, sensation [light touch, sharp, proprioception], coordination, gait) and musculoskeletal (posture, range of motion, limb length) clinical examinations. Each participant gave informed consent before entering the study and received \$100 at the end of their participation.

Experimental Design

All subjects underwent standardized neuropsychometric testing with a battery that included the Stroop test, the Trail-Making Test (TMT), beta mazes, the Controlled Oral Word Association Test, the Wechsler Memory Scale–Revised paragraph and visual reproduction memory tests (including both immediate and delayed recall trials), the Auditory Verbal Learning Test, and the Visuospatial Learning Test. These tests have demonstrated reliability and validity and were selected for their emphasis on cognitive flexibility, processing speed, and the ability to dual-task in addition to new learning and retention.²¹ A skilled psychometrist administered the tests. The results were analyzed by a board-certified clinical neuropsychologist (JFM).

Clinical Assessment

Tinetti Balance Assessment. The Tinetti Balance Assessment Scale is a validated,²⁰ simple clinical assessment of balance (eg, during quiet standing, on challenge, and with the eyes closed) and gait (eg, quality, speed, symmetry). Scores range from 0 to 28, with higher scores indicating improved performance. Healthy young individuals typically score 28, and even the healthy elderly may be expected to score above 24.^{22,23}

Dizziness Handicap Inventory. The Dizziness Handicap Inventory (DHI) is a standardized, validated 25-question instrument that is used to assess an individual's handicap resulting from symptoms of dizziness.²⁴ It is divided into 3 components that assess the physical (eg, Does looking up increase your problem?), emotional (eg, Because of your problem, do you feel frustrated?), and functional (eg, Because of your problem, do you have difficulty getting into or out of bed?) ramifications of a person's symptoms. Answers are graded 0

(never), 2 (sometimes), and 4 (always). The highest score possible is 100, with the physical, emotional, and functional components contributing 28, 36, and 36 points, respectively. Higher scores indicate a greater degree of impairment.

Dix-Hallpike Test. All subjects were tested for benign paroxysmal positioning vertigo (BPPV) with a Dix-Hallpike Test.²⁵ Testing procedures were standardized. The individual was taken from a sitting position with his/her head turned 45° to each the right and left, and then he/she was moved from a sitting to a supine position with the head hanging off the end of an examining table. The subject's eyes were monitored for nystagmus. (BPPV of the right posterior semicircular canal produces, from the tester's perspective, a counterclockwise torsional nystagmus, and BPPV of the left semicircular canal produces the opposite.²⁵) The examiner recorded whether nystagmus was present, as well as the direction the eyes moved when nystagmus was detected. The presence or absence of nystagmus was recorded as a dichotomous variable.

Caloric irrigation. A standardized bilateral, bithermal caloric irrigation procedure^a was used to assess vestibular function. Each external auditory canal was irrigated with 250mL of warm (44°C) and cool (30°C) water for 30 seconds. Electro-oculography was used to measure the slow phase velocity of the eye movements (nystagmus) generated in degrees per second. Peak values for the slow phase eye velocities were measured for each irrigation. Findings were compared with normative data used in our vestibular laboratory.²⁶ A unilateral weakness was defined as a velocity difference between the ears of $\geq 20\%$. If the total caloric response of 4 irrigations was $\leq 30^\circ/s$, the response was classified as a bilateral weakness. Subjects were also classified dichotomously as normal or abnormal for unilateral and bilateral weakness.

Optokinetic testing. Subjects were seated in a darkened room beneath a sphere that had an internal light source. The sphere was perforated with approximately 1300 small holes and projected spots of light on the walls of the room as it was rotated at 20°, 40°, and 60°/s. Two dependent variables were measured. The first, the velocity of the slow phase component of nystagmus, was measured in degrees per second by using electro-oculography. These velocities were compared with our laboratory's normative data²⁷ to determine whether there was a concomitant increase in velocity with stimulus rates of 20°, 40°, and 60°/s. The second dependent variable was the subjects' perceptions of movement. They were asked if they felt as if they were turning (circularvection) or if they perceived themselves to be stationary with the light spots moving around them (egocentric motion perception). If the moving light spots produced a sensation that he/she was rotating, the subject was graded as having a normal optokinetic reflex.²⁸ If a subject perceived the lights to be moving, he/she was assessed as having an abnormal optokinetic reflex.

