Acute Changes in Postural Control after Soccer Heading

Abstract
This study intended to determine if an acute bout of soccer heading alters postural control and pronounced self-reported symptoms of cerebral concussion. Collegiate soccer players were randomly assigned to one of 2 groups. Each participant completed a baseline postural control assessment prior to heading. Participants either simulated (control group; CG) or performed (experimental group; EG) 10 headers at 11.2 m/s in 10 min. The postural assessment was repeated post heading at hrs 1, 24, and 48. The postural control parameter assessed was the root mean square (RMS) of the center of mass (COM). COM RMS were calculated for the anterior-posterior (AP) and medial-lateral (ML) time series. Compared to the CG, for the AP and ML time series COM RMS values were significantly higher in the EG at hr 24 (p <0.05). An acute bout of heading results in quantifiable alterations in postural control that are detectable 24 h post heading and dissipate within an additional 24 h. The significant findings may be due to the dynamic postural control assessment that incorporated robust discordant environmental conditions.

Introduction
Soccer is unique in sport as the unprotected forehead is actively impacted to stop and redirect the ball. Known as a “header”, the impact on the head is believed to expose the brain to subconcussive trauma due to rapid deceleration of the skull post impact [7]. Whether heading results in neurological dysfunction similar to a concussion has yet to be determined. Research has shown a significant increase of biochemical markers of neurological damage following a soccer match which correlates with the number of headers taken [25, 26]. Other research groups have reported chronic cognitive deficits in experienced players [6,15,−20,31−33] while others have reported the opposite [1, 16, 18, 27, 28]. The discrepancy may be attributed to methodological differences: lack of baseline data, failure to control confounding variables (e.g., alcohol abuse), selection bias, over reliance on self-report, and type I error inflation. Research into short-term effects of a single bout of heading has identified no acute deficits in cognition [22] or balance [4,17,20]; however, there are a number of reasons why they may have been missed. Some studies did not look for such deficits, while others failed to control for ball velocity, verify sensitivity of the postural assessment, or test for statistical validity threats.

Methods
Subjects
16 volunteers, randomly assigned to an experimental group (EG, n=8, 21±2 years, 173±9 cm, 70±10 kg, 6 males, 2 females) or control group
(CG, n = 8, 23 ± 3 years, 180 ± 9 cm, 76 ± 13 kg, 6 males, 2 females), were included if they were a club or collegiate soccer team member with at least 5 years of heading experience and excluded if there was any history of head, neck, or face injury in the previous 6 months. Subjects were told to refrain from substances that could affect their nervous systems (e.g., stimulants and/or depressants) during the period of testing. All subjects signed university IRB approved informed consent and HIPAA forms [12].

Soccer heading model
An established standardized head impact model was used, consisting of a machine (JUGS Sports International, Tualatin, OR) capable of projecting a soccer ball as if kicked [13]. Balls were projected at 40° from horizontal at 11.2 m/s (25 mph). Subjects stood 10 m from the machine and were instructed to head the ball back in the air towards the machine.

Postural assessment
3D kinematic data were captured via a 6-camera infrared motion analysis system (Motion Analysis Corp., Santa Rosa, CA) at 120 Hz and low-pass filtered at 4 Hz using a 4th order Butterworth filter. Reflective markers were placed on the body to derive whole body center of mass (COM) motion (see Fig. 1), by applying a theoretical model [11] in custom written code (Mathworks, Natick, MA).

During quiet stance, subjects stood on a dynamic 3 degree-of-freedom posture platform (Neurocom International Inc., Clackamas OR) and were exposed to different conditions. The platform was situated within a 3-wall VE back-projection system [24]. The assessment consisted of 6 environmental conditions: (1) stationary VE with a stationary support surface, (2) in the dark with a moving support surface, (3) rotating VE with a stationary support surface, (4) rotating VE with a moving support surface, (5) in the dark with a stationary support surface, and (6) stationary VE with a moving support surface. In conditions 4 and 6, COM motion was used to simultaneously drive platform translation in the anteroposterior (AP) and mediolateral (ML) directions, and tilt about the ML axis. Gain was adjusted to the subject’s comfort level. A gain of 1.0 would move the platform to a maximum of 100% of COM movement in the same direction as COM. All conditions were repeated 3 times for a total of eighteen 30 s trials. The order of conditions was randomized and counter-balanced across subjects. The testing environment was controlled for noise and distraction.

