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Symptoms upon Postural Change and Orthostatic Hypotension in Adolescents with Concussion

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Contributions' Statement

Drs Haider, Patel, Willer, Wilber, Master, Arbogast, Mayer, Johnson, Mannix, and Leddy conceptualized and designed the study, drafted the initial manuscript, and reviewed and revised the manuscript. Ms Videira and Storey, Mr Mariotti, Wertz, Oglesbee and Bezherano, and Drs Park, Aguirre, and Fodero designed the data collection protocols, collected data and performed data quality analysis, and reviewed and revised the manuscript. Dr Meicznikowski designed the study, performed the statistical analysis, and reviewed and revised the final manuscript. All authors approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

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Abstract

Objective: Concussion is associated with cardiac dysautonomia, altered blood pressure (BP) control, and may cause Orthostatic Hypotension (OH). We measured the prevalence of OH using the 1-minute supine-to-standing OH Test in adolescents with concussion and controls.

Participants: Adolescents within 10 days of injury (Concussion Group, n=297, 15.0±1.7 years, 59% male, 79% sport-related) were compared with matched controls (Control Group, n=214, 15.0±1.5 years, 58% male).

Methods: BP, heart rate (HR) and complaints of lightheadedness or dizziness were measured after 2-minute supine rest and 1-minute standing. Control Group was assessed once. Concussion Group was assessed twice; (1) initial visit while symptomatic (mean 6.0±3 days-since-injury) and (2) after clinical recovery (mean 46.3±42 days-since-injury).

Results: At initial visit, Concussion Group reported feeling lightheaded/dizzy on postural change more often than the Control Group (37% vs 4%, $p<0.001$) but did not differ in meeting standard OH criteria (3% vs 5%, $p=0.32$). Experiencing symptoms did not correlate with meeting OH criteria, but strongly correlated with an abnormal vestibulo-ocular reflex. After clinical recovery, Concussion Group did not differ in experiencing lightheaded/dizziness on postural change than controls (4%, $p=0.65$).

Conclusion: Adolescents commonly experience orthostatic intolerance after concussion but do not meet the standard criteria for OH.

INTRODUCTION

Concussion, a subset of mild traumatic brain injury (mTBI), is a physiological(1) metabolic, (2) and microstructural(3) insult to the brain resulting in, among other findings, altered cardiovascular autonomic nervous system (ANS) function.(4–7) Damage to the primary ANS control center, located in the brainstem, has been confirmed in a recent diffusion tensor imaging study following concussion.[8] Physiological research has shown that subjects with concussion demonstrate reduced baroreflex sensitivity moving from supine to standing(9) and reduced heart rate variability (HRV, a measure of sympathovagal balance) at rest and during exercise,(10) which is consistent with functional uncoupling of the cardiovascular ANS after concussion. Symptoms reported after head injury are not specific to concussion, (11) which makes it challenging to diagnose concussion using symptoms alone.(12) Physical examination maneuvers that stress the cardiovascular ANS may therefore improve a clinician's ability to more objectively diagnose concussion beyond that of resting symptom reports, and help identify symptom generators amenable to treatment.(13)

Moving from a supine to standing position causes a reduction in systemic blood pressure (BP) due to caudal redistribution of blood(14) and BP reaches the nadir at 10 to 20 seconds after standing.(14) This drop is sensed immediately by the aortic and carotid baroreceptors, resulting in parasympathetic downregulation and sympathetic activation (referred to as the baroreflex-mediated autonomic response)(15) to increase cardiac contractility, heart rate (HR), and vascular tone.(16) According to the American Autonomic Society, Orthostatic Hypotension (OH) is defined as a 20 mm Hg or greater reduction in systolic BP or a 10 mm Hg or greater reduction in diastolic BP after 1 and 3 minutes of standing from a supine position.(17) This drop in BP causes a disruption in the brain's perfusion pressure precipitating a sensation of lightheadedness or dizziness.(18) The prevalence of OH varies in the non-concussed population. Prevalence in the elderly ranges from 5–30%;(18) however, rates in adolescents are unknown because screening for OH is not routine.(19)

Several clinical reviews and expert opinion papers suggest that concussion causes OH;(20–22) however, there are scarce data on the incidence of OH after concussion. To assess the prevalence of OH after concussion, we measured BP, HR and precipitation of a complaint of lightheaded/dizzy on a standard 1-minute supine-to-standing OH Test in adolescents within 10 days of injury and again after clinical recovery, and compared them to age- and sex-matched non-concussed controls. Adolescents were studied because they are at high risk for concussion and appear to take the longest to recover.(23, 24) We hypothesized that acutely injured adolescents with concussion would more often meet standard BP criteria for OH when compared with healthy controls and that normal BP control would return upon clinical recovery.