Pure-tone hearing testing. Pure-tone hearing thresholds were measured with an ANSI calibrated audiometer (GSI 16 audiometer)^b in decibel hearing levels at .25, .5, 1.0, 2.0, 4.0, and 8.0kHz with Telephonic Dynamic Headphones (TDH-50)^b and a standard ascending and descending procedure.²⁹

Computerized dynamic posturography. Subjects were informed about the testing procedure and were tested barefoot on a computerized dynamic posturography platform^c while wearing a safety harness that was attached to an overhead bar. All participants underwent a sensory organization test (SOT) that assessed the 3 sensory components of balance (vision, proprioception, vestibular function) under 6 conditions: (1) eyes open, fixed support; (2) eyes closed, fixed support; (3) sway-referenced vision, fixed support; (4) sway-referenced support surface, eyes open; (5) eyes closed, sway-referenced support

surface; and (6) sway-referenced vision and support surface. Testing progressed from condition 1 to 6 and involved 3 consecutive 20-second trials for each condition. The dependent variable measured in each condition was the amount of sway compared with age-matched nondisabled subjects and a 12.5° cone of stability. Scores near 100 represent little sway, and scores near 0 represent excessive sway. Compensations such as touching a support surface or shifting the feet were considered “falls” and were graded 0.³⁰

Motion Analysis

Subjects wore a safety harness that was attached to a trolley on the ceiling; they were instructed to walk barefoot along a level, 10-m walkway at a comfortable self-selected speed. Twenty-seven reflective markers were placed on bony landmarks, and an 8-camera ExpertVison™ system^d was used to collect 3-dimensional marker trajectory data at a 60-Hz sampling rate. Three trials were performed by each subject. Data were recorded from all trials. Motion data were analyzed throughout a stride, that is, from the heel strike to heel strike of the same limb.

Our biomechanical model consisted of 13 body segments (4 upper extremity, 6 lower extremity, 1 each for the pelvis, trunk, and head) and was used to compute the COM for each body segment.³¹ The 3-dimensional trajectory of each body segment's COM was computed from the measured trajectories of markers placed on body landmarks in a manner that has produced gait determinants of sufficient repeatability to warrant their use in clinical gait studies.³² The mass, location of the COM, and moments of inertia of each individual body segment were derived from predictive equations and data in the literature.^{33,34} The 3-dimensional trajectory of the whole body's COM was computed from the weighted sum of the COMs from each body segment.³⁵⁻³⁷ The linear velocities of the whole body's COM were computed with the GCVSPL algorithm.³⁸

Data Analysis

Demographic data and Tinetti scores were assessed with 2-sample *t* tests. Psychometric data were also assessed with 2-sample *t* tests, but because analysis of that data was more complex, it is discussed in more detail in the neuropsychometric results section. DHI scores were analyzed with the nonparametric Wilcoxon 2-sample test. The Dix-Hallpike and optokinetic test results were studied with nonparametric statistics. Bithermal, bilateral caloric irrigation data were assessed by using a 2-factor analysis of variance (ANOVA) with repeated measures on 1 factor. The mean totals for the 4 caloric responses were analyzed with a 2-sample *t* test. Pure-tone hearing threshold measures were evaluated with a 2-factor ANOVA with repeated measures on 1 factor. Comparisons of the sensory organization posturography data were made by using separate 2-sample *t* tests (or ranked sum tests if significant departures from normality were observed) for each testing condition and for the composite score. A 1-way multivariate ANOVA was used for the statistical analysis of the gait parameters. The independent variable was group (subjects with TBI vs those without brain injury), and the dependent variables were the 3-dimensional (AP, ML, vertical) displacement and velocity of the COM.

RESULTS

Data were collected, as planned, on 10 subjects with TBI and their 10 matched controls. However, it was necessary to recruit 11 subjects for each group. One subject with TBI withdrew after completing only the initial neurologic screening exami-

Table 1: Subject Demographics

Variable	TBI Subjects (n=10)	Control Subjects (n=10)	P Value
Age (y)	40.9±11.3	41.2±11.4	.70*
Gender (men/women)	6/4	6/4	NA
Height (m)			
Men	1.71±.047	1.81±.06	.08*
Women	1.64±.044	1.69±.08	.07*
Weight (kg)			
Men	76.8±6.4	77.8±13.3	.81*
Women	77.3±21.5	71.8±10.8	.74*
BMI (kg/m ²)			
Men	26.3±2.4	23.7±3.0	.31*
Women	28.6±7.8	24.8±5.6	.56*
Severity of TBI			
Mild	4/19	NA	NA
Moderate	2/10	NA	NA
Severe	4/10	NA	NA
Mean duration of injury, y (range)	2.83 (.36–15.4)	NA	NA
Tinetti score	27.5±0.3	28.0±0	.14*

NOTE. Data, except for the severity of TBI and duration of injury, are mean ± standard deviation (SD).

Abbreviation: NA, not applicable.

