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Dynamic instability during obstacle crossing following traumatic brain injury

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Abstract

Patients with traumatic brain injury (TBI) complain of "imbalance" or "unsteadiness" while walking, despite a normal gait on clinical examination. Thus, the purpose of this study was to determine if it was possible to quantitatively assess dynamic stability that did not have an obvious neuromuscular origin in individuals following TBI. Ten patients with documented TBI and 10 age, gender, and stature-matched healthy individuals participated in the study. All subjects were instructed to perform unobstructed level walking and to step over obstacles corresponding to 2.5%, 5%, 10%, and 15% of their height. A 13-link biomechanical model of the human body was used to compute the kinematics of the whole body center of mass (COM). Subjects with TBI walked with a significantly slower gait speed and shorter stride length than their matched controls. Furthermore, subjects with TBI displayed a significantly greater and faster medio-lateral (M-L) COM motion and maintained a significantly greater M-L separation distance between their COM and center of pressure (COP) than their matched control subjects. These measurements indicate that subjects with TBI have difficulty maintaining dynamic stability in the frontal plane and have a reduced ability to successfully arrest their sagittal momentum. These findings provide an objective measurement that reflects the complaints of instability not observable on clinical examination for individuals who have suffered a TBI. This ability to identify any functional impairment after a traumatic brain injury that may affect patient safety is critical for prevention of re-injury during the recovery period.

Keywords: Traumatic brain injury; Center of pressure; GCS

1. Introduction

Traumatic brain injury (TBI) is one of the most challenging problems faced by the medical community. It is estimated that 5.3 million Americans, a little more than 2% of the US population, currently live with disabilities resulting from TBI [1]. Each year over one million people are treated for TBI and released from hospital emergency rooms. The cost of TBI in the US is estimated to be US$ 48.3 billion annually. After one traumatic brain injury, the risk for a second injury is three times greater, and after the second injury, the risk for a third injury increases by a factor of eight [1]. Therefore, the ability to identify any functional impairment that may affect safety after a TBI is critical to prevention of re-injury. The utility of neuropsychometric testing in evaluating cognitive impairment resulting from TBI is well established [2–6]. However, comparatively little information is available on the performance of dynamic motor tasks following TBI.

Many individuals with a mild or moderate TBI complain of symptoms long after their injury, even though their clinical examinations and measurable cognitive deficits are small [7,8]. Approximately one third of these patients complain of sensorimotor problems, in particular, poor balance and poor coordination [9,10]. These balance and coordination complaints may not be surprising since effective coordination and balance involves a complex interaction of the sensory, motor programming and musculoskeletal systems. Sensory

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systems monitor the location of the whole body center of mass (COM) relative to the base of support, provide information about vertical orientation, and supply environmental information, particularly regarding the support surface. Appropriate motor responses include an appropriate latency of onset as well as measured and coordinated force generation in activated muscles [11].

Biomechanical studies of individuals with TBI have, for the most part, been limited to postural sway during quiet standing or during standing with altered sensory inputs [12–19]. Subjects with TBI exhibit an increased reliance on visual input and are not able to use their vestibular systems as effectively as uninjured subjects to resolve conflicts in information from the visual and somatosensory systems [16]. Geurts et al. [16] reported that subjects who had sustained a TBI (mild, $n = 13$; moderate, $n = 2$; and severe $n = 5$) exhibited at least 50% more static anterior–posterior (A–P) and lateral sway than healthy controls at 6 months following injury. These same subjects showed no sensorimotor impairments in a standard neurological examination. Guskiewicz [20], on the other hand, reported little relationship between symptoms and measures of cognitive function and static postural stability during the first 2 days following a concussion. These data suggest that motor function may recover more slowly than cognitive function or may not be closely related to standard neuropsychological assessments.

Many studies have used the whole body COM motion and its interaction with the center of pressure (COP) as indicators to examine an individual's dynamic stability. It has been demonstrated that COM motion is tightly regulated to move between the alternating COP of each supporting foot [21–25]. Furthermore, our previous studies

