Static and Dynamic Postural Stability in Veterans with Combat-Related Mild Traumatic Brain Injury

Running title: Posture and Military Mild TBI

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ABSTRACT

Persistent post-concussive symptoms are reported by 10-15% of individuals who suffer mild traumatic brain injury (mTBI), but their basis is often uncertain. One such symptom is disequilibrium, a sensation of impaired balance during standing and walking. The hypothesis for this study was that this subjective symptom is associated with objective and measurable deficits in static and dynamic postural stability. An infrared motion tracking system was used to record body motion during quiet standing and in response to waist perturbations in fourteen veterans (age 22-40 years, 13 male) of the Operations Enduring Freedom (OEF) and Iraqi Freedom (OIF), who had a history of mTBI that occurred 7 months to 7 years prior to testing. We compared body sway between veterans with mTBI reporting persistent disequilibrium (T_D , $n=8$) and those with no vestibular symptoms ($n=6$), as well as to a group of non-veterans with no balance symptoms ($n=10$). Static postural stability was reduced in T_D veterans in comparison to each of the other two groups ($p < 0.0002$), most notably on a compliant surface with eyes closed. The T_D group also had decreased dynamic stability of the upper trunk ($p < 0.05$) and enhanced postural oscillations ($p < 0.02$) following waist perturbations. Our findings support a physiological basis for persistent disequilibrium after mTBI and are consistent with impaired vestibular processing. Disruption of semicircular canal inputs is unlikely to be the cause, as head impulse responses were normal in all groups. The unexpected finding of dynamic postural oscillations requires further study but may indicate enhanced instability in sensorimotor networks responsible for postural control.

INTRODUCTION

Mild traumatic brain injury (mild TBI) has been identified as a major morbidity of recent military conflicts. Although estimates vary, as many as 10% or more of U.S. service members deployed over the last decade to Iraq (Operation Iraqi Freedom, OIF) and Afghanistan (Operation Enduring Freedom, OEF) may have experienced combat-related mild TBI as a consequence of blast exposure and/or blunt head trauma [1,2]. Civilian TBI is similarly common, due to a variety of causes, such as athletic injuries, motor vehicle collisions, falls, assaults, and other accidental injuries [3].

Most civilians who experience mild TBI recover within several days. In about 10-15% of cases, however, recovery is delayed and symptoms persist [4,5]. Post-concussive symptoms are reported with a similar frequency after combat mild TBI [1,2]. To what extent these persistent symptoms are consequences of the mild TBI or due to other factors, such as post-traumatic stress disorder (PTSD) or depression, remains a matter of debate [6].

There are several reasons why it might be difficult to find physiological correlates to post-concussive symptoms. First, the symptoms reported, such as difficulty concentrating or headaches, tend to be non-specific and could result from a variety of diverse causes. Investigations that rely on symptom reporting rather than objective measures may be unable to distinguish among these etiologies. Second, deficits associated with post-concussive symptoms may be sufficiently subtle not to be detected on a routine neurological examination; more sensitive physiological measures may be required. Third, many studies have considered post-concussive symptoms together as a group rather than studying each individually [7,8]. Lumping post-concussive symptoms into symptom scales is useful for the identification and tracking of injuries [9], but it offers little insight into the pathophysiological mechanisms underlying specific symptoms.

Dizziness is an example of a common post-concussive symptom after combat mild TBI that

could be due to a number of causes, both physical and psychological [10,11]. For example, some individuals have episodic dizziness due to benign paroxysmal positional vertigo or vestibular migraine. In other cases, dizziness may be a somatic correlate of anxiety or depression. In this study, we have focused on one particular form of post-concussive dizziness, namely a chronic sensation of imbalance during standing and walking that we refer to as disequilibrium. A few studies that have assessed post-concussive symptoms after combat injuries have found an association of dizziness and imbalance with mild TBI that is independent of PTSD [2,12,13]. To elucidate further a potential physiological basis, we measured static and dynamic postural stability in OEF/OIF veterans with a history of mild TBI with and without chronic persistent disequilibrium.

MATERIALS AND METHODS

Subjects

Fourteen OEF/OIF veterans (age 22-40 years, median 26.5) with a clinical history of mild TBI were studied. The diagnosis of mild TBI was based on the subjects' self-report of a combat event involving blast exposure and/or blunt head trauma without penetrating injury that resulted in loss of consciousness for no more than 30 minutes, alteration of consciousness for no more than 24 hours, and/or post-traumatic amnesia for no more than 24 hours. One additional veteran was excluded from analysis because the history of injury was inconsistent.