* *t* test.

nation because of feelings of generalized anxiety and fatigue that she had had since she was injured. One control subject declined to continue after becoming nauseated during vestibular testing.

Demographics

The 2 groups were well matched in terms of gender, height, weight, and body mass index (BMI) (table 1). On the basis of their initial GCS score obtained from their medical records, 4 subjects with TBI had severe (GCS score, <9), 2 had moderate (GCS score, 9–12), and 4 had mild (GCS score, >12) brain injuries. Despite the range of initial injury severity, the 10 individuals had recovered well: 4 were living independently and were able to work at a reduced capacity (level 6 on the Glasgow Outcome Scale–Extended³⁹ [GOS-E]), and 5 were living and working independently with postconcussive symptoms (GOS-E level 7). One person required significant supervision in daily life (GOS-E level 4).

Neuromuscular examinations were, as required and defined by the inclusion criteria, unremarkable in the 2 groups. This uniformity extended to the Tinetti balance assessments, in which the groups with and without TBI differed insignificantly (27.5±0.3 vs 28±0, respectively, *P*=.14). Neuroimaging findings obtained during the chart review at admission to the study revealed cerebral hemorrhage or contusions compatible with TBI in 6 of 10 subjects with TBI. There were no images for any of the control subjects.

Neuropsychometric Testing

Neuropsychometric testing revealed that the subjects with TBI, although not markedly impaired, performed more poorly (ie, had lower scores) than did the controls (table 2). Student *t* tests were performed for the average scaled score across all tests for each subject, as well as for the scaled scores for each test. Specifically, the raw score for each test was converted to a scaled score (mean ± standard deviation [SD], 10±3) on the basis of the best available norms. The average scaled score

Table 2: Standardized Neuropsychometric Testing

Test	TBI Subjects (n=10)	Control Subjects (n=10)	P* Value
Paragraph immediate recall	7.66±2.81	10.98±3.24	.02
Paragraph delayed, % retained	8.41±2.99	11.08±1.28	.02
Designs immediate recall	10.24±3.34	13.65±1.19	.007
Designs delayed, % retained	6.91±2.63	10.39±1.74	.001
Word list learning	7.48±4.56	9.91±2.60	.16
Visuospatial learning	9.70±3.95	13.00±3.37	.06
Word fluency	10.50±3.60	13.30±2.16	.05
Mazes	8.50±3.54	12.00±2.45	.02
TMT-A	9.22±3.67	11.80±3.56	.13
TMT-B	8.41±2.57	12.07±2.22	.003
Stroop word	8.08±2.18	10.99±2.64	.02
Stroop color	7.35±3.41	10.63±3.20	.04
Stroop color-word	8.80±2.60	10.72±2.65	.12
Within-subject average	9.45±2.34	12.84±1.54	.001

NOTE. Values are mean ± SD.

* Unpaired *t* tests.

across neurocognitive measures was 9.45 for people with TBI, compared with 12.84 for controls ($P<.001$). Although there is some scatter, it is clear that the 10 subjects with TBI had mild cognitive residuals (particularly with immediate and delayed recall) relative to the control subjects, despite 9 patients having recovered to the level of independent living.

Balance and Vestibular Function

DHI scores were consistent with the subjects' complaints of unsteadiness and imbalance. Specifically, subjects with TBI scored significantly higher than did the control subjects (ie, had significantly more impairment) in the inventory's overall mean score (32.2 ± 23.0 vs 0.2 ± 0.6 , $P<.001$), as well as in all 3 domains (physical, emotional, functional) (see table 3).

Dix-Hallpike testing revealed that none of the control group members had evidence of BPPV. Three subjects with TBI had BPPV, but the difference in incidence did not reach statistical significance (table 3).

Caloric irrigation revealed vestibular impairment in 8 of 10 subjects with TBI (7 with unilateral weakness, ie, $\geq 20\%$ interear difference; 1 with bilateral weakness, ie, 4 irrigations totaling $\leq 30^\circ/s$). None of the 10 controls had vestibular impairment (table 3). Total caloric response (the sum of the warm and cool irrigations for each ear) was significantly weaker in the subjects with TBI than in the control group (mean, 85.7 ± 48.9 , 147.0 ± 71.0 , respectively, $P=.02$). ANOVA of the group means for slow phase peak eye velocity in subjects with (21.4 ± 15.7) and without (38.2 ± 23.0) TBI revealed significant differences between the groups ($P<.05$), indicating a weaker response from the subjects with a TBI. Post hoc multiple comparisons identified warm irrigations as significantly stronger responses than cool irrigations ($P<.001$).

