Running abnormalities after traumatic brain injury

ABSTRACT

Primary objectives: The aim of this study was to identify the type and incidence of running abnormalities following TBI when compared to a group of healthy controls (HC), and report if these abnormalities were similar to those which are present during gait.

Research design: A convenience sample of 44 people with TBI receiving therapy for mobility limitations, and a sample of 15 healthy controls (HCs).

Main outcomes and results: Spatio-temporal, kinematic and kinetic data at self-selected walking and running speeds were collected. People with TBI ran at significantly slower self-selected speeds than HC. At matched running speeds, people with TBI used a higher cadence and shorter step length. The most commonly observed biomechanical abnormalities occurred at the knee during stance phase. Few trunk, pelvic or hip abnormalities were detected. Ankle power generation at push-off was significantly reduced whereas hip extensor power generation at initial contact was significantly increased.

Conclusion: Many people with TBI may actually be capable of running despite the presence of significant biomechanical abnormalities during gait. A stable trunk may be an important requirement for people following TBI to achieve running.
Traumatic brain injury (TBI) is a leading cause of death and disability for adolescents and young adults. Mobility limitations are prevalent and surprisingly little information is known about the effect of TBI on mobility. The key biomechanical abnormalities of gait following TBI have only recently been described. Williams et al. (2009) reported kinematic abnormalities which were particularly prevalent in all three planes of movement around the trunk and pelvis. Excessive knee flexion at initial foot contact was the most frequently observed biomechanical abnormality. Gait abnormalities that have been previously been cited in the TBI literature, such as ankle equinovarus, were found to occur infrequently.

However, gait is only one part of the mobility spectrum. Determinants of outcome following TBI include the ability to participate in a wide variety of social, leisure, sporting and employment activities, all of which may require higher levels of mobility, such as the ability to run. For example, Lindstrom et al. (2009) found that young stroke survivors were nearly three times more likely to return to work if they regained the ability to run, even if for only a few steps. The relationship between running and participation rates for social, leisure, sporting and employment activities has not been established in TBI. Despite the importance of running for participation in many day to day activities, the ability to run, or methods for retraining running are rarely reported.

Little is known about the biomechanical abnormalities limiting high-level mobility following TBI. One reason for this may be that the majority of outcome measures used to quantify mobility do not include higher level tasks such as running, jumping or hopping. Another reason may be that higher level mobility is most often treated on an outpatient basis where funding resources may be restricted. As far as we are aware, no studies have reported the biomechanical abnormalities associated with running following TBI.

To the best of our knowledge, only four published studies, representing five single case studies, have reported running training following neurological injury. Three papers involved single case designs describing the use of partial body weight support on a treadmill to
improve running ability for incomplete spinal cord injury, stroke and TBI. More recently, Williams & Schache (2010) reported a conceptual framework for retraining running following TBI based on the mobility hierarchy outlined in the HiMAT and task-specific practice. The hierarchical ordering of the mobility items within the HiMAT identifies the order of difficulty associated with tasks. For example, it outlines that after independent walking is achieved, the next challenging task is ascending stairs, followed by bounding and then running. This hierarchical ordering is used for goal setting and treatment planning. The task-specific practice is based on knowledge of the biomechanics of able-bodied walking and running. For example, joint kinematic data obtained from able-bodied running is used to guide exercise prescription with reference to the required range of motion and angular velocities.

Similarly, joint kinetic data were used to assist in the identification of muscle groups for targeted strengthening programs. The two case studies illustrated how the conceptual framework could be applied for people with contrasting movement disorders; in this instance one person with hemi-spasticity and one person with ataxia. Williams & Morris (2009) also used this conceptual framework to investigate the feasibility of retraining high-level mobility in a cohort of 28 people with a variety of neurological conditions including TBI and stroke. It was demonstrated that clinically significant improvements in high-level mobility can be achieved, even for patients with conditions of longstanding duration. While studies have reported the key biomechanical abnormalities during gait following TBI and other neurological populations, we are unaware of any studies to date that have investigated the typical abnormalities associated with running following TBI.

Three dimensional gait analysis (3DGA) is the current gold standard for evaluating gait disorders. It has been used to identify the main biomechanical abnormalities of gait following TBI, and has had a positive impact on clinical evaluation and surgical intervention in cerebral palsy, and stroke. To date, 3DGA has not been used to quantify the key biomechanical abnormalities associated with running following TBI. The identification, targeted assessment and treatment of these abnormalities are pivotal for improving higher level mobility outcomes.
for people following TBI. The aim of this study therefore was to identify the type and incidence of running abnormalities following TBI when compared to a group of healthy controls (HC), and determine if these abnormalities are similar to those which are present during gait.