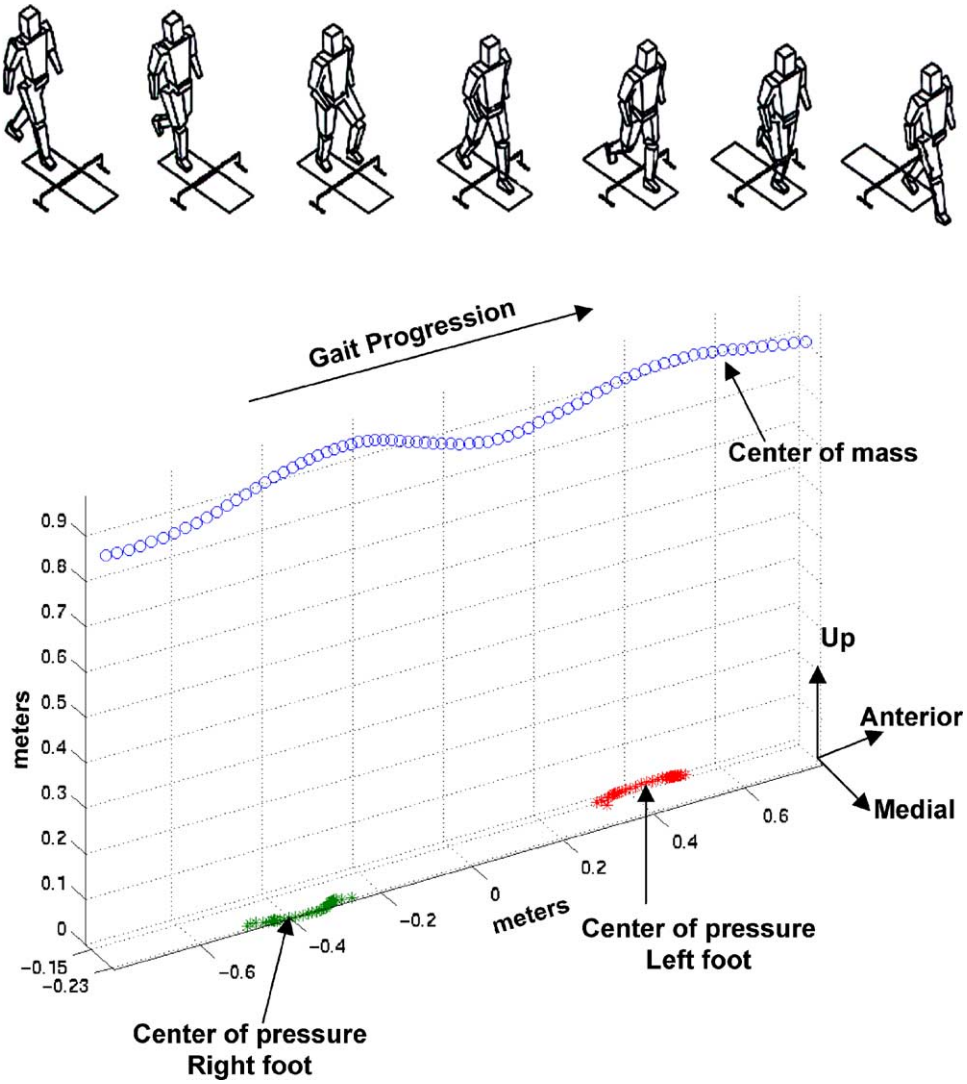


Fig. 1. The gait was analyzed from the heel-strike of the trailing limb before stepping over the obstacle to the heel-strike of the trailing limb after the obstacle crossing. A 13-link biomechanical model of the human body was used to compute the kinematics of the whole body's COM. A typical three-dimensional trajectories of the whole body COM (○) and the corresponding COP (∗) during an unobstructed walking cycle (heel-strike to heel-strike) of a control subject is shown.

demonstrated that elderly adults complaining of "dizziness" or "unsteadiness" displayed significantly greater and faster medio-lateral (M-L) COM motion than the healthy elderly while negotiating obstacles of different heights [26,27]. Therefore, COM motion in the frontal plane could be a functional indicator of balance maintenance during walking, and with the addition of the obstacle it could be a more sensitive measurement of dynamic stability.

Given that most falls appear to stem from tripping over objects [28–31], it is important to determine the influence of pre-existing brain injury on an individual's balance control while interacting with environmental hazards, such as stepping over an obstacle during walking. Therefore, the purpose of this study was to quantitatively assess dynamic stability of individuals who have complaints of "instability" or "imbalance" following TBI despite having an apparently normal gait on clinical examination. We hypothesized that patients with TBI would demonstrate less stability, as indicated by greater and faster M-L motion of the whole body COM, when stepping over progressively higher obstacles than subjects without a similar injury.

2. Methods

Ten patients (six men and four women) with a TBI were recruited by physician referral from the National Institute on Disability and Rehabilitation Research (NIDRR)