Optokinetic testing revealed a trend toward an increased incidence of abnormalities in the subjects with TBI (table 3). The differences between the groups, however, did not reach statistical significance. The perception of circularvection (ie, the sense that the subject is turning when, in fact, he/she is sitting still and the light spots are moving) was normal in all control subjects. It was, however, abnormal in 4 of 10 ($P=.087$) subjects with TBI, who did not appreciate the sense of rotation (ie, circularvection) that most people feel when sitting in a darkened room with spots of light moving around them. Differences between the groups in terms of their slow phase eye velocities measured from the nystagmus generated by the optokinetic stimuli were even less pronounced: 2 control subjects and 4 subjects with TBI failed to meet our laboratory norms.²⁷

Figure 1 summarizes the mean pure-tone hearing thresholds for the right and left ears of the 2 groups. Although a trend was present, that is, the mean pure-tone thresholds for the subjects with TBI were always poorer than they were for the control subjects, the group differences did not reach statistical significance (right audiogram: subjects with TBI, 26.5 ± 25.7 ; control subjects, 12 ± 9.6 ; $P=.06$; left audiogram: subjects with TBI, 22.4 ± 17.8 ; control subjects, 15.7 ± 15.4 ; $P=.23$). Our sample size did not provide the power needed to show significance for these measures of hearing.

Table 3: Balance and Vestibular Data

Variable	TBI Subjects (n=10)	Control Subjects (n=10)	P
DHI scores	32.2±23.0	0.2±0.6	.001*
Physical domain (maximum score, 28)	10.4±5.3	0.0±0.0	<.001*
Emotional domain (maximum score, 36)	10.2±9.6	0.0±0.0	.006*
Functional domain (maximum score, 36)	11.6±10.7	0.2±0.6	<.001*
BPPV	3/10	0/10	.211 [†]
Unilateral abnormality [‡]	2/10	0/10	.474 [†]
Bilateral abnormality [‡]	1/10	0/10	1.0 [†]
Caloric irrigation	8/10	0/10	<.001 [†]
Total caloric response (peak slow phase eye velocity) (deg/s)	85.7±48.9	147.0±71.0	.02 [†]
Unilateral weakness	7/10	0/10	.003 [†]
Bilateral weakness	1/10	0/10	1.0 [†]
Optokinetic testing			
Subjects with abnormal circularvection	4/10	0/10	.087 [†]
Subjects with abnormal eye velocities	4/10	2/10	.628 [†]
Mean composite SOT score	69.6±35.8	79.5±40.5	.02*

NOTE. Values are mean ± SD or n/n.

* Unpaired *t* tests.

[†] Fisher exact test.

[‡] Unilateral weakness was defined as caloric differences $\geq 20\%$ between the 2 ears and is based on our age-matched laboratory norms. Bilateral weakness was defined as a total of the 4 irrigations that is $\leq 30^\circ/s$.