Veterans with mild TBI were divided into those who reported ongoing chronic disequilibrium (T_D , $n=8$) and those who reported no disequilibrium (T_{ND} , $n=6$). All subjects in the T_{ND} group and all but one in the T_D group were male. At least one mild TBI included loss of consciousness in 4/8 of the T_D group and in 4/6 of the T_{ND} group. Post-traumatic stress disorder had been diagnosed in 7/8 of the T_D group and in 4/6 of the T_{ND} group. The duration

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2
3 since the most recent mild TBI ranged from 7 months to 7 years and was similar in both groups
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5 (p > 0.55, t-test).
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7 We also studied 10 non-veterans, who had no history of TBI and had no vestibular or
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9 neurological complaints. All subjects gave written informed consent under a protocol that was
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11 approved by the Institutional Review Boards of the Louis Stokes Cleveland Department of
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13 Veterans Affairs Medical Center and the University Hospitals of Cleveland Case Medical
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15 Center, as well as by the Human Subjects Protection Office of the U.S. Department of
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17 Defense.
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22 23 *Data Recording* 24

25 An infrared motion tracking system (Vicon, Oxford, UK) was used to record kinematics of
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27 body motion at the waist (reflective markers over the sacrum and each anterior superior iliac
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29 spine) and the upper trunk (markers over the spinous process of C7 and over each shoulder).
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31 Three-dimensional marker positions were sampled at 100 Hz.
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36 37 *Experimental Paradigms* 38

39 Subjects were studied in a series of tasks designed to test static and dynamic balance.
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41 Static tasks included quiet standing on the floor and standing on a compliant surface (10 cm
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43 thick foam), in both cases with eyes open and closed. Dynamic balance was tested with
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45 sudden postural perturbations delivered to the lower trunk via a computer-controlled linear
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47 motor coupled to the body through a taut rope attached to a belt worn at the waist. At an
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49 unpredictable time, the motor was commanded to generate a linear force equal to 10% of the
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51 subject's body weight; the force was sustained for 0.5 s. Because (for safety reasons) the force
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53 was transmitted by a non-rigid rope, the force profile felt by the subject was not identical to the
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55 force generated by the motor; thus, for analysis (see below) the acceleration of the pelvis was
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57 used to determine the time of force onset. Responses were measured to both forward and
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rightward perturbations. Peak accelerations were 0.31 +/- 0.11 g (mean +/- S.D.) for forward perturbations and 0.30 +/- 0.14 g for rightward perturbations. There was no difference in peak accelerations and, therefore, in the perturbation force felt by the subjects, between groups ($p > 0.5$).

Data analysis: static balance

Three-dimensional marker positions were reconstructed by the Vicon software. Subsequent analysis was performed using custom programs, written by the authors in Python and MATLAB™. Marker velocity was calculated by component-wise differentiation of marker position, after filling missing points by linear interpolation and low-pass filtering (interp1d and firwin routines in scipy, 15 Hz cutoff frequency, bidirectional filtering for zero phase shift). For each of the four conditions, we quantified postural stability by calculating the two-dimensional sway path (line integral of the marker position in the earth-horizontal plane) for the first seven seconds of the recording. Finally, we determined trunk angle as a function of time from the C7 and sacral marker positions. These angles were calculated based on the position of the C7 marker relative to the sacral marker. For example, the pitch angle $\theta_{\text{Pitch}} = \tan^{-1}[(x_{\text{C7}} - x_{\text{SACR}}) / (z_{\text{C7}} - z_{\text{SACR}})]$, where x is the fore-aft position and z is the vertical position of each marker.

Data analysis: dynamic balance

Marker positions and velocities were determined as for static balance. For each trial, the onset of the perturbation was determined from the acceleration of the sacral marker, using a thresholding technique (see Figure 5). Occasionally, the sacral marker or C7 marker was obscured during the recording; in these cases, the average motion of the RASI and LASI markers (for pelvis motion) or of the shoulder markers (for upper trunk motion) was used instead. Postural sway length and displacement were calculated for the first 2.5 s after the onset of each perturbation.