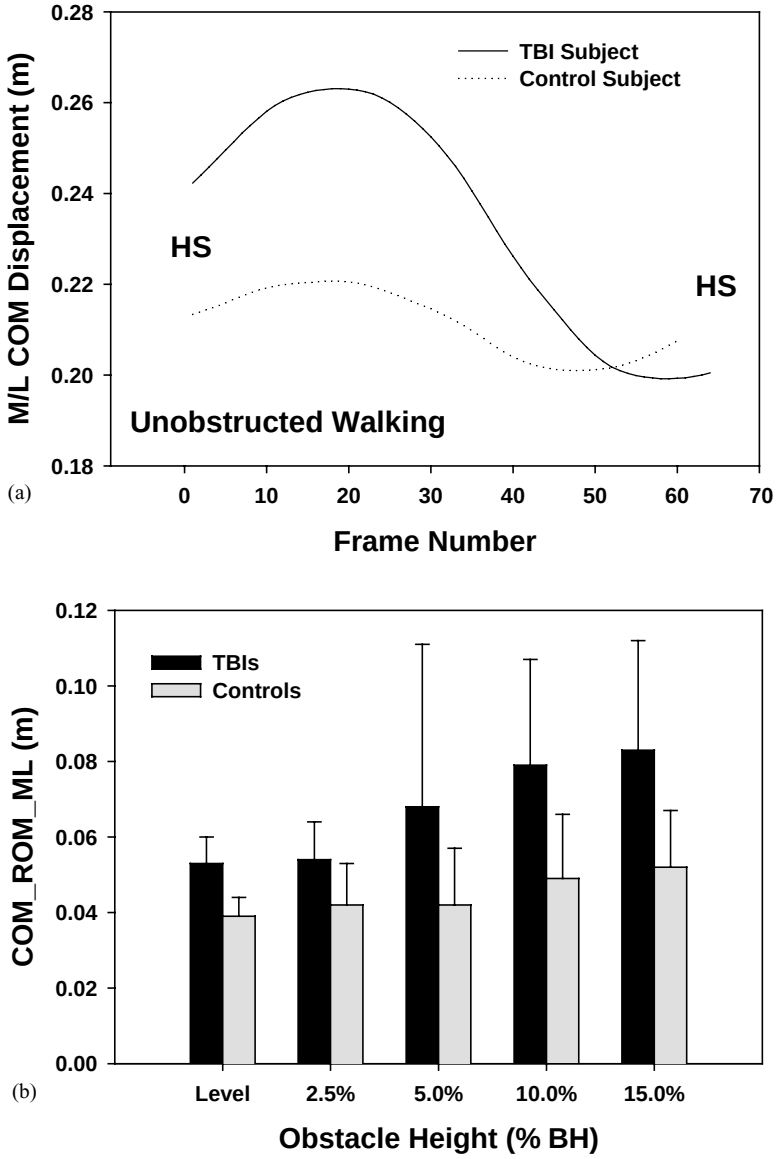
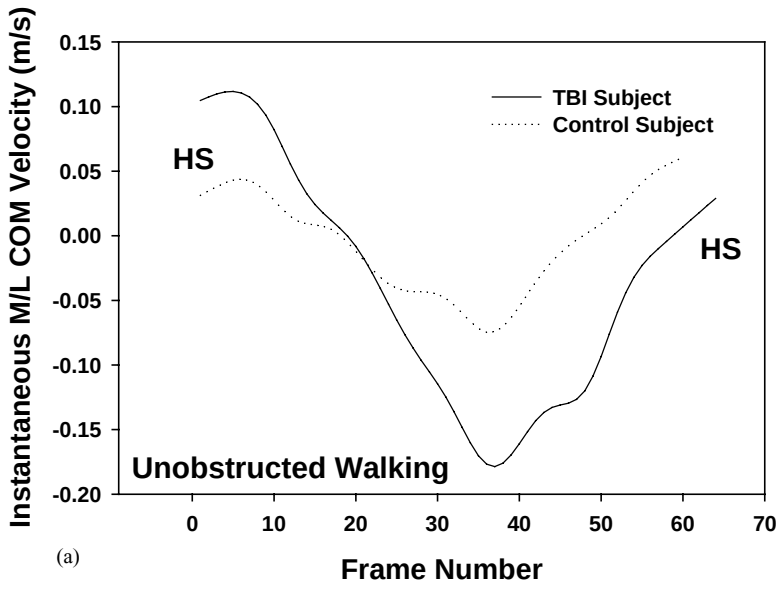


Fig. 2. (a) Typical profiles of the M-L COM trajectories during an unobstructed walking cycle (heel-strike to heel-strike) from a representative subject with TBI (—) and a matched control subject (· · ·). (b) Means and standard deviations of the medio-lateral (M-L) COM displacement during the crossing stride. Subjects with TBI demonstrated significantly greater M-L displacement across all obstacle conditions. The M-L COM displacement increased linearly with obstacle height. HS: heel-strike and BH: body height.

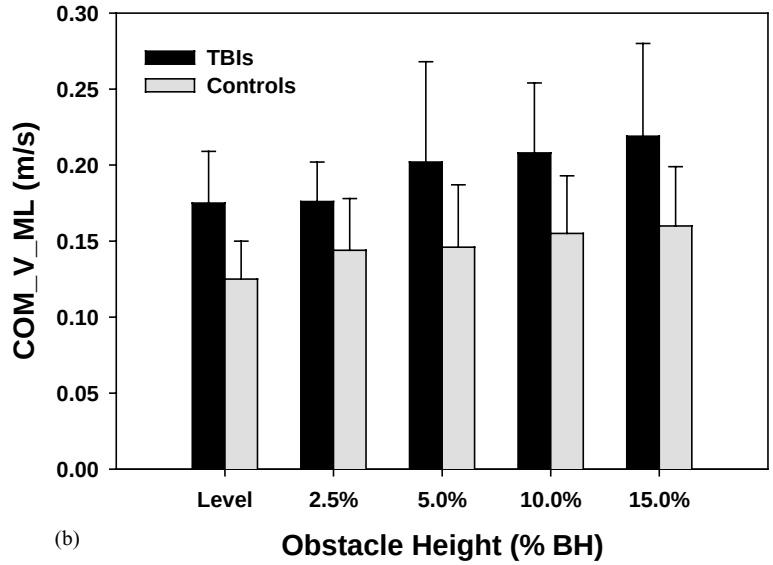
Table 1
Gait temporal–distance measurements for both groups during the crossing stride