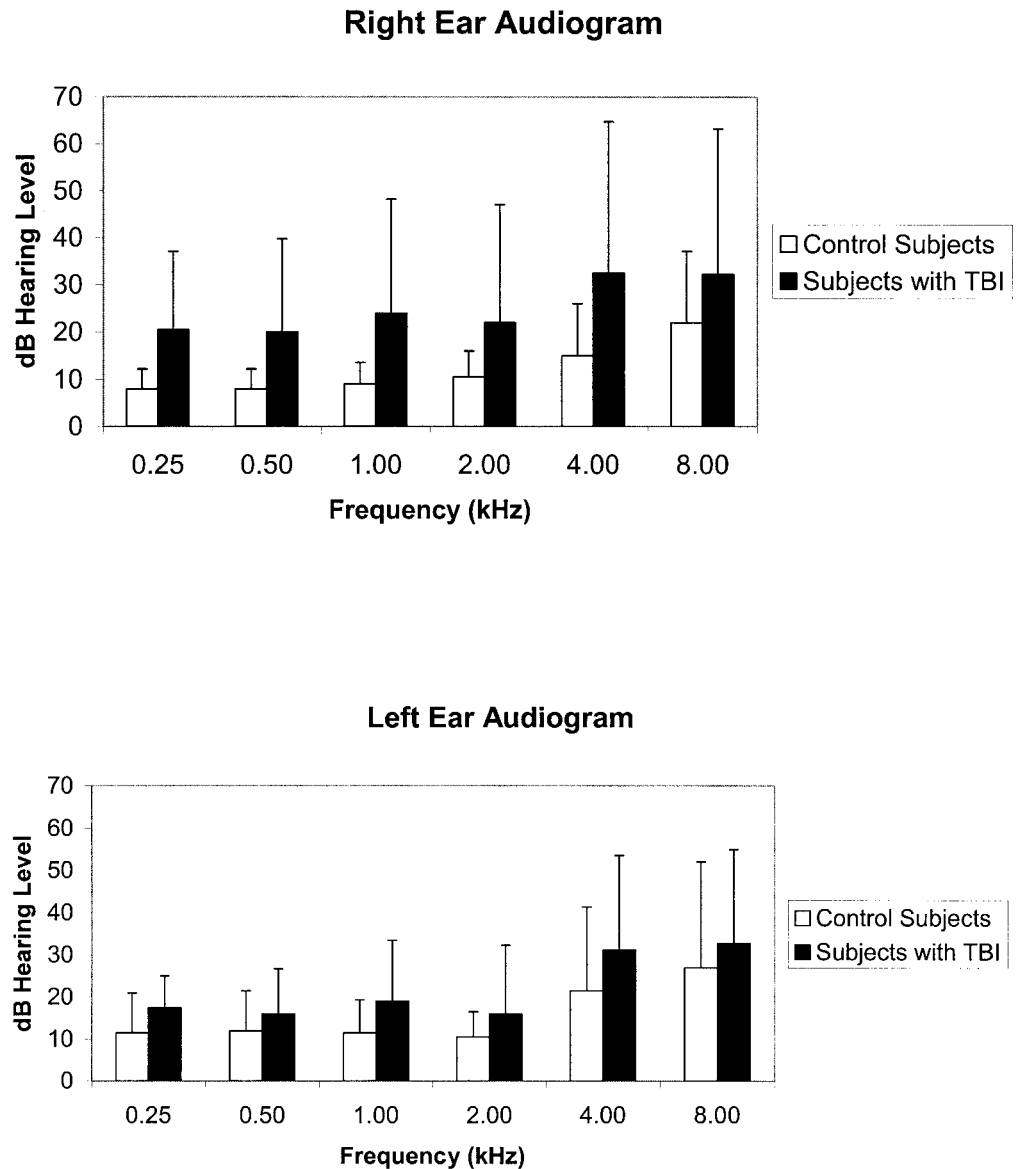


Fig 1. Audiograms of subjects with and without TBI. Error bars represent ± 1 SD.

Mean composite SOT scores differed significantly between the subjects who had had a TBI (69.6 ± 35.8) and the controls (79.5 ± 40.5) ($P = .02$), with lower scores representing greater body sway (poorer balance) (table 3). The scores of the individual test conditions are not presented, for the sake of brevity. Analysis, however, revealed that although the mean scores of the subjects with TBI were poorer than those of the control subjects for all 6 conditions tested, except for the unchallenged situation in condition 1 (eyes open, stable platform), these differences reached statistical significance only for condition 4 (sway-referenced support surface, eyes open) ($P = .04$).

Motion Analysis

The 3-dimensional gait patterns of the subjects with and without TBI differed significantly ($P = .05$). The ranges of displacement (m) and peak instantaneous velocity (m/s) of the whole body's COM in the AP direction during a gait stride were significantly lower in the subjects with TBI than in the controls ($P = .021$, $P < .001$, respectively; fig 2A); that reflects a

reduced walking velocity and stride length in these subjects. The subjects with a TBI also displayed significantly greater ML COM displacements and velocities (ie, increased ML instability) than their matched controls during level walking ($P < .0001$, $P < .002$, respectively; fig 2B). No significant group differences were identified in the vertical displacements or velocities of the whole body's COM (fig 2C).

DISCUSSION

This study provides a comprehensive assessment of people who, despite a seemingly good recovery from a TBI, continue to experience imbalance and instability. Our findings provide some interesting insights. First, the DHI indicates that this instability has pervasive physical, emotional, and functional effects on their lives (table 3). Second, even though this imbalance is not detectable by either clinical examination or a Tinetti Balance Assessment Scale score, it can be detected and quantified with specialized vestibular testing and gait analysis (table 3, fig 2). Third, the abnormal caloric responses and,