Head Impulse Testing

The vestibulo-ocular reflex was recorded on the same day as postural testing. Eye movements were recorded using either scleral coils or video-oculography in response to manual head impulses. The VOR gain was calculated in MATLAB™ based on a least-squares optimization of eye to head velocity. Gains were determined separately for each direction of rotation.

Statistical analysis

Statistical testing was performed using multivariate repeated-measures ANOVA based on a linear mixed-effects model in R (lme in nlme package, www.cran.r-project.org). Response measures were subject to power transformation to more nearly approximate normality, based on Box Cox analysis (boxCox routine in R car package). Between-groups factors included history of TBI and history of disequilibrium. Depending on the specific paradigm, within-subjects factors included standing surface, eyes open vs. closed, perturbation direction, head orientation, and frequency. Post-hoc comparisons for subject group were performed using the R routine *glht* with the Tukey method.

RESULTS

Veterans with mild TBI and disequilibrium have decreased postural stability while standing

Figure 1 depicts using data ellipses the combined postural sway of all subjects in each group while standing. Each plot is based on the positions of the sacral marker in the two-dimensional horizontal plane during the first seven seconds of the trial. In all conditions, the positions of the T_D group cover a wider area; this difference is markedly increased when the eyes are closed and during standing on a compliant surface. Overall, there is more sway in the

sagittal (fore-aft, anterior-posterior) plane than in the coronal (left-right) plane.

In addition to comparing pelvis positions across groups, we also calculated the total length of the sway path in two dimensions (Figure 2A). Sway path length was longest for the T_D group, especially for the most difficult condition, standing on the compliant surface with eyes closed. Based on the mixed effects model, there was a significant effect on sway path of subject group ($p < 0.0002$), of eye closure ($p < 0.0001$), and of standing surface ($p < 0.0001$). Post-hoc comparison of groups found T_D to be different from both T_{ND} ($p < 0.005$) and N ($p < 0.001$). We also investigated the stability of the upper body during standing by calculating the range of pitch (Figure 2B) and roll trunk angles (Figure 2C). Subjects with mild TBI and disequilibrium had considerably larger maximal sway deviations of the trunk. In the linear model, there was a significant effect of group for both pitch and roll tilt ($p < 0.0005$). In the post-hoc comparison, T_D was again different from both T_{ND} and N ($p < 0.0001$).

Veterans with mild TBI and disequilibrium have decreased postural stability when perturbed with eyes closed

Representative responses to abrupt forward postural perturbations are shown in Figure 3 for veterans with mild TBI with and without disequilibrium and for a subject with no history of TBI. The plots show forward acceleration and velocity as a function of time and 2-D trajectory of position of the pelvis. In the non-TBI subject, responses were very stereotyped (there was relatively little intra-trial variability), and the postural sway was small and essentially limited to the fore-aft direction (along the pull axis). In contrast, the two TBI subjects (especially the T_D subject) showed more variability and longer sway paths, and there was not only fore-aft but also lateral sway, in the direction orthogonal to the perturbation.

Similar to standing posture, we calculated the lengths of the 2-D sway paths at the pelvis and upper trunk for the first 2.5 s after the perturbation (Figure 4). With eyes closed, there was

a significant effect of group on the length of the upper trunk sway path (Figure 4, bottom panels, $p < 0.03$), but the group effect for pelvis motion was not significant ($0.09 < p < 0.10$). For upper trunk sway, the post-hoc analysis found a difference between T_D and N ($p < 0.05$) and between T_D and T_{ND} ($p < 0.02$).

Veterans with mild TBI and disequilibrium have enhanced postural oscillations following passive perturbations

As illustrated by the examples of Figure 3, T_D subjects demonstrated small amplitude postural oscillations following perturbations that were most easily seen in the marker acceleration traces. To characterize these oscillations, we calculated an FFT on the pelvis marker data, averaging the results for similar trials in a given subject (Figure 5). Most of the power of these oscillations was concentrated below 2 Hz, but there were also higher frequency components. Considering summed power from 2-10 Hz, there was a significant effect of group for forward perturbations ($p < 0.02$) but not for lateral perturbations ($0.09 < p < 0.10$). The post-hoc analysis for forward perturbations found a difference only between groups N and T_D .

Vestibular function in veterans with disequilibrium

Clinical vestibular testing was not a part of the study design, but most (6/8) subjects in the T_D group had been previously referred by their physicians to the audiology service for balance and vestibular assessments (Supplementary Table 1). Although there were isolated abnormal findings in some subjects, there was not a consistent pattern of vestibular impairment in this group.