	Obstacle height										P-value[a]
	None		2.5%		5%		10%		15%		
	Control	TBI	Controls	TBI	Controls	TBI	Controls	TBI	Controls	TBI	
Gait velocity (m/s)	1.310[b] (0.109)	1.151 (0.167)	1.220 (0.117)	1.070 (0.153)	1.168 (0.123)	1.047 (0.152)	1.108 (0.117)	0.968 (0.132)	1.036 (0.127)	0.902 (0.125)	$P_g = 0.022$ $P_h < 0.001$
Stride length (m)	1.408 (0.115)	1.269 (0.151)	1.410 (0.083)	1.284 (0.138)	1.410 (0.088)	1.297 (0.132)	1.415 (0.086)	1.283 (0.116)	1.412 (0.079)	1.282 (0.115)	$P_g = 0.018$ $P_h = 0.604$
Step width (m)	0.121 (0.025)	0.138 (0.047)	0.124 (0.025)	0.130 (0.032)	0.127 (0.023)	0.143 (0.023)	0.131 (0.021)	0.141 (0.027)	0.126 (0.022	0.145 (0.033)	$P_g = 0.242$ $P_h = 0.284$

[a] P_h and P_g represent height and group effects.
[b] Mean value, with standard deviation in parentheses.



(a)



(b)

Fig. 3. (a) Typical profiles of the instantaneous M-L COM velocity during an unobstructed walking cycle (heel-strike to heel-strike) from a representative subject with TBI (—) and a matched control subject (· · ·). (b) Means and standard deviations of the peak M-L COM velocity during the crossing stride. The peak M-L COM velocities of TBI subjects were significantly faster than that of control subjects across all obstacle conditions and increased linearly with obstacle height.

sponsored Mayo Clinic Traumatic Brain Injury Model Center. The diagnosis of a TBI was based on their history and medical records (e.g., a decreased Glasgow Coma Score (GCS) within 24 h following initial admission and documented loss of consciousness). Based on the initial GCS obtained from their medical records, four of the subjects had a severe TBI (GCS < 9), two had moderate (GSC $=$ 9–12), and four had mild (GCS > 12) brain injuries. Nine of the subjects were evaluated within 2 years after their injury (mean duration: 12.3 months; range: 4–22 months). One of the subjects had a duration of injury of 15 years and 4 months. The subjects were living in the community, had normal gait and balance prior to their injury, and had complaints of dizziness or unsteadiness when walking. Importantly, all subjects with TBI in this study had a normal neurological and musculoskeletal examination. Cognitive, medical, or behavioral problems that would interfere with participation were considered exclusion criteria. The subjects with TBI had a mean age of 40.9 ± 11.3 years, mean height of 168 ± 5.6 cm and mean body mass of 77 ± 13.3 kg. Detailed information of their clinical evaluations and demographics were reported previously [8]. Ten healthy individuals without a history of TBI were recruited by advertisement to serve as age, gender, height and weight-matched control subjects. These individuals had a mean age of 41.2 ± 11.4 years, mean height of 176 ± 8 cm and mean body mass of 75.9 ± 11.5 kg. The protocol for this study was approved by the Institutional Review Board of the Mayo Foundation.