Experimental protocol
4 data sessions were collected: hr 0, hr 1, hr 24, and hr 48. At hr 0, prior to heading, platform gain was set, and a baseline assessment was performed. No practice trials were allowed. After hr 0 session, subjects performed 10 headers (EG = actual headers, CG = a heading maneuver with no ball contact) within a 10 min period. Postural assessments of all subjects were then repeated at hr 1, hr 24, and hr 48 starting roughly 60 min after the 10th header.

Data analysis
Root mean square (RMS) was calculated for AP and ML components of each COM time series. RMS represents the average spread of a time series distribution relative to its mean. Higher RMS values are traditionally interpreted as greater postural instability or error [11]. 2 separate mixed-model 2 (group) × 4 (time) × 6 (condition) ANOVAs for AP and ML RMS with repeated measures of time and condition were performed (SPSS, Chicago, IL). Planned contrasts were used to test the a priori hypothesis. Significant interaction effects were explored using post-hoc comparisons (one-way ANOVA’s and t-tests using a Bonferroni correction to adjust level of significance). Significance was set at α < 0.05.

Results

ML COM
ANOVA revealed a group by session interaction (F\textsubscript{3,42} = 2.907, p = 0.046). Post-hoc comparisons revealed an increase in the EG at hr 24 compared to baseline (hr 0, p = 0.018, see Fig. 2). A condition by time interaction was found (F\textsubscript{15,210} = 2.73, p = 0.001), which post-hoc tests revealed was due to condition 4 only (p = 0.031). Hrs 1, 24, and 48 were not significantly different than baseline; however, hrs 1 and 24 were higher than hr 48 (p = 0.001, p = 0.005, respectively).

![Fig. 1 Marker placement and experimental set-up.](image)

![Fig. 2 ML COM group means collapsed across condition for each time period. Control (N = 8) and experimental (N = 8). CG = control group and EG = experimental group. *Significant differences from baseline for EG only (p < 0.05).](image)
A significant main effect of condition was found ($F_{2,84,39.76} = 28.14$, $p < 0.000$, $\epsilon = 0.568$). Post-hoc tests indicated conditions 2 (p = 0.001), 4 (p < 0.000), and 6 (p = 0.003) were higher than baseline (i.e., condition 1, Fig. 3).

Planned comparisons between the most challenging condition (i.e., 4) and baseline revealed a group by session interaction, ($F_{3,42} = 3.78$, p = 0.017). The RMS increase from baseline to hr 24 in the EG was significantly different from the CG (p = 0.023, Fig. 4).

**AP COM**

The main effect of time ($F_{3,42}, 7.07, p = 0.001$) was due to a significant RMS decrease at hr 48 relative to the baseline (p = 0.001, Fig. 5).

A main effect of condition ($F_{3,42}, 13.25, p = 0.000, \epsilon = 0.339$) was due to an increase in condition 4 relative to baseline (p = 0.001, Fig. 6).

Planned comparisons between condition 4 and baseline, revealed a group by session interaction ($F_{3,42} = 4.695$, p = 0.006). RMS increased from baseline (hr 0) to hrs 1 and 24 significantly more in the EG than in the CG (p = 0.033 and p = 0.040, respectively, Fig. 7).

**Discussion**

Significant findings in this study reveal that a bout of heading results in acute changes in postural control. The repetitive, sub-concussive forces on the brain when heading, even at the low velocities used in this experiment, appear to cause a large enough trauma to reduce postural stability. The postural deficits found in this study appear within 1 h of heading and dissipate within 2 days (48 h) with postural stability improving from baseline due to a possible practice effect that was blunted from the subconcussive head trauma. Contrary to the heading literature, the observed deficits follow a similar, yet accelerated, timeline relative to previously reported deficits associated with concussion which suggests the possibility of a subconcussive injury [9,10].

Post-concussive postural instability typically appears within 24 h of injury and dissipates within 3–5 days [10]. It has been suggested that such deficits are a result of a sensory integration problem which prevents successful processing of visual, vestibular, and somatosensory systems. Such integration is essential for balance under conditions of altered somatosensory and/or visual input. Healthy individuals can integrate sensory information and select the most reliable cues for precise motor control, whereas concussed individuals may have difficulty maintaining equilibrium in challenging or discordant sensory environments when this process is impaired.

