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Abnormal muscle activation patterns are associated with chronic gait deficits following traumatic brain injury

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ABSTRACT

Background: Gait and balance disorders are common among individuals who have experienced a mild to moderate traumatic brain injury (TBI). However, little is known about how the neuromuscular control of gait is altered following a TBI.

Research question: Investigate the relationship between lower limb muscle activation patterns and chronic gait deficits in individuals who previously experienced a mild to moderate TBI.

Methods: Lower extremity electromyographic (EMG) signals were collected bilaterally during treadmill and overground walking in 44 ambulatory individuals with a TBI > 1 year prior and 20 unimpaired controls. Activation patterns of TBI muscles were cross-correlated with normative data from control subjects to assess temporal phasing of muscle recruitment. Clinical assessments of gait and balance were performed using dynamic posturography, the dynamic gait index, six-minute walk test, and preferred walking speed.

Results: TBI subjects exhibited abnormal activation patterns in the tibialis anterior, medial gastrocnemius, and rectus femoris muscles during both overground and treadmill walking. Activation patterns of the vastus lateralis and soleus muscles did not differ from normal. There was considerable heterogeneity in performance on clinical balance and gait assessments. Abnormal muscle activation patterns were significantly correlated with variations in the dynamic gait index among the TBI subjects.

Significance: Individuals who have experienced a prior TBI do exhibit characteristic changes in the temporal coordination of select lower extremity muscles, which may contribute to impairments during challenging walking tasks.

1. Introduction

Individuals with prior mild to moderate traumatic brain injury (TBI) often report difficulties with balance and walking. Unfortunately following the first few months of recovery, there is often little improvement in walking ability [1]. It is generally believed that motor patterns become relatively fixed after this period, such that residual gait deficits become chronic [2–4]. Among TBI survivors, these chronic gait deficits have been linked to falls, a loss of mobility, and decreased quality of life [1,5,6]. Thus, there is a need to understand the motor patterns underlying chronic TBI gait. This understanding could help in identifying targets for treatment and evaluating the efficacy of emerging neurorehabilitation protocols, e.g. noninvasive neuromodulation [7], that are designed to induce fundamental shifts in the sensorimotor control of gait.

Prior studies have used clinical and quantitative gait analysis protocols to describe chronic TBI gait. Such studies consistently observe marked heterogeneity among the degree of balance and gait impairment present [3,8–12]. This heterogeneity has been linked to both self-reported balance problems and falls [13,14]. For example, individuals with a prior TBI who exhibited diminished scores on the Dynamic Gait Index (DGI) [15], a clinical assessment of an individual's ability to walk under challenging conditions, were identified as being at risk for falls [14]. Spatiotemporal aspects of gait also differ, with TBI being linked to a tendency to walk more slowly, take shorter steps, and exhibit greater mediolateral sway [8,16,17]. Joint kinematics of the lower extremity has also been shown to accurately classify a range of TBI-related gait disorders [3,8,18]. Thus, there seems to be a role for quantitative gait analysis to provide insights into gait deficits associated with prior TBI. However, to our knowledge, previous studies have not analyzed the

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underlying muscle coordination patterns that give rise to observed gait dynamics following TBI.

The purpose of this study was to investigate lower limb muscle activation patterns during walking in individuals who experienced a TBI more than one year prior. Muscle activation patterns were compared to those of healthy controls [19] to identify muscles which most often exhibit deviations from normal temporal phasing. We also assessed if abnormal muscle activation patterns were associated with clinical assessments of gait and balance.

2. Methods

2.1. Participants

Forty-four people with a balance disorder as a result of a traumatic brain injury (age: 53.4 ± 8.5 years, range: 28–64 years; 28 females; time since injury: 6.3 ± 7.6 years, range: 1–33 years) and twenty control subjects (25.3 ± 3.3 years; 10 females) participated in the study. This protocol was approved by the University of Wisconsin–Madison Health Sciences Institutional Review Board, and all subjects provided written informed consent before participating. We only recruited individuals with a mild to moderate non-penetrative TBI, as confirmed by a "non-remarkable" neurological report from their medical records (i.e. no loss of brain matter or evidence of refractory hematoma, etc.), with a loss of consciousness < 24 h. Each individual was at least one year post-injury, had completed a focused physical rehabilitation program for their TBI, and felt that he/she had reached a plateau in recovery. Participants were community dwelling and had normal gait and balance prior to their brain injury. Further inclusion for the TBI group were: ambulatory and able to walk independently for 20 min; a sensory organization test (SOT) [20] composite score at least 8 points below the lower limit of normal (i.e. mean – 1.67*SD, adjusted for height and weight); absence of any other neurological disorder besides those attributed to their TBI, and no changes in health or medications in the 3 months prior to participation. Unimpaired control subjects had no history of neurological or musculoskeletal disorder or injury, had no contraindication to exercise, and were not at-risk for cardiovascular events. For complete inclusion/exclusion criteria, see Supplementary information.