METHODS

This project was approved by Epworth Hospital’s HREC (study number 34006), and the University of Melbourne (Ethics ID: 060496.1). It formed part of a broader study investigating mobility limitations following TBI, and the methodology has been previously reported in detail.³

Participants

People with TBI were recruited for this project from Epworth Hospital, Melbourne, Australia. Patients currently attending physiotherapy for mobility limitations following TBI were asked to participate in this project. The inclusion criteria were patients who (a) had sustained a TBI and; (b) were able to run independently over a distance of 20 m. Exclusion criteria were: (a) patients who were unwilling or unable to provide informed consent; (b) those who presented with concurrent central nervous system disorders and; (c) those with severe cognitive or behavioral problems that prevented assessment. All patients who were invited to participate consented to do so. Data were also collected for comparative purposes from a sample of HCs, recruited via staff, family and friends. The HCs were matched for sex, age (+/- 5 years), height (+/- 7cm) and weight (+/- 15kg) to the first 15 participants with TBI. All HCs were screened for pre-existing conditions which may impact on ability to run. Table 1 summarizes the demographic data for the TBI and HC samples.

Instrumentation

Three dimensional gait analysis was performed at the Centre for Health, Exercise and Sports Medicine, in the School of Physiotherapy at The University of Melbourne. Kinematic data
were acquired using a motion analysis system (Vicon 512, Oxford Metrics, Oxford, England) with eight cameras sampling at a rate of 120 Hz. Ground reaction force data were collected using three force plates (Advanced Mechanical Technology Inc., Watertown, MA, USA) sampling at a rate of 1080 Hz.

**Procedures**

Twenty-five small (14 mm diameter) passive reflective markers were mounted on the skin at specific locations on the pelvis and both lower limbs following a previously described protocol. Three markers were also placed on the trunk overlying the spinous processes of T2 and T10 as well as the sternal notch. Subjects initially performed a standing calibration trial, with additional markers placed bilaterally on the medial femoral condyle, medial malleolus and proximal calcaneum of both legs. These markers were used to define joint centre locations and anatomical coordinate systems. The hip joint centre was found using the method of Harrington et al.

Participants with TBI performed walking and running trials over a 15 metre walkway whilst data were collected at their self-selected speeds. Spatio-temporal, kinematic and kinetic data for five trials were collected for each lower limb to gain a representative sample for each condition. In order to control for the effect of speed on kinematic and kinetic data, the HCs walked and ran at a speed comparable to the mean (+/- 5%) TBI self-selected speeds. Healthy controls were given verbal feedback regarding the accuracy of the matched speed. Only trials within 5% of the mean TBI self-selected walking and running speeds were included. Five trials with complete data were collected for each variable on each leg for every HC to generate the normative values for comparative purposes. Healthy controls also performed five self-selected walking and running trials in order to compare spatio-temporal data.

Due to the large number of gait variables that could potentially be investigated, a prioritized list of 24 variables (see Tables 4 & 5) was identified for this study. These variables were generated from a review of known gait abnormalities following TBI, the key gait variables for
normal walking and running, and specific variables which have been identified as problematic in other neurological populations. Several additional kinetic variables were also prioritized, based on the recent findings of Williams et al. (2010) which showed people with TBI compensate for distal power generation deficits with increased hip power generation when walking at faster gait speeds. Only data for participants capable of performing the walking and running trials are reported here.

In addition to 3DGA, TBI participants performed a clinical assessment of high-level mobility. The high-level mobility assessment tool (HiMAT) was chosen as it is the most responsive measure of high-level mobility for people with TBI.

Data Analysis

Three-dimensional joint kinematic and kinetic calculations were performed using Bodybuilder software (Oxford Metrics, Oxford, England). All lower limb joint kinematic and kinetic data were computed using a previously described approach. The three markers mounted on the trunk were used to define a local coordinate system, and the angular orientation of the trunk was described with respect to the pelvis. The model for the trunk was only available after the first 12 participants with TBI were recruited, therefore Tables 4 & 5 only report data for 32 participants.

Summary statistics (mean, standard deviation and range) were generated for all of the key running variables. Comparisons to the HC sample were made using a t-test. Due to the large number of comparisons (24 variables of interest), a Bonferroni adjustment was made and the alpha level for significance was set at 0.002. Values for the spatio-temporal variables were compared to data collected from a sample of 15 matched HCs walking and running at their self-selected speeds. Values for the kinematic and kinetic variables were compared to data collected from a sample of 15 matched HCs walking and running at speeds matched to the mean speeds for their TBI counterparts. Data for the TBI cohort were are reported for the more affected leg, while the mean of both legs was calculated for HCs.
In addition to comparing group data, individual results for the TBI sample were compared to the 95% confidence intervals calculated for each variable for the HC sample. All values within +/- 2 standard deviations (SD) from the mean were categorized as normal. Those values greater than 2 SD from the mean were categorized as being increased and those values less than 2 SD from the mean were categorized as being decreased.