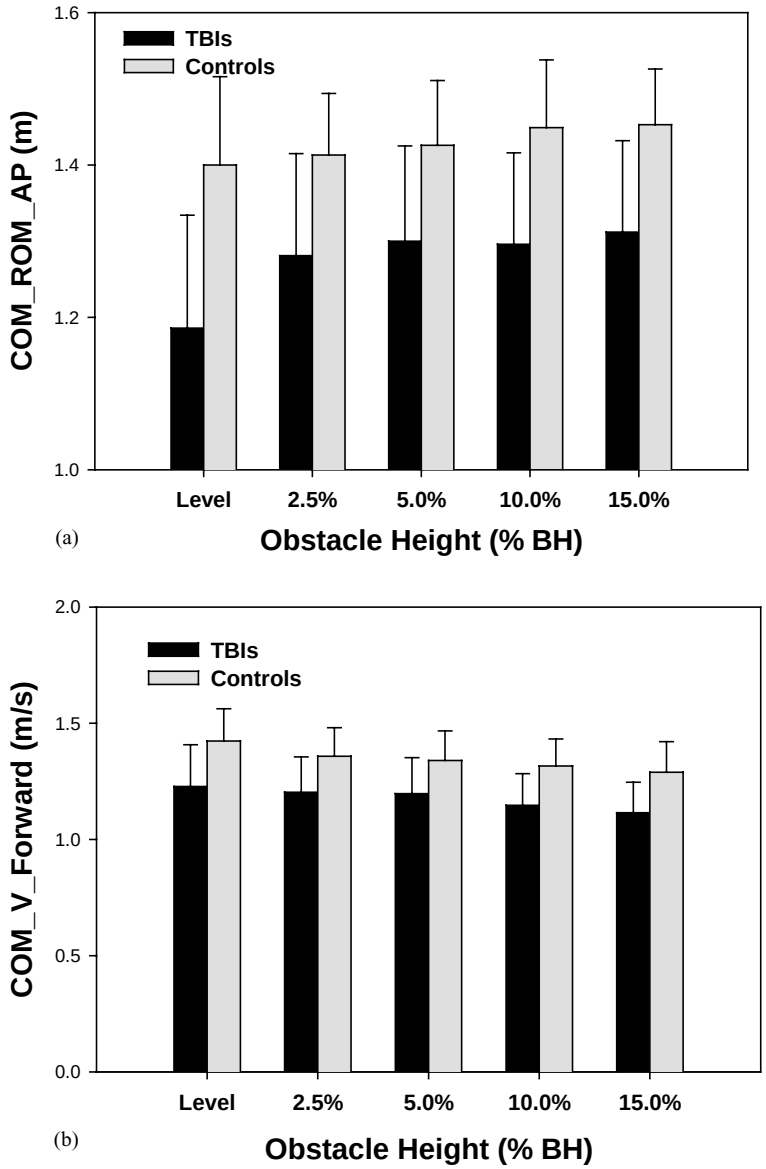


Fig. 4. Means and standard deviations of the (a) anterior–posterior (A–P) displacements and (b) peak COM forward velocities of the COM during the crossing stride. The A–P COM displacements in subjects with TBI were significantly shorter and increased linearly as the obstacle height increased. The peak forward COM velocities in subjects with TBI were significantly slower and decreased linearly as the obstacle height increased.

The experimental procedures for the study were explained to the subjects and written informed consent was obtained prior to commencing the study.

The experimental protocol included level unobstructed walking and crossing over obstacles set to heights equal to 2.5%, 5%, 10%, and 15% of each individual's body height. These normalized obstacle heights were designed to produce a similar level of challenge for individuals with different body heights. The lowest height (∼5 cm) represented that of a typical door threshold, and the greatest height (∼20 cm) was similar to that of a high curb or stair. The obstacle was made of two adjustable upright standards and a padded crossbar (∼2.5 cm in diameter) two meters in length. The crossbar rested loosely on the upright standards, so that any foot contact would dislodge it easily, lowering the risk of tripping to the subject. A more detailed description of the obstacle design was reported previously [26]. Subjects were instructed to walk along a 6 m walkway, stepping over the obstacle and to continue walking along at a self-selected pace. All subjects were tested barefoot. Each subject was allowed to select his/her preferred limb for leading over the obstacle. Starting positions were selected for each subject to ensure that a comfortable pace was reached before encountering the obstacle. Unobstructed walking trials were performed first, followed by obstacle-crossing trials. The obstacle height was randomly selected for each trial. Three trials were performed for each obstacle condition.A set of 27 reflective markers modified from Kadaba et al. [32] and Jian et al. [33] was placed on bony landmarks of the subject. An eight-camera ExpertVison^{TM} system (Motion Analysis Corp., Santa Rosa, CA) was used to collect 3-D marker trajectory data. The three-dimensional marker trajectory data were collected at 60 Hz and low-pass filtered using a fourth-order Butterworth filter with a cutoff frequency of 8 Hz. Ground reaction forces of the supporting foot right before and after the obstacle were collected with two force plates (Kistler 9281B and Bertec 4060A) at 960 Hz. The force data were time-synchronized to the video sampling.

The location of the whole body COM was computed as the weighted sum of each body segment's COM forming a 13-link biomechanical model, consisting of six links for the lower extremities, four links for the upper extremities, one for the pelvis, one for the trunk, and one for the head [33,34]. The range of the 3-D COM motion (the maximum minus minimum value achieved during the crossing stride) was then computed. The linear velocities of the whole body COM were calculated using the generalized cross-validation spline (GCVSPL) algorithm [35]. The COP under the stance foot was computed based on the measured ground reaction force and moment about the origin of the force plate. The motion data were analyzed from the heel-strike of the trailing limb before stepping over the obstacle to its next heel-strike when crossing the obstacle (i.e., crossing stride; Fig. 1).The mean of three trials for each obstacle condition was used in formulating the results. Effects of subject group and obstacle height on the temporal–distance parameters, COM kinematics, and the difference between the COM and corresponding (i.e., in time) COP during the single support phase of the crossing stride were assessed using a two-way ANOVA with repeated measures on one factor (obstacle height). If a significant obstacle height effect was detected, a polynomial test was performed at the $\alpha = 0.05$ level of significance to determine if a linear, quadratic, or cubic trend existed. All statistical analyses were conducted with SYSTAT (version 9, SPSS Inc., Chicago, IL).