METHODS

Study Design

This multi-center prospective cohort study was approved by the University at Buffalo, University of New Mexico and University of Pennsylvania Institutional Review Boards. Adolescents within 10 days of injury were seen at university-affiliated concussion clinics at Buffalo, Albuquerque and Philadelphia. Written informed consent was obtained at initial clinic visit. Concussions were diagnosed according to international Concussion In Sport Group (CISG)(12) guidelines by experienced clinicians based on history (including a standardized concussion symptom checklist(25)) and clinical assessment. Diagnosis of concussion was standardized between all study physicians prior to enrollment. For healthy controls at Buffalo and Philadelphia, sports-medicine physicians performed routine high school pre-season sport physicals that included history, vital signs, and a physical exam. Albuquerque invited healthy adolescents to their center and measured their vital signs and performed the physical exam using standardized procedures.

Adolescents diagnosed with a concussion were followed up until they had recovered or for up to 4 months. Recovery was defined as return of symptoms to baseline and a normal physician-performed physical examination, plus normalization of adjunct tests if performed (e.g. exercise tolerance testing). It is important to note that the date of clearance to begin a return to sport strategy did not always coincide with date of recovery as participants could

have recovered several days or weeks before their final clinic visit, or even at the beginning of their next season. Hence, no analysis comparing recovery times were performed.

Participants

Adolescents with concussion (Concussion Group) consisted of male and female adolescents (aged 12 to 18 years) who came to the university concussion management programs within 10 days of injury. Participants were excluded for: (1) evidence of focal neurological signs on physical examination or mass lesion on imaging (if available); (2) history of moderate or severe TBI (i.e., Glasgow Coma Scale ≤12); (3) history of cervical spine injury, autoimmune disease, or known heart disease that would affect the ANS or resting HR; and (4) currently on medications that would affect HR such as medications for ADHD, depression, or anxiety. Those who were still symptomatic at 4 months were retrospectively classified as Prolonged Recovery Group and analyzed as a separate cohort. Healthy adolescents (Control Group) consisted of male and female participants (aged 12 −18 years) with no history of concussion in the past year. Exclusion criteria were identical to those of the Concussion Group. Not all participants had sustained a sport-related concussion (SRC) but since most of the injured participants were athletes, we enrolled a control group that was athletic and matched for height and weight since BP is closely related to height and BMI.(26, 27)

Outcome Measures

Physical Assessment—Clinicians performed a standardized concussion-oriented physical assessment as part of a larger study, but only the vestibular components (tandem gait and horizontal vestibulo-ocular reflex [VOR]) are presented here. Directions for the physical exam were standardized for all study centers.(28, 29) For tandem gait, inability to walk a straight line, extreme truncal sway, stumbling, or stepping out of line with eyes open was considered abnormal, but one misstep or minimal sway during the eyes closed portions was considered within normal limits. For horizontal VOR, any abnormal saccadic eye movements, very slow performance, inability to maintain visual fixation (i.e., beating back to the center), or symptom provocation of dizziness or headache was considered abnormal.[24]

OH assessment—A standard 1-minute supine-to-standing OH Test was used, prior research does not recommend use of the 3-minute OH Test in clinic practice.(30) All study physicians and assistants received the same instructions on how to perform vital signs.(28) An automated BP cuff (Welch Allyn Connex ProBP 3400 Digital Blood Pressure Device in Buffalo, Panasonic EW-3109 in Albuquerque, and Carescape Dinamap V100 and Omron 5 Blood Pressure Monitor in Philadelphia) was used to measure HR and BP. The first measurement was taken after the participant was resting supine on the examination table for at least 2 minutes. Keeping the BP cuff in the same position, the participant was then asked to stand up without support and with both feet firmly on the ground and a second measurement was taken after standing for 1 minute. The participant was asked to report lightheadedness or dizziness at supine, immediately upon standing, and after one minute. Lightheadedness or dizziness on postural change was considered positive only if there was new onset or worsening of lightheadedness/dizziness upon standing when compared to supine. Pulse pressure (PP, a clinical proxy for stroke volume)(31) was calculated by subtracting diastolic BP from systolic BP. Change in systolic and diastolic BP was

calculated by subtracting supine values from standing values and change in HR was calculated by subtracting standing values from supine values.