**RESULTS**

The 44 participants with TBI were predominantly young (mean 27.9 ± 10.1 years) and male (36 men, 8 female), consistent with the broader TBI population (Table 1). All participants except one had sustained an extremely severe TBI, determined by the length of post-traumatic amnesia (PTA). The length of time post-injury varied considerably in this sample. No significant difference was identified between the TBI and HC samples for age, height or weight.

The TBI and HC summary statistics are outlined in Tables 2 & 4. When running, people with TBI used a higher cadence and shorter stride length to attain a similar speed to HCs (Table 2). In addition to the higher cadences, people with TBI ran with reduced stance and flight phases. Although stance time on the affected leg was significantly reduced, Table 3 shows that the majority of people with TBI had a stance time within the range obtained from the HCs. Width of base of support and lateral COM displacement (Table 4) were increased, indicating postural instability.

No significant difference was identified between the TBI and HC groups for trunk, pelvic or hip movements, except total range of pelvic axial rotation for which 29.5% of the TBI cohort had greater amplitude of axial rotation during the running cycle. Participants with TBI were more flexed at the knee at initial contact, more extended in mid-stance and then more flexed at toe-off (Table 4 & Figure 1), indicating a dampening of normal sagittal plane knee kinematics during stance phase. This finding was associated with significantly reduced knee power absorption in early stance phase (Table 4). Despite excessive knee flexion at toe off,
there was no significant difference between TBI and HCs for maximum knee flexion during swing. Although 77.3% of participants with TBI had peak knee flexion during swing within normal limits, 68.1% of these patients had excessive knee flexion during swing when walking. Ankle power generation at push-off was significantly reduced, whereas hip extensor power generation in early stance significantly increased compared to HCs (Figure 2).

Table 5 was generated to determine if abnormalities that occurred during running also occurred during walking. Tables 4 and 5 demonstrate that during running 30 of the 31 participants with TBI displayed normal trunk flexion/extension profiles but during walking the same was true for only 17 of the 31 participants. For other movements of the trunk, pelvis and hip, parameters which were normal or increased during running were classified similarly during walking. The knee demonstrated considerable variability in relation to whether movements classified as normal when running were also normal when walking. For example, 23/44 (52.3%) of participants had excessive knee flexion at toe-off when running, yet when walking 26.1% of these participants had reduced knee flexion, 43.5% had normal knee flexion and 30.4% had excessive knee flexion at toe-off when walking. A similar finding was evident at the ankle when running, where dorsiflexion was classified as normal at initial contact for the majority of people with TBI, yet nearly half had abnormally increased or decreased ankle dorsiflexion when walking. In relation to muscle power generation, there was a trend for excessive hip power generation in early and late stance during walking to become within normal limits when running.

**DISCUSSION**

This sample of people with TBI, although able to achieve running, experienced considerable mobility limitations. The mean self-selected walking speed was significantly reduced compared to the HC cohort and none of the TBI cohort achieved a HiMAT score within the normative range. In contrast to TBI gait where cadence and step length were not significantly different to HCs at a matched speed, this cohort of people with TBI used a
significantly higher cadence and shorter step length to attain a matched running speed. When asked to walk at faster gait speeds, people with TBI have demonstrated their ability to increase cadence and step length.\(^{34}\) However, these results indicate the ability to further increase affected step length for running appears to be limited. The majority of participants in this study (75.0%) had significantly increased knee flexion at initial contact. An excessively flexed knee at initial contact limits step length, and excessive knee flexion at initial contact was one of the most common kinematic abnormalities found in TBI gait.\(^3\) Given 77.3% of participants had maximum knee flexion within normal limits during swing phase, further examination of the role of hamstrings in terminal swing is warranted. Postural instability, whilst prevalent when walking following TBI,\(^3, 34, 38-40\) did not prevent this cohort from being able to run, despite them having a significantly greater lateral COM displacement and wider of base of support (Table 4).

Trunk and pelvic abnormalities were reported to be common during gait following TBI.\(^3\) However, in this sample of people with TBI the majority of participants had normal trunk and pelvic movement, even when this wasn’t the case when walking (Table 5). This finding indicates that the control of trunk and pelvic movements may be one of the key criteria for the ability to run, even if they may appear to be abnormally increased or decreased whilst walking. The only trunk, pelvic or hip movement which was identified as being significantly different to HC's when running was the range of motion (ROM) of pelvic axial rotation. Although the average pelvic axial rotation position was not significantly different to normal, indicating no gross asymmetry, people with TBI moved through greater amplitude of pelvic axial rotation when running (Table 4 & 5). Running at slower speeds is associated with less pelvic axial rotation compared with walking,\(^{41}\) so the finding of increased amplitude of pelvic axial rotation may be a compensatory strategy for excessive knee flexion at initial contact and reduced step length. The data indicate that stable trunk and pelvis may be required in order to run following TBI, as some people with abnormal trunk, pelvic or hip movement when walking actually ‘normalized’ their movement during running.
As with TBI gait, these findings identified significantly altered knee kinematics during stance phase. Figure 1 and Table 4 demonstrate a dampening of normal sagittal knee kinematics during stance phase. That is, excessive knee flexion at initial contact, followed by insufficient knee flexion in mid-stance and greater knee flexion at toe-off. This finding indicates that despite excessive initial knee flexion, some people with TBI experience difficulty with eccentric knee extensor muscle function required for knee power absorption during the loading response phase. Although ‘stiff-legged’ swing phase is reported to be common during gait following TBI, only 11.4% and 15.9% of participants had reduced knee flexion at toe-off and during swing phase, respectively.