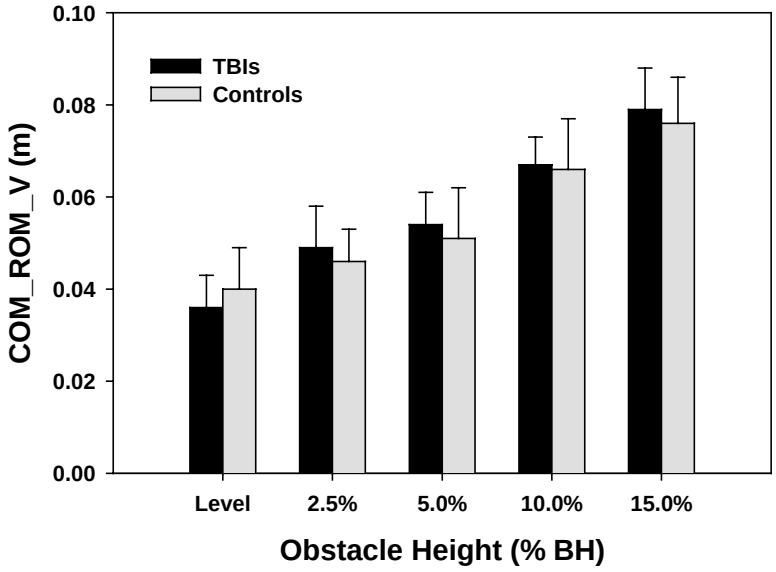


Fig. 5. Means and standard deviations of the vertical COM displacements during the crossing stride. The vertical COM displacements increased linearly with obstacle height. No significant group differences were detected.

3. Results

Eight of the subjects with TBI were able to consistently perform all testing trails with either their right or left limb leading (five right and three left leading). All control subjects except for one selected their left limb as the leading limb. No incidents of tripping occurred for any of the obstacle height conditions in either subject group. Post-TBI subjects adopted a gait pattern with a significantly slower walking speed ($P = 0.02$) and shorter stride length ($P = 0.018$) than controls during all conditions (Table 1). The walking speed of all subjects was found to decrease linearly ($P < 0.001$) as obstacle height increased. There were no significant group or obstacle height effects for step width.

The 3-D trajectories of the COM and the corresponding COP were similar in both subject groups. Typical trajectories of the COM and COP during an unobstructed walking trial of a control subject are shown in Fig. 1. Subjects in the TBI group demonstrated significantly greater M-L COM sway ($P = 0.005$) during the crossing stride across all obstacle conditions when compared to control subjects (Fig. 2a). M-L COM sway increased linearly ($P < 0.001$) as obstacle height increased (Fig. 2b). The instantaneous M-L COM velocities had a similar pattern in both subject groups (Fig. 3a). However, the peak M-L COM velocities during the weight-shifting period for subjects with TBI were significantly faster ($P = 0.007$) than that of control subjects and increased linearly ($P = 0.001$) with obstacle height (Fig. 3b).

The A–P range of motion for the COM during the crossing stride was significantly reduced ($P = 0.004$) in the TBI group and increased linearly ($P < 0.001$) with obstacle