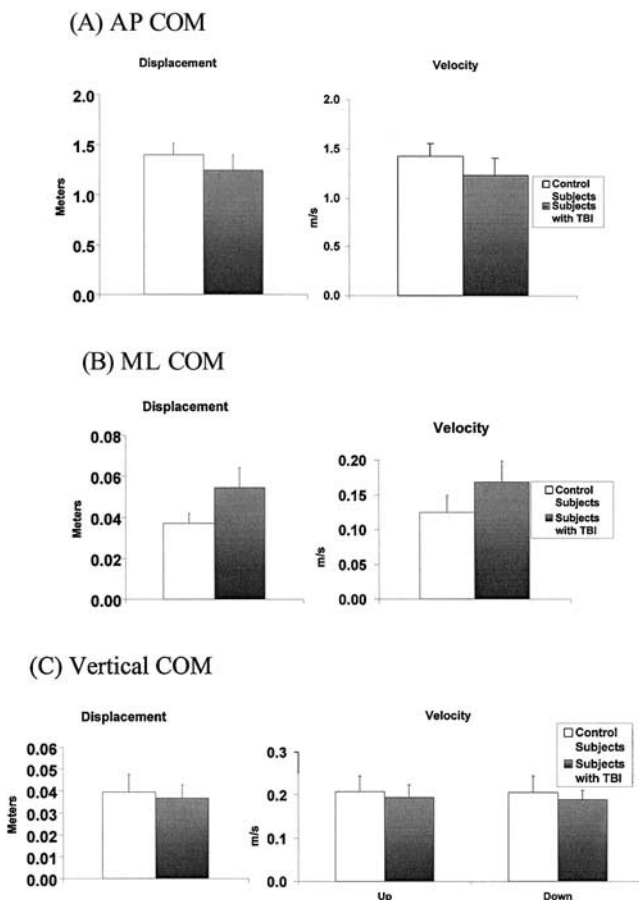


Fig 2. Three-dimensional movement of the whole-body COM displacements (m) and velocities (m/s) during gait in (A) the AP, (B) ML, and (C) vertical directions. Note that the COM AP displacements and velocities are significantly smaller ($P < .021$, $P < .013$, respectively), whereas the COM ML displacements and velocities ($P < .0001$, $P < .002$, respectively) are significantly larger in the subjects with TBI. Error bars represent ± 1 SD.

perhaps, the trend toward reduced pure-tone hearing threshold that were found in the subjects with TBI (table 3, fig 1) suggest that this instability may be associated with sensory and neurologic deficits in the vestibular system. The trend toward an increased incidence of abnormal optokinetic reflexes among people with brain injury may also suggest a central deficit in that they did not perceive the rotation noted by the control subjects when the small spots of light rotated around them.

The efficacy of the SOT in assessing the effect of vestibular dysfunction on the balance and function of people with vestibular injuries is controversial.^{40,41} This study reinforces these concerns in that we found that the SOT was relatively insensitive in our subjects. More specifically, although the mean overall SOT scores of the subjects with TBI were significantly lower than the scores of their noninjured counterparts, differences between the groups reached statistical significance for only 1 of the 6 testing conditions.

The fact that cranial nerve trauma is known to occur in association with even mild TBI⁴²⁻⁴⁴ supports the idea that the pathophysiology of these complaints has a traumatic origin. For example, the abnormalities found during caloric irrigation of the subjects with TBI support the potential importance of the interaction of differential forces among the base of the skull,

the cranial nerves, and the inferior brain surface.⁴⁵ In particular, the vestibular nerve passes through the internal acoustic meatus, and, thus, it would seem to be tethered relative to the more mobile brain adjacent to it and to be particularly subject to injury by shearing or tensional forces. Similarly, otoliths are known to be dislodged by trauma.^{24,46} BPPV (a manifestation of otolith displacement) was present only in subjects (3/10) with a TBI. This difference did not reach statistical significance. Nevertheless, its presence, as well as the fact that the 2 subjects with TBI who we treated in this study (as well as 4 others who were referred after the study) responded to otolith-repositioning maneuvers,⁴⁷ is encouraging. Our sample size was small. It is possible that the abnormalities in the subjects with TBI were present before they were injured. However, this seems unlikely because of their history of normal gait and balance before their injuries and the dichotomous distribution of the findings between the groups.

During gait, the reductions in the whole-body COM AP displacement and peak instantaneous velocity in the subjects with TBI were primarily the result of a slower walking speed and a shorter stride length. Pai and Patton⁴⁸ showed that both the distance between the whole body's COM and the supporting foot and the COM's horizontal (AP) velocity are critical to maintaining balance. Therefore, our data imply that the subjects with TBI reduced their sagittal plane movement during gait to maintain their balance. Furthermore, our findings of increased COM ML excursion and peak velocity during gait in the subjects with TBI indicate an impaired balance control in the frontal plane that is similar to that reported for balance-impaired elderly adults.^{49,50}

CONCLUSION

This study suggests that (1) even subtle complaints of persistent imbalance by patients after a TBI should be investigated; (2) gait analysis and balance and vestibular testing can document subtle changes in gait and balance among those with TBI; and (3) imbalance may not be due merely to diffuse brain injury. Comprehensive vestibular testing seems appropriate in all patients with persistent complaints of imbalance and instability after TBI.