During walking, forward progression is primarily provided by ankle power generation at push-off, and this contribution increases with increasing gait speed in unimpaired young adults. Ankle power generation at push-off was significantly reduced when running, a finding which is consistent with walking and fast walking following TBI. Increased hip flexor and extensor power generation has been suggested as a compensatory strategy for reduced ankle power generation following TBI. Similar results have been found following stroke where reduced ankle power generation is associated with a compensatory increase in hip flexor power generation in early stance. The role of hip flexor power generation becomes increasingly important at faster walking speeds and running. Although this cohort of people with TBI ran at a lower self-selected speed than the HCs, it seems that the relative excessive use of hip flexor power generation becomes less so when running. Excessive hip flexor power generation in late stance by people with TBI at self-selected walking speeds is less prevalent at fast walking speeds, and rarely occurred in this sample when running. In contrast, ankle power generation at push-off is reduced at self-selected and fast walking speeds, and remains significantly reduced when running, indicating that interventions aimed at improving ankle power generation for push-off seem warranted when training walking and running.
The long-term implications of running with an abnormal pattern remain unclear. The ability to run, if only for a few steps, may be important for participation in many social, leisure and employment roles. However, it is reasonable to assume that compensatory movement patterns used to attain running may be associated with detrimental forces just as they can be when walking. On the other hand, running strides typically only account for a minority of a person’s mobility performance from day to day, so the cumulative effect of potential detrimental forces may be insubstantial. Further investigation is required to establish when it is safe for a person with TBI to run, not just from a ‘risk of falling’ perspective, but also in relation to the larger and possibly abnormal joint moments when running on long-term musculoskeletal integrity.

Although this is a relatively small sample of people with TBI, to the best of our knowledge it represents the first report of the spatio-temporal, kinematic and kinetic abnormalities associated with running in any neurological population. Participants were able to run despite high-level mobility limitations and a wide variety of spatio-temporal, kinematic and kinetic abnormalities. Further, many of the abnormalities identified when walking actually shifted to become within normal limits when running, indicating that many variables associated with gait disorders may not prevent the ability to run. For example, over one-third of participants had reduced ankle power generation when walking, yet were still able to generate sufficient power for push-off into a flight phase for running. This finding indicates that the presence of some walking abnormalities may be of little use in clinical judgments about when someone is ready to commence higher level activities such as running. Further investigation is required to determine if there are any biomechanical variables associated with walking which predict or preclude the ability to run.

CONCLUSION

Many people with TBI can run despite significant mobility limitations and biomechanical abnormalities associated with their walking patterns. The biomechanical abnormalities
associated with running are diverse, yet are more prevalent in the sagittal plane at the knee. Few trunk, pelvic or hip abnormalities were detected, indicating that a stable torso may be an important feature of running. Ankle power generation at push-off was significantly reduced, and although partially compensated for by increased proximal power generation, is the likely cause of slow self-selected running speed following TBI.

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References


This graph demonstrates knee joint movement in the sagittal (flexion/extension) plane during one complete gait cycle. The graph begins at initial foot contact. The vertical dashed line represents toe off for the TBI cohort and the adjacent vertical solid line represents toe-off for the HCs. The portion of the graph from initial foot contact to the toe off line represents knee joint movement during stance phase while swing phase movement is to the right of the toe off line. Knee joint position is represented along the y-axis in degrees. The shaded portion of the graph represents the 95% confidence intervals for movement in HCs. The solid black line and the lightly-dashed lines represent the mean and ±2SD for the TBI sample respectively.
These graphs demonstrate ankle and hip joint power during one complete gait cycle for walking and running. Each graph begins at initial foot contact. The vertical dashed line represents toe off for the TBI cohort and the adjacent vertical solid line represents toe-off for the HCs. Joint power is represented along the y-axis in Watts/kg. The shaded portion of the graph represents the mean ±2SD for the HC sample. The solid black line and the surrounding dashed lines represent the mean and ±2SD for the TBI sample.