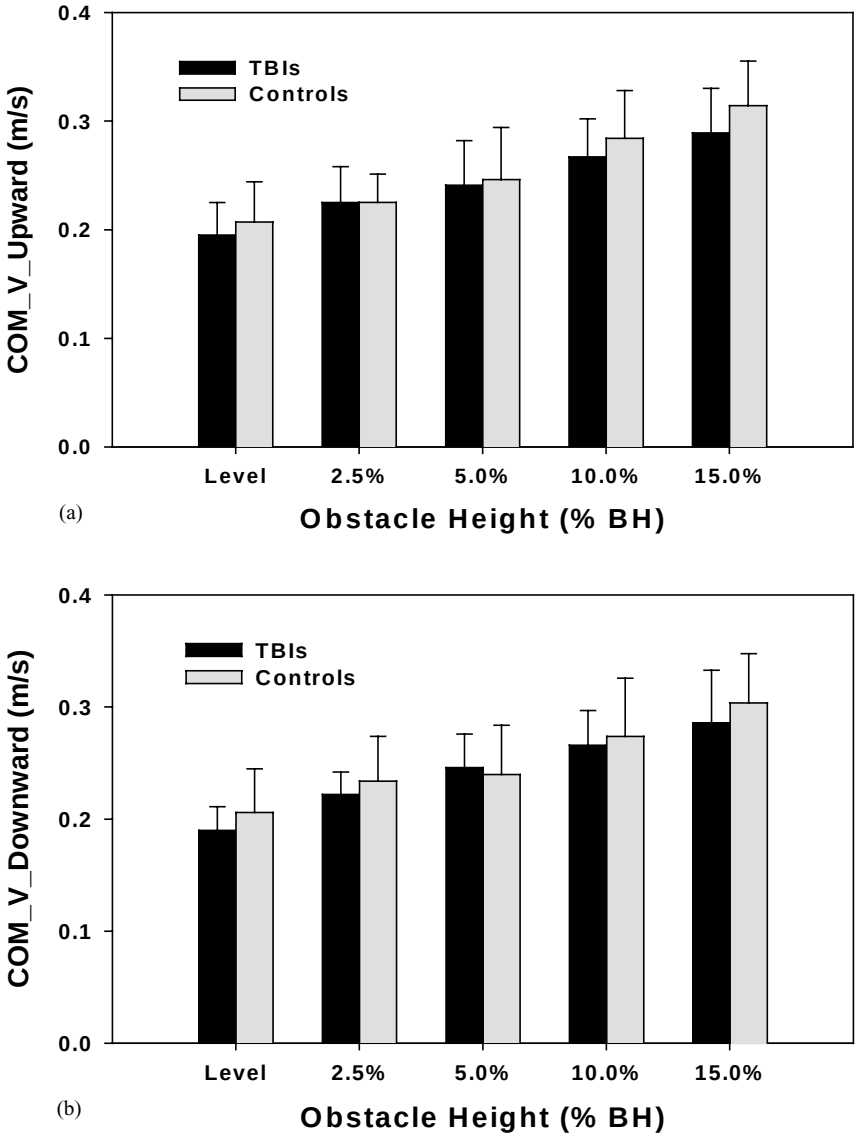


Fig. 6. Means and standard deviations of the peak (a) upward and (b) downward COM velocities during the crossing stride. No significant group differences were found in either peak COM upward or downward velocities. Both peak upward and downward velocities increased linearly as obstacle height increased.

height (Fig. 4a). Similar to walking speed, the peak instantaneous forward COM velocities in the subjects with TBI were significantly slower ($P = 0.012$) and decreased linearly ($P < 0.001$) as the obstacle height increased (Fig. 4b). No significant group differences were found in the vertical COM range of motion during the crossing stride (Fig. 5). Vertical COM range of motion increased linearly ($P < 0.001$) as obstacle height increased. No significant group differences were found in peak COM vertical (upward or downward) velocities (Fig. 6). Both peak upward and downward velocities increased linearly ($P < 0.001$) as the obstacle height increased.

The greatest horizontal A–P separation between the whole body COM and the corresponding COP occurred immediately before heel-strike of the swing limb. The maximum A–P distance between the COM and COP during the crossing stride increased linearly ($P < 0.001$) as the obstacle height increased in both subject groups (Fig. 7a). However, no significant group differences in maximum COM–COP A–P distances were detected. In contrast, maximum M-L distances between the whole body COM and the corresponding COP during single stance periods were significantly greater ($P = 0.016$) in subjects with TBI, when compared to their controls, and were not significantly affected by the obstacle height (Fig. 7b).

No significant interactions of obstacle height and subject group were identified in any of the variables reported above.