References

- Kay A, Teasdale G. Head injury in the United Kingdom. *World J Surg* 2001;25:1210-20.
- Pickett W, Arden C, Brison RJ. A population-based study of potential brain injuries requiring emergency care. *CMAJ* 2001; 165:288-92.
- Gurr B, Moffat N. Psychological consequences of vertigo and the effectiveness of vestibular rehabilitation for brain injury patients. *Brain Inj* 2001;15:387-400.
- Sosin DM, Sniezek JE, Thurman DJ. Incidence of mild and moderate brain injury in the United States, 1991. *Brain Inj* 1996; 10:47-54.
- Meythaler JM, Peduzzi JD, Eleftheriou E, Novack TA. Current concepts: diffuse axonal injury-associated traumatic brain injury. *Arch Phys Med Rehabil* 2001;82:1461-71.
- Malec JF. Cognitive rehabilitation. In: Evans R, editor. *Neurology and trauma*. Philadelphia: WB Saunders; 1996. p 231-48.
- Malec JF. Mild traumatic brain injury: scope of the problem. In: Varney NR, Roberts RJ, editors. *The evaluation and treatment of mild traumatic brain injury*. Mahwah (NJ): Lawrence Erlbaum Assoc; 1999. p 15-31.
- Mrazik M, Ferrara MS, Peterson CL, et al. Injury severity and neuropsychological and balance outcomes of four college athletes. *Brain Inj* 2000;14:921-31.
- Alves WM, Colohan A, O'Leary TJ, Rimel RW, Jane JA. Understanding posttraumatic symptoms after minor head injury. *J Head Trauma Rehabil* 1986;1(2):1-12.