Statistical Analysis

Normality of continuous variables (age, BMI, HR, and BP) was assessed and a series of Mann Whitney U Tests were used to compare differences between groups. χ^2 test was used to compare differences in categorical/binary variables between groups (sex, history of OH/fainting spells, vestibular examination findings, symptoms on postural change, and meeting OH criteria). Concussion Group participants' first visit was compared to Control Group participants' only visit to see if acute concussions were associated with OH. Concussion Group participants' first visit was compared to Prolonged Recovery Group participants' first visit to see if there were any variables associated with prolonged recovery. Concussion Group participants' last visit was compared to Control Group participants' only visit to see if these values returned to baseline after clinical recovery. Finally, we analyzed Concussion Group according to sex. To adjust for multiple comparisons, post-hoc Bonferroni correction was used and a *p*-value of ≤ 0.01 (0.05/5) was considered significant.

A series of ANCOVAs was performed in adolescents with concussion (Concussion Group and Prolonged Recovery Group, n = 323) with location as a covariate (to control for differences in population and automated BP devices) to assess the relationship between experiencing symptoms on postural change and the following: (1) meeting OH criteria, (2) abnormal VOR, and (3) abnormal tandem gait. Prior publications on OH after concussion or mTBI do not provide prevalence rates of meeting standard OH criteria after injury and the prevalence of OH in healthy athletic adolescents is uncertain because screening for OH is not routine.(19) Hence, we aimed to recruit at least 176 participants in each group (total n = 352) because this sample size would have enough power to detect a small effect size (d = 0.3, $\alpha = 0.05$, $\beta = 0.80$)(32) between binary outcomes in two populations. All data analyses were performed using the SAS Version 9.4.

RESULTS

Four hundred nine participants were seen within 10 days of injury and were diagnosed with a concussion. Twenty-nine participants were on medications and/or had a condition that would affect HR, 40 participants did not have complete HR and/or BP values at first visit, and 18 participants were lost to follow-up. Hence, 323 adolescents with concussion were included in the analysis. Twenty-six participants did not recover by 4 months and comprised the Prolonged Recovery Group. Hence, 297 adolescents comprised the Concussion Group. Adolescents with concussion not included in the analysis (n = 58) did not differ significantly in age or sex versus those included in the analysis. Two hundred and thirty-five healthy adolescents consented for the study but 21 participants were either on medications, had a concussion within the past year, or had a history of moderate to severe TBI and were removed. Hence, a total of 214 adolescents comprised the Control Group. The groups were matched on age, sex, and BMI but Concussion Group participants had a significantly higher number of previous concussions than Control participants. There was also a much higher

percentage of non-sport related injuries in Prolonged Recovery Group participants' than in the participants with concussion. Demographics for all groups are presented in Table 1.

Physical examination and 1-minute OH Test results are presented in Table 2. Control participants had a much lower incidence of having an abnormal VOR, tandem gait and dizziness on postural change than participants with concussion at the first visit. Control participants also had significantly higher mean supine and standing HR than participants with concussion. Participants with concussion had a smaller increase in HR than the control participants, but this was not significant after Bonferroni correction. No significant differences were seen in any physical exam or OH test components between the Concussion and Prolonged Recovery Groups after Bonferroni correction. After participants with concussion had recovered (last visit), there were multiple differences in standing BP and HR values when compared withi controls that were not present at their first visit.

At all first visits for participants with concussion (Concussion Group + Prolonged Recovery Group, n = 323), symptoms on postural change did not correlate with meeting OH criteria (r = 0.129, $p = 0.027$) or abnormal tandem gait (r = 0.083, $p = 0.153$) and both relationships were not affected by location ($p = 0.155$ and 0.911, respectively). However, feeling lightheaded or dizzy on postural change strongly correlated with abnormal VOR (r = 0.255, $p = 0.001$), which was not affected by location ($p = 0.812$).

Demographics and clinical assessment results for male and female adolescents in the Concussion Group are presented in Table 3. Females had significantly higher initial symptom severity scores and longer recovery times than males. Females also had a lower mean supine systolic BP than males but did not differ on any other clinical assessments. Of note, females had a higher incidence of meeting OH criteria than males, but this was not significant after Bonferroni correction.