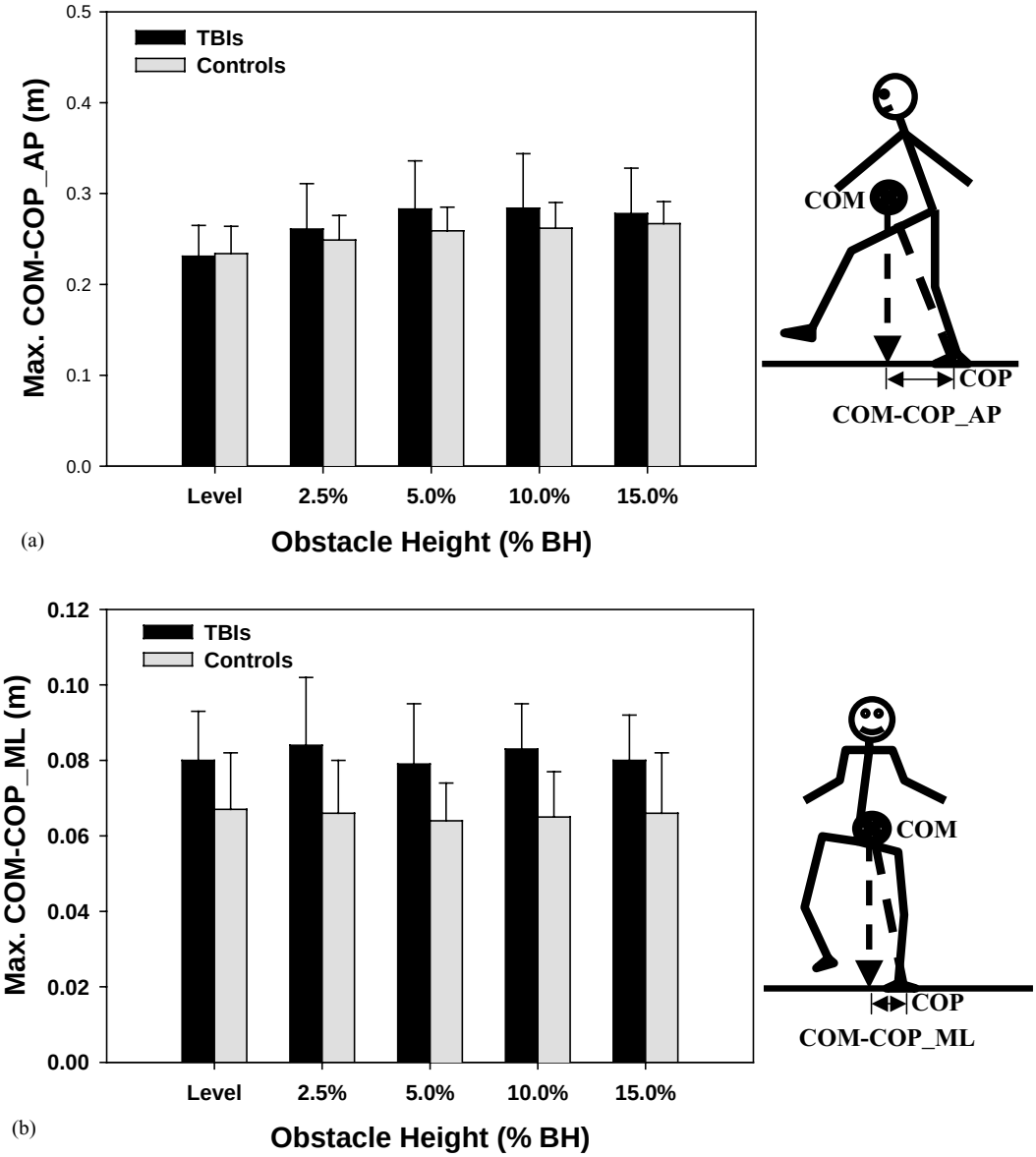


Fig. 7. Means and standard deviations of the maximum (a) A–P and (b) M-L separation distances between the COM and COP of the stance foot. The maximum M-L separation distances were significantly greater in subjects TBI. The maximum A–P separation distance increased linearly as obstacle height increased.

4. Discussion

Subjects with TBI often complain of "dizziness" or "unsteadiness" despite what appears to be a clinically normal gait. The results of this study clearly demonstrate that these individual do have gait abnormalities based on objective testing. Specifically, the subjects with a history of TBI adopted a gait pattern with a significantly slower speed, a shorter stride length and an increase in the M-L COM motion. These individuals also maintained a significantly greater separation distance between their COM and COP in the M-L direction than age and gender-matched controls during both unobstructed walking and while stepping over an obstacle.

The increased M-L COM motion in subjects with TBI indicated difficulty in maintaining dynamic stability in the frontal plane, which may directly reflect their sensation of instability. Magnitudes of the increased M-L COM motion in subjects with TBI during walking and obstacle crossing appear to be similar to those of older individuals with vestibular disorders [25,27]. These differences in the M-L COM motion between TBI and control subjects were smaller during unobstructed walking and stepping over a lower obstacle (2.5% body height). Greater differences were demonstrated while negotiating higher obstacles. These data demonstrate that body motion in the frontal plane is an objective measurement to quantitatively document the complaints of instability during walking from patients with a TBI. The addition of an obstacle enhances this measurement of dynamic instability.

Reductions in the whole body COM A–P displacement and peak instantaneous velocity in subjects with TBI are primarily due to their adoption of a significantly slower walking speed and a shorter stride length. It has been demonstrated previously that both the distance between the COM and the supporting foot and the instantaneous A–P velocity of COM are critical for the ability to successfully arrest forward momentum and maintain dynamic stability in the sagittal plane [36]. Thus, in subjects with TBI the feasible A–P range of COM movement during which dynamic balance can be maintained has been reduced, i.e., only a slower COM forward velocity is permissible at a given separation distance between the COM and supporting foot. In addition, an alternate explanation could be that if subjects with TBI were to walk any faster with a significantly reduced stride length, the cadence would need to be uncomfortably high.

It appeared that a greater variability in the M-L COM motion was observed within subjects with TBI. This might be a result of a small sample size or a diverse range of brain injuries. However, magnitudes of the M-L COM motion of subjects with TBI were not found to correlate with either severity or duration of the brain injury. It might be also speculated that increases in the M-L COM motion in TBI subjects are associated with decreases in their gait speed. A further analysis of our data was performed to include the gait speed during unobstructed walking as a covariate. The results indicated that there was no significant association ($P = 0.582$) between the increase in M-L COM motion and decrease in gait speed. Furthermore, it has been reported recently [37] that, in young adults, walking with a faster or slower speed (ranges from 1.03 to 1.65 m/s) is associated with subtle changes in frontal plane COM motion. However, these gait speed-induced differences (approximately 1 cm displacement and 2.4 cm/s peak velocity) are much less than those differences detected between subjects in the TBI group and their controls (2.3 cm displacement and 5 cm/s peak velocity). Therefore, increases in the M-L COM motion in subjects with TBI are, at least in part, results of their inability to maintain dynamic stability in the frontal plane while interacting with environmental obstacles during walking. Consequently, subjects with TBI may have a higher risk of falling sideways during obstacle crossing.

In conclusion, the findings of this study demonstrate that examining the COM motion provides an objective measurement that reflects complaints of instability not observable with a clinical examination for individuals who have suffered a TBI. Inclusion of an obstacle-crossing task in the clinical gait analysis may enhance the assessment of dynamic instability in this population.

Acknowledgements

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