10. Cicerone KD, Kalmar K. Persistent postconcussion syndrome: the structure of subjective complaints after mild traumatic brain injury. *J Head Trauma Rehabil* 1995;10(3):1-17.
11. Newton RA. Balance abilities in individuals with moderate to severe traumatic brain injury. *Brain Inj* 1995;9:445-51.
12. Lehmann JF, Boswell S, Price R, et al. Quantitative evaluation of sway as an indicator of functional balance in post-traumatic brain injury. *Arch Phys Med Rehabil* 1990;71:955-62.
13. Ingersoll CD, Armstrong CW. The effects of closed-head injury on postural sway. *Med Sci Sports Exerc* 1992;24:739-43.
14. Wober C, Oder W, Kollegger H, et al. Posturographic measurement of body sway in survivors of severe closed head injury. *Arch Phys Med Rehabil* 1993;74:1151-6.
15. Rubin AM, Woolley SM, Dailey VM, Goebel JA. Postural stability following mild head or whiplash injuries. *Am J Otol* 1995;16:216-21.
16. Geurts AC, Ribbers GM, Knoop JA, Limbeek J. Identification of static and dynamic postural instability following traumatic brain injury. *Arch Phys Med Rehabil* 1996;77:639-44.
17. Lahat E, Barr J, Klin B, Dvir Z, Bistrizer T, Eshel G. Postural stability by computerized posturography in minor head trauma. *Pediatr Neurol* 1996;15:299-301.
18. Wade LD, Canning CG, Fowler V, Felmingham K, Baguley IJ. Changes in postural sway and performance of functional tasks during rehabilitation after traumatic brain injury. *Arch Phys Med Rehabil* 1997;78:1107-11.
19. Geurts AC, Knoop JA, van Limbeek J. Is postural control associated with mental functioning in persistent postconcussion syndrome? *Arch Phys Med Rehabil* 1999;80:144-9.
20. Tinetti ME, Speechley M, Ginter SF. Risk factors for fall among elderly persons living in community. *N Engl J Med* 1988;319:1071-7.
21. Lezak MD. *Neuropsychological assessment*. 3rd ed. New York: Oxford Univ Pr; 1995.
22. Kerber KA, Enrietto JA, Jacobson KM, Baloh RW. Disequilibrium in older people: a prospective study. *Neurology* 1998;51:574-80.
23. Whitman GT, Tang T, Lin A, Baloh RW. A prospective study of cerebral white matter abnormalities in older people with gait dysfunction. *Neurology* 2001;57:990-4.
24. Jacobson GP, Newman CW. The development of the Dizziness Handicap Inventory. *Arch Otolaryngol Head Neck Surg* 1990;116:424-7.
25. Dix R, Hallpike CS. The pathology, symptomatology and diagnosis of certain common disorders of the vestibular system. *Ann Otol Rhinol Laryngol* 1952;6:987-1016.
26. Jacobson GP, Newman CW, Peterson EL. Interpretation and use of caloric testing. In: Jacobson GP, Newman CW, Kartush JM, editors. *Handbook of balance function testing*. St Louis: Mosby Year Book; 1993. p 195-6.
27. Baird RJ. Optokinetic response to 20, 40, 60, 80, and 100 deg/s full visual field drum and sphere stimulation [master's thesis]. Provo (UT): Brigham Young Univ; 2000.
28. Baloh R. How do vestibular disorders affect balance, spatial orientation and motion perception? In: Baloh RW, Halmagyi GM, editors. *Disorders of the vestibular system*. New York: Oxford Univ Pr; 1996. p 126-39.
29. Carhart R, Jerger JF. Preferred method for clinical determination of pure-tone thresholds. *J Speech Hear Disord* 1959;24:330-45.
30. EquiTest System. *Data interpretation manual*. Clackamas (OR): NeuroCom International; 1991.
31. Chou LS, Kaufman K, Brey RH, Draganich LF. Motion of the whole body's center of mass when stepping over obstacles of different heights. *Gait Posture* 2001;13:17-26.
32. Growney E, Meglan D, Johnson M, Cahalan T, An KN. Repeated measures of adult normal walking using a video tracking system. *Gait Posture* 1997;6:147-62.
33. Dempster WT, Gaughran GR. Properties of body segments based on size and weight. *Am J Anat* 1967;120:33-54.
34. Zatsiorsky V, Seluyanov V. Estimation of the mass and inertia characteristics of the human body by means of the best predictive regression equations. In: Winter DA, Norman RW, Wells RP, Hayes KC, Patla AE, editors. *Biomechanics IX-B*. Champaign (IL): Human Kinetics; 1985. p 233-9.
35. Eng JJ, Winter DA. Estimations of the horizontal displacement of the total body centre of mass: considerations during standing activities. *Gait Posture* 1993;1:141-4.
36. Jian Y, Winter DA, Ishac MG, Gilchrist L. Trajectory of the body COG and COP during initiation and termination of gait. *Gait Posture* 1993;1:9-22.
37. Winter DA. *The biomechanics and motor control of human gait: normal, elderly and pathological*. Waterloo (Ont): Univ Waterloo Pr; 1991.
38. Woltring HJ. A FORTRAN package for generalized, cross-validatory spline smoothing and differentiation. *Adv Eng Software* 1986;8:104-13.
39. Lindsay JT, Pettigrew LE, Teasdale GM. Structured interviews for the Glasgow Outcome Scale and the Extended Glasgow Outcome Scale: guidelines for their use. *J Neurotrauma* 1998;15:573-8.
40. Evans MK, Krebs DE. Posturography does not test vestibulospinal function. *Otolaryngol Head Neck Surg* 1999;120:164-73.
41. O'Neill DE, Gill-Body KM, Krebs DE. Posturography changes do not predict functional performance changes. *Am J Otol* 1998;19:797-803.
42. Mariak Z, Mariak Z, Stankiewicz A. Cranial nerve II-VII injuries in fatal closed head trauma. *Eur J Ophthalmol* 1997;7:68-72.
43. Muthu P, Pritty P. Mild head injury with isolated third nerve palsy. *Emerg Med J* 2001;18:310-1.
44. Lahbabi M, Levy JD, Laxenaire A, Scheffer P. [Bilateral paralysis of the 6th cranial nerve pair and minor head injury. Apropos of a case. Review of the literature] [French]. *Rev Stomatol Chir Maxillofac* 1997;98:295-8.
45. Bradshaw DR, Ivarsson J, Morfey CL, Viano DC. Simulation of acute subdural hematoma and diffuse axonal injury in coronal head impact. *J Biomech* 2001;34:85-94.
46. Baloh RW, Honrubia V, Jacobson K. Benign positional vertigo: clinical and oculographic features in 240 cases. *Neurology* 1987;37:371-8.
47. Epley JM. The canalith repositioning procedure: for treatment of benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg* 1992;107:399-404.
48. Pai YC, Patton J. Center of mass velocity-position predictions for balance control. *J Biomech* 1997;30:347-54.
49. Krebs DE, Jette AM, Assmann SF. Moderate exercise improves gait stability in disabled elders. *Arch Phys Med Rehabil* 1998;79:1489-95.
50. Kaya BK, Krebs DE, Riley PO. Dynamic stability in elders: momentum control in locomotor ADL. *J Gerontol A Biol Sci Med Sci* 1998;53:M126-34.

Suppliers

- a. ICS Medical Corp, 125 Commerce Dr, Schaumburg, IL 60173.
- b. Grason-Stadler Inc, 5225 Verona Rd, Bldg 2, Madison, WI 53711-4495.
- c. EquiTest@; NeuroCom International Inc, 9570 SE Lawnfield Rd, Clackamas, OR 97015.
- d. Motion Analysis Corp, 3617 Westwind Blvd, Santa Rosa, CA 95403.