Despite evidence which suggests heading has no effect on postural stability [4,17,23], it should be noted the conditions tested in prior studies may have failed to challenge the postural control system enough to elicit instability. Simply removing visual input has not been reported to cause instability in concussed populations [9] and the Clinical Test of Sensory Interaction and Balance is only validated for concussed populations who experience syncope [14]. A closely related test, the Sensory Organization Test (SOT), has been reported to be an effective diagnostic test for concussed populations by some [10] and ineffective by others [5]. Moreover, the SOT was not able to elicit postural instability in non-concussed football players after a high magnitude impact (e.g., >90 g) [21]. Thus, it is unlikely to be a sensitive test of postural instability following low force impacts (e.g., 20 g) that occur as a result of heading.

The current findings may be due to the dynamic environmental conditions used, which were challenging enough to differentiate the healthy from the impaired postural system. It is already known that such dynamic discordant sensory input (e.g., an unstable support surface with a VE) can challenge even a healthy postural control system [29]. Our results indicate that discordant sensory input may be diagnostically sensitive following trauma induced by a bout of heading.

There is speculation about the mechanisms for dysfunction following trauma [4]. The rapid deceleration of the skull and brain during ball impact could replicate the type of physical trauma that affects underlying neural mechanisms in a concussion. However, given the low ball velocities used in this study it is unlikely the impact could cause large enough shearing forces to structurally damage neurons [3].

An alternative explanation for the postural deficits found in the current study could be result of a biochemical cascade [18] induced by the 10 subconcussive impacts which occurred over a relatively short period of time. According to a recently developed post-concussive biochemical cascade model, the timeline for post-concussive alterations in glucose metabolism shows that...
within 30 min of injury increased localized cerebral glucose metabolism occurs, followed by hypometabolism after 6 h, which can then last up to 5 days [2]. This glucose imbalance has been proposed previously as an explanation for post-concussive postural deficits [10].

Postural deficits found in this study are not as robust as postural deficits following a concussion, yet there is a similar, albeit truncated, recovery timeline with deficits appearing after the onset of glucose hypometabolism (i.e., hr 1), dissipating before hr 48. This faster recovery period supports previous hypotheses that propose milder injuries (e.g., subconcussive trauma) may recover more rapidly from glucose hypometabolism than a concussion. Previous researchers may not have seen any postural deficits because post heading assessments were performed immediately after heading, thus, prior to glucose hypometabolism [17, 18].

Some limitations of our study are that we examined only the acute effects of heading and with a limited number of trials. Ball speed was not scaled according to gender which may play a factor due to different neck strengths; however, the effects may be minimal. Researchers have reported 10% higher head accelerations (20.2 vs. 18.2 g or ~2 g difference) in females vs. male college-aged soccer players using ball speeds similar to ours (22 mph). This difference was attributed to females exhibiting significantly less head mass and neck strength vs. their male counterparts [30]. Gender was controlled for in subject selection (2 females each in each group). The number of headers performed may have contributed to our results and future studies should perform 5 or 6 headers which is a more representative of game conditions. The protocol only addressed one aspect of neurological functioning. To be comprehensive future studies should also assess symptomatic, cognitive, and other motor domains using advanced methodologies such as biochemical markers and imaging techniques. Additionally, future studies should investigate the role of practice effects by training all subjects beforehand until a stable baseline is reached then randomly assigning subjects to the EG or CG.

This study did not address whether long-term soccer participation has a cumulative effect on neurological functioning. The effect of years of repetitive heading may lead to a cumulative degrading effect in some players that manifest similar to a concussion, a condition that would be not unlike dementia pugilistica. A header is difficult to perform, however if proper technique is taught early, the loading of the head may be controlled to prevent injury. Future longitudinal research examining cumulative effects is warranted to assure continued safe participation in soccer.

Conclusions

Soccer participation is on the rise, especially in the US. With its high number of head injuries, evaluating the safety of the sport is paramount. This study is the first to report statistically significant alterations in postural control post heading. Even more symptoms may have been detected if the ball velocity (11.2 m/s) was more representative of actual collegiate playing conditions (~30 m/s). Though our sample was small, EG and CG were similar in the most relevant characteristics, including familiarity with the postural stimuli, thus, it is unlikely the results are due to sampling error. The use of discordant sensory stimulation in an
enriched large field-of-view VE may have increased our ability to
detect postural differences between groups.