All subjects in the study, except for one T_{ND} subject, had laboratory recordings of eye movements in response to head impulses. There was no difference in the VOR gain ($p > 0.35$) or symmetry ($p > 0.95$) across the three groups (Supplementary Figure 1).

DISCUSSION

In this study, we found that veterans who experienced mild TBI due to combat trauma and who report continued disequilibrium have measurable physiological deficits of balance, even months to years after their injuries. They have increased postural sway, both during quiet standing and when suddenly perturbed. The combination of an increased range of pelvis sway and larger upper body tilts abnormally displaces the center-of-mass, potentially increasing the risk of falling when balance is challenged. The fact that stability is much worse when vision and proprioceptive feedback are disrupted by eye closure and a compliant surface also provides further indirect support for a role of vestibular dysfunction in chronic mild TBI-related balance impairment.

There have been several previous studies of balance after mild TBI, typically in the context of sports-related or accidental injuries. Unlike our veterans, these individuals can be studied within hours to days of their injuries, and it has been shown that balance impairment is common after acute mild TBI [14]. In most cases, these deficits recover within a few days of a concussion [15,16], but there is evidence of persistent postural instability in some cases of mild TBI [16-18], especially when more complex measures of postural control are included [19]. Our findings are consistent with this prior work and show that chronic postural deficits can persist for months to years and can be symptomatic. Although mild, they are likely to have a meaningful functional impact in challenging environments, including combat, where higher cognitive demand may further degrade balance through dual-task effects [20].

Are combat-related post-concussive symptoms due to mild TBI?

Post-concussive symptoms, including dizziness and imbalance, are not uncommon among combat veterans with mild TBI. As reviewed in the Introduction, to what extent these symptoms are due to physiological and/or psychological causes has been debated. Although our findings

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3 may not apply to all post-concussive symptoms, they do suggest that at least some symptoms,
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5 and post-concussive disequilibrium in particular, are rooted in a physiological impairment that
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7 may be a direct consequence of the mild TBI. Psychological factors may contribute to or
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9 intensify the symptom and its functional impact, but they are unlikely to be the only cause of
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11 the symptom. Moreover, imbalance in the T_D group scaled with task difficulty and was minimal
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13 with eyes open while standing on the firm surface. This argues against malingering as an
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15 explanation for these veterans' symptoms and the associated postural instability.
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20 *Implications for localization of TBI-related balance impairment*

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23 Prior studies have suggested that mild TBI may result in both peripheral and central
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25 vestibular dysfunction, ranging from direct labyrinthine damage to impairment of multi-sensory
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27 integration, and that the pattern of injury may differ among individuals [16,21-26]. Similar to
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29 these prior reports, we also did not find a consistent pattern of peripheral vestibular impairment
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31 in the T_D group. Moreover, all three groups had normal head impulse responses, suggesting at
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33 least that disturbed semicircular canal function does not play a major role in mTBI-related
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35 disequilibrium.
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40 In many cases, the injury may be multifocal, and this may be especially true for combat
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42 mild TBI that combines both blast and blunt head trauma. One brain area that may be playing
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44 a role in postural instability is the cerebellum. For example, similar to our findings, patients with
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46 cerebellar degeneration, particularly affecting the anterior lobe (superior vermis), demonstrate
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48 increased postural instability that is greater in the fore-aft (anterior-posterior) direction [27,28].
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50 Damage to the cerebellum in mild TBI is supported by recent reports of axonal injury to the
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52 middle cerebellar peduncles in acute military TBI [29] and of cerebellar hypometabolism in
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54 veterans with blast mild TBI and post-concussive symptoms [30].
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59 A novel finding of our study is that veterans with mild TBI demonstrated high-frequency
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2 oscillations in responses to postural perturbations. We are not aware of prior reports of such
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4 oscillations after mild TBI, although this may be related to a separate finding of decreased
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6 entropy in postural sway signals [19]. The mechanism for such oscillations is uncertain, but it
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8 suggests that mild TBI leads to increased instability in sensorimotor postural control loops,
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10 enhancing oscillations that may occur even in neurologically normal individuals when there is
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12 an abrupt change in the sensory context [31].
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18 *Limitations and Conclusions*