2.2. Clinical assessment

2.2.1. Sensory organization test (SOT)

Computerized dynamic posturography assessed standing balance under six conditions challenging the visual, vestibular, and proprioceptive systems (NeuroCom Intl., Clackamas, OR). Three consecutive 20-s trials were performed while standing barefoot on a force platform under each condition: (1) eyes open; (2) eyes closed; (3) eyes open, sway-referenced visual surround; (4) eyes open, sway-referenced platform; (5) eyes closed, sway-referenced platform; (6) eyes open, sway-referenced platform and visual surround. An overall composite score (0–100) was calculated from body sway.

2.2.2. Dynamic gait index (DGI)

Gait ability was assessed using the Dynamic Gait Index [15]. Subjects performed eight functional walking tasks that included normal overground walking, changing gait speeds, walking with head turns, walking while turning, walking over and around obstacles, and stair climbing. Each task was scored 0–3 by a trained physical therapist, where 3 indicates normal. DGI scores ≤19 have been linked to falls for individuals with TBI [14].

2.2.3. Six-minute walk test (6MWT)

Walking capacity was assessed using the six-minute walk test [21,22]. Subjects were instructed to walk along a quiet, level hallway as fast as they could safely for six minutes. The hallway formed a continuous circular path such that no sharp turns were required. The test administrator followed each subject's path with a measuring wheel and recorded the total distance walked.

2.2.4. Preferred walking speed (PWS) and treadmill speed (PTS)

Each subject's preferred overground walking speed was measured as subjects walked two times down a six-meter walkway at a "normal, comfortable pace". Speed was calculated from the average time to cross the middle four meters of the walkway. Preferred walking speed has shown excellent test-retest reliability among TBI participants [23]. TBI subjects were put on the treadmill (Bronze Basic Treadmill, PaceMaster, Logan, UT) and the speed was initially set to match their PWS. Some subjects were unable to comfortably match their PWS on the treadmill. For these subjects, we lowered their treadmill speed to the highest speed where they felt they could walk comfortably. This speed was defined as the preferred treadmill speed (PTS).

2.3. Electromyographic collections during overground and treadmill walking

Lower limb electromyographic (EMG) activities were recorded bilaterally while subjects performed two 60-s walking trials: one on a treadmill at their PTS, and another while walking at a comfortable pace down a level hallway with no sharp turns. Two subjects exhibited difficulty walking on the treadmill even at very slow speeds and were allowed to hold on to the handrails for support. Healthy subjects walked on the treadmill at their preferred walking speed (1.2 ± 0.2 m/s), and at a slower speed (1.0 m/s) that more closely matched the average PTS of the TBI subjects.

Surface electrodes (Trigno™ Wireless EMG: 4 bar contacts, 99.9% Ag, 5 × 1 mm, 10 mm inter-electrode distance, CMMR > 80 dB, signal-to-noise ratio < 0.75 μV) were placed over the tibialis anterior (TA), medial gastrocnemius (MG), soleus (SL), vastus lateralis (VL), rectus femoris (RF), and medial hamstrings (MH) muscles. After the skin was shaved and cleansed with an isopropyl alcohol swab, electrodes were coated with conductive gel and placed over the muscle bellies in line with fiber orientation. The standard EMG electrode locations were determined by the same investigator for each subject. EMG activities were recorded using a wireless system (Trigno™ Personal Monitor; Delsys Inc., Boston, MA). Heel-strike events were detected using the accelerometers of two Trigno sensors positioned at the ankle over the Achilles tendons [24,25]. All sensors were secured with elastic wrap. EMG and accelerometer signals were digitally sampled at 1926 and 148 Hz, respectively.

The EMG and accelerometery signals were processed using a custom MATLAB script (R2017a, MathWorks Inc., Natick, MA). Accelerometery data was low-pass filtered at 25 Hz using a 3rd order Butterworth filter, and heel strikes were identified from magnitude peaks in the filtered data [26,27]. EMG data was band-pass filtered (1–350 Hz) using a 4th order Butterworth filter, then full-wave rectified and low-pass filtered at 10 Hz to obtain the linear envelopes of muscle activation. Muscle activity over each gait cycle were extracted and time normalized to 101 points per stride. Muscle activation patterns were then ensemble averaged over all consecutive strides in the 60-s trials. Ensemble averaged muscle activation patterns were amplitude normalized to their root-mean-squared to represent the underlying fixed muscle activation present in each subject. We also assessed the EMG variability over consecutive strides using the coefficient of variation (CV), defined as the root-mean-square of the EMG standard deviation over the gait cycle, divided by the mean ensemble average [28]. The control subject EMG activities were processed similarly and averaged across subjects to create a comparative activation profile for each muscle.

2.4. Comparing activation patterns between TBI and control subjects

The timing and shape of individual muscle activation patterns were compared to normal using an EMG cross-correlation method [19].