Acknowledgements

Supported by NIH-NIA grant AG26470 and NIH-NIDCD grant
DC05235. We gratefully acknowledge VRCO, Inc., for the use of
the CAVELIB to develop the VE.

References

1 Abreu F, Templer D, Schuyler B, Hutchinson H. Neuropsychological assess-
2 Barkoudarian G, Hovda H, Giza C. The molecular pathophysiology of
3 Blumbergs P, Jones N, North J. Diffuse axonal injury in head trauma.
J Neurol Neurosurg Psychiatry 1989; 52: 838–841
4 Broglio S, Guskiewicz K, Sell T, Lebhart S. No acute changes in postural
Detecting altered postural control after cerebral concussion in athletes
6 Downs D, Abwender D. Neuropsychological impairment in soccer ath-
7 Erlanger D, Kutner K, Barth J, Barnes R. Neuropsychology of sports-
related head injury: Dementia Pugilistica to Post Concussion Syn-
8 Guskiewicz K, Marshall S, Broglio S, Cantu R, Kirkendal D. No evidence of
impaired neurocognitive performance in collegiate soccer players.
9 Guskiewicz K, Perrin D, Gansneder B. Effect of mild head injury on
10 Guskiewicz K, Scott S, Marshall S. Postural stability and neuropsy-
chological deficits after concussion in collegiate athletes. J Athl Train
2001; 36: 263–273
11 Haran F, Keshner E. Sensory reweighting as a method of balance train-
12 Harris DJ, Atkinson G. Update – Ethical standards in sport and exer-
13 Higgins M, Tierney RT, Caswell S, Driban J, Mansell J, Clegg S. Reliability
2009; 223: 117–123
14 Ingersoll C, Armstrong C. The effects of closed-head injury on postural
15 Jordan S, Green G, Galanty L. Acute and chronic brain injury in United
States National Team soccer players. Am J Sports Med 1996; 24:
205–210
16 Kaminski T, Coissno E, Glutting J. Examining the relationship between
purposeful heading in soccer and computerized neuropsychological
17 Matser J, Kessels A, Jordan B, Lezak M, Troost J. Neuropsychological
impairment in amateur soccer players. JAMA 1999; 282: 971–973
18 Matser J, Kessels A, Jordan B, Lezak M, Troost J. Chronic traumatic brain
19 Matser J, Kessels A, Lezak M, Troost J. A dose-response relation of head-
ers and concussions with cognitive impairment in professional soccer
20 Mansus B, Wallmann H, Ledford M. Analysis of postural stability in
collegiate soccer players before and after an acute bout of heading
21 McCaffrey M, Mihalik J, Crowell M, Shields E, Guskiewcz K. Measure-
ment of head impacts in collegiate football players: clinical measures
of concussion after high- and low-magnitude impacts. Neurosurg
2007; 61: 1236–1243
22 Putukian M, Echemendia R, Mackin S. The acute neuropsychological
10: 104–109
23 Schmitt D, Hertel J, Evans T, Olsted L, Putukian M. Effect of an acute
bout of soccer heading on postural control and self-reported concus-
24 Slaboda J, Barton J, Maitin I, Keshner E. Visual field dependence influ-
Soc 2009; 1147–1150
25 Stalnake B, Tegner Y, Sojka P. Playing soccer increases serum concen-
thrations of the biochemical markers of brain damage S-100B and
18: 899–909
26 Stalnake B, Ohlsson A, Tegner Y, Sojka P. Serum concentrations of
two biochemical markers of brain tissue damage S-100B and neuron
specific enolase are increased in elite female soccer players after a
27 Stephens R, Rutherford A, Potter D, Fernie G. Neuropsychological
impairment as a consequence of football (soccer) play and football
heading: a preliminary analysis and report on school students (13–16
28 Straume-Naesheim T, Andersen T, Dverak J, Bahr R. Reproducibility of
computer based neuropsychological testing among Norwegian elite
29 Streepy J, Kenyon R, Keshner E. Visual motion combined with base
of support width reveals variable field dependency in healthy young
Sex differences in head acceleration during heading while wearing
31 Tyrvaer A, Lachen E. Soccer injuries to the brain. A neuropsychologi-
56–60
32 Webbe F, Ochs S. Recency and frequency of soccer heading interact
to decrease neurocognitive performance. Appl Neuropsychol 2003;
10: 31–41
33 Wittol A, Webbe F. Soccer heading frequency predicts neuropsychologi-