DISCUSSION

This prospective multi-center study found that adolescents in the acute/sub-acute phase after concussion experienced symptoms of lightheadedness or dizziness when standing up from a supine position more often than healthy athletic controls. They did not, however, satisfy standard criteria for OH more often than controls and there was no correlation in adolescents with concussion of experiencing lightheadedness/dizziness on postural change with meeting standard OH criteria. The clinical utility of assessing the postural symptom response is that it may make the diagnosis of concussion more specific since resting symptoms do not always reliably distinguish concussion from other injuries (e.g., to the cervical spine).(11)

Dizziness can be divided into distinct categories of vertigo and lightheadedness.(33) Vertigo is defined as the hallucination of movement whereas lightheadedness is secondary to diminished cerebral perfusion. In our study, feeling dizziness upon standing was strongly correlated with an abnormal VOR, suggesting the cause of the specific complaint of dizziness was vestibular as opposed to altered cerebral perfusion. The symptom of dizziness in the supine position is considered to be more indicative of peripheral vestibular dysfunction(34) whereas dizziness experienced upon postural change may involve either

central or peripheral vestibular dysfunction; current literature is inconclusive regarding peripheral versus central causes of dizziness in concussion.(35) Recent diffusion tensor imaging studies support the idea of a central component to post-traumatic vestibulopathy; (36) however, there is likely a combination of peripheral and central causes.(37) Further research is needed to understand the different causes of dizziness in the concussed population.

The symptom of lightheadedness upon standing is thought to emanate from cardiovascular autonomic dysfunction as the brain experiences a sudden instability in cerebral perfusion pressure that may precipitate symptoms that typically are short-lived.(14) Acutely injured participants with concussion had lower HRs and a smaller change in HR moving from supine to standing when compared with controls. Changes in BP due to posture are detected by baroreceptors in the aortic arch and carotid bodies.(15) The physiologic response is to increase arterial vasoconstriction and venoconstriction, increase cardiac contractility and HR, and stimulate adrenal epinephrine release to maintain homeostasis and cerebral perfusion.(15) One study reported a greater prevalence of orthostatic intolerance on the Active Stand Test in adolescents with concussion within 3 months of injury.(38) Orthostatic intolerance is defined as experiencing symptoms upon postural change that are abated by recumbency, with or without a measurable reduction in BP,(19) and OH is one of several orthostatic intolerance syndromes. Although acutely injured participants with concussion in our study did not meet standard OH criteria, they were intolerant to postural change and demonstrated significantly lower resting and standing HRs than controls, suggesting some level of ANS dysfunction. One possibility is that smaller changes in BP may cause clinically significant changes in cerebral perfusion pressure to precipitate symptoms in the concussed brain, which is in a state of impaired autoregulation.(4–7, 39) Future studies should be designed to assess this possibility.

When comparing sexes within the Concussion Group, females had a higher initial symptom severity and took longer to recover than males. They did not, however, experience symptoms on postural change or meet standard OH criteria more often than males. Males had an approximately 4 mmHg greater mean supine systolic BP than females, the clinical significance of which is debatable. Systolic BP is closely related to height and weight,(27) and males are generally taller and weigh more than females so our findings could be explained by these baseline physiological differences. After clinical recovery, adolescents with concussion had several differences in HR and BP values when compared with control participants. A systematic review reported that several studies found that cardiovascular dysfunction persists after clinical recovery from concussion, and that there is no specific modality of cardiac function that coincides with clinical recovery.(40) In our sample, the majority of participants with concussion were athletic and received their head injury during sport. They were kept out of most physical activities for a mean of 46 days, which could have led to aerobic deconditioning with changes in baseline BP and HR values.(41) Deconditioning can affect autonomic function, but this would not have affected BP and HR values in the first visit for participants with concussion since it was a mean of 6 days after injury, which is not sufficient time to cause significant physical deconditioning in athletes. (42)

Some adolescents with concussion did not recover by 4 months, characterized by a significantly higher proportion of non-sport related injuries, including motor vehicle accidents and assaults. Non-SRC patients have been reported to take longer to recover due to concomitant injuries.(43) The Prolonged Recovery Group also had a higher rate of vestibular physical examination abnormalities, consistent with previous research that vestibular dysfunction is associated with prolonged recovery.(44, 45) We did not find any difference in meeting standard OH criteria or experiencing lightheadedness/dizziness symptoms on postural change in those with prolonged recovery versus those who recovered within 4 months.

The major limitation of this study is that healthy controls were assessed at one time-point. Due to limited funding, healthy controls were evaluated during routine pre-season sports physicals that included the 1-minute OH test. OH measurements in the clinical setting may not be reliable(46) and we did not asses re-test reliability for our OH assessment. Future studies should assess controls at intervals consistent with symptomatic and recovered time points for participants with concussion. We also used different brands of automated BP monitors in each center; however, the method for BP measurement was standardized between sites,(29) the same monitor was used for each patient's visit, and our correlations were not affected by location, so we do not suspect these factors affected our results. Medically approved automated BP devices are regularly monitored according to manufacturer protocols and are known to be reliable medical devices. Fitness level affects vital sign measurements because of training-induced improvement of autonomic regulation.(47) We accounted for this by recruiting controls from the same schools as the athletes with concussion and matched the groups on athletic status and BMI. Lastly, baseline HR and BP values are affected by factors such as mood, food intake, time of day, and environment, which we did not control for.(27, 48, 49) These factors would not be controlled for in a clinical setting, however, which enhances the external validity of this study. The Concussion Group was seen in a clinical setting, which could cause some anxiety when compared with the scheduled pre-season physical examinations of the Control Group. Future studies should be designed to prospectively compare individuals with and without concussion at similar time points while attempting to control as much as possible for modifiable factors.