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20 In combat mild TBI, the mechanisms of injury are complex and due to a variable
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22 combination of blunt and blast forces. Moreover, records of the event and initial neurological
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24 assessment are generally not available, so the diagnosis of TBI must be made based on the
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26 veteran's self-report of the traumatic event. Thus, it is not possible from our data to make
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28 interpretations based on injury type or severity or to identify factors that might predict which
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30 individuals will recover after an mild TBI and who will go on to have persistent imbalance.
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32 Other limitations of the study include the small sample size and the wide range of intervals
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34 between the times of the injury and balance testing. Despite these limitations, our data provide
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36 additional strong evidence that chronic disequilibrium after mild TBI is not simply a
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38 psychological symptom but is associated with physiological deficits of static and dynamic
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40 postural control. Further research is needed to clarify the nature and sites of injury contributing
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42 to this imbalance, the natural history of these deficits from the time of acute injury, and the role
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44 of directed rehabilitation in recovery of balance and resolution of symptomatic disequilibrium.
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FIGURE LEGENDS

1. Data ellipses combining sway path data for all subjects in each group: non-TBI (N), TBI with disequilibrium (T_D), and TBI without disequilibrium (T_{ND}). The red ellipses represent the central 25% of all data, yellow ellipses 50%, green ellipses 75%, and cyan ellipses 95%. For each group and condition, ellipses were calculated using R (data.ellipse function in car package, www.cran.r-project.org). The analysis takes into account differences in absolute positions among subjects by subtracting the initial marker position from the sway path positions before combining the data.
2. **A.** Box and whisker plot of 2-D sway path length, calculated by integrating the position trajectory of the sacral marker, for the first 7 seconds of recording, for all subjects. Data are shown for each of the four conditions: standing on floor or foam with eyes open (EO) or closed (EC). For each data column, the gray bar covers the middle quartiles (25-75%), and the line shows the median. Whiskers contain the remaining data points, up to 1.5x the size of the middle quartile range. Points beyond this are shown as individual outliers (+). Under all conditions, median postural stability was lower (sway path was longer) for the mild TBI group with disequilibrium (T_D) than for the normal subjects (N) and the mild TBI group with no disequilibrium (T_{ND}). See text for statistical results. **B and C.** Box-and-whisker plots showing upper body sway during standing, represented as the trunk angle range (maximum – minimum) in the sagittal (pitch angle) and coronal (roll angle) planes. These angles were calculated based on the position of the C7 marker relative to the sacral marker. For example, the pitch angle $\theta_{Pitch} = \tan^{-1} [(x_{C7} - x_{SACR}) / (z_{C7} - z_{SACR})]$, where x is the fore-aft position and z is the vertical position of each marker. TBI subjects with disequilibrium (T_D) had the largest trunk angles, especially with eyes closed and on foam. See text for statistical results.
3. Responses to abrupt postural perturbations at the waist in a veteran with mild TBI and

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2 disequilibrium (top row), a veteran with mild TBI but no disequilibrium (middle row), and
3 non-TBI subject (bottom row). Each row shows responses for a series of similar trials
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5 (forward perturbations, head straight ahead, eyes closed). The left panels show the forward
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7 acceleration and the middle panel the forward velocity of the sacral marker as a function of
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9 time; trials are aligned on the onset of the pull as determined from the acceleration. The
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11 right panels show the 2-D sway path of the sacral marker referenced to the starting
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13 position.
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- 20 4. Box-and-whisker plots of 2-D sway path length for a range of time intervals (0.5-2.5 s)
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22 following perturbation onset, for both forward (left panels) and rightward (right panels)
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24 perturbations. The top panels represent motion at the pelvis, and the bottom panels sway
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26 of the upper trunk. The group designations are the same as for Figure 2. See text for
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28 statistical results.
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32 5. Power spectral density (0-10 Hz) of the response to abrupt postural perturbations during
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34 the first 2.5 seconds after perturbation onset. Each panel represents the data from one of
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36 the three subject groups, shown at the same scale. The solid line depicts the median of all
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38 subjects' data, and the shaded area represents the middle two quartiles. These data are
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40 from trials with eyes open. In our statistical model, there was no effect of eye closure ($p >$
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42 0.34). See text for additional statistical results.
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Figure 1

Figure 2

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Figure 3

Figure 3

Figure 4

Figure 4

Figure 5