CONCLUSION

Male and female acutely injured adolescents with concussion experienced symptoms of lightheadedness and dizziness upon postural change significantly more often than healthy adolescents; however, they did not more often meet standard OH criteria. The symptom of dizziness (or vertigo) on postural change could have been from vestibular dysfunction since it was associated with an abnormal vestibular ocular reflex. The symptom of lightheadedness could have been from cardiovascular autonomic dysfunction since acutely injured adolescents with concussion had lower HRs and a smaller increase in HR moving from supine to standing when compared with controls. Upon clinical recovery, adolescents with concussion had more significant differences in HR and BP values versus healthy controls, consistent with cardiovascular deconditioning. The clinical utility of assessing the postural symptom response is that it may make the diagnosis of concussion more specific since resting symptoms do not always reliably distinguish concussion from other injuries (e.g., to

the cervical spine). Further research into the causes of a variety of symptoms and their utility in concussion assessment is warranted.

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Table 1.

Groupwise participant demographics

	Control Group (n = 214)	Concussion Group (n = 297)	*p*-value[1]	Prolonged Recovery Group (n = 26)	*p*-value[2]
Age in years	14.96 ± 1.5	15.04 ± 1.7	0.555	15.26 ± 1.7	0.628
Sex	58.4% male (n = 125)	58.6% male (n = 174)	0.968	46.2% male (n = 12)	0.266
Body Mass Index in kgm^{-2}	22.2 ± 4.7	22.4 ± 4.5	0.764	23.1 ± 4.3	0.722
Previous Concussions	0.10 ± 0.3	0.62 ± 1.0	< **0.001**	0.58 ± 0.8	0.842
History of Orthostatic Hypotension	0.5% (n = 1)	1.4% (n = 4)	0.319	0% (n = 0)	0.466
Days since injury to first visit	-	5.98 ± 3.0	-	5.64 ± 3.0	0.021
Days since injury to last visit		46.27 ± 41.9	-	> 120	-
Sport-related injury	-	79.4% (n = 235)	-	46.2% (n = 12)	< **0.001**
Initial Visit symptom severity *	-	31.8 ± 19.6	-	29.8 ± 28.7	0.738

*: symptom severity on Post-Concussion Symptom Scale (max = 132)

1 = *p*-value of comparison between Control Group and Concussion Group

2 = *p*-value of comparison between Concussion Group and Prolonged Recovery Group.

Table 2.

Physical examination, blood pressure and heart rate values, symptoms on postural change and meeting standard OH criteria

	Control Group (n = 214)	Concussion Group First Visit (n = 297)	*p*-value[1]	Prolonged Recovery Group (n = 26)	*p*-value[2]	Concussion Group Last Visit (n = 297)	*p*-value[3]
Abnormal Tandem Gait	19.2% (n = 41)	38.7% (n = 115)	< **0.001**	53.8% (n = 14)	0.017	9.8% (n = 29)	**0.002**
Abnormal VOR	7.9% (n = 17)	33.3% (n = 99)	< **0.001**	53.8% (n = 14)	0.013	3.0% (n = 9)	0.011
Supine Systolic BP	118.2 ± 11.9	117.5 ± 12.1	0.550	118.2 ± 10.3	0.891	116.6 ± 10.8	0.100
Supine Diastolic BP	70.2 ± 9.1	69.0 ± 7.6	0.113	68.3 ± 9.3	0.786	68.7 ± 7.9	0.040
Supine HR	71.0 ± 12.9	66.9 ± 12.2	< **0.001**	64.7 ± 14.3	0.483	69.7 ± 12.7	0.270
Standing Systolic BP	119.4 ± 12.7	118.2 ± 13.6	0.306	119.2 ± 11.6	0.854	115.8 ± 12.4	**0.001**
Standing Diastolic BP	77.2 ± 9.3	75.9 ± 7.6	0.085	74.2 ± 9.4	0.792	73.0 ± 7.6	< **0.001**
Standing HR	87.0 ± 15.6	80.3 ± 14.2	< **0.001**	82.1 ± 17.6	0.521	82.8 ± 13.5	**0.001**
Mean change Systolic BP	−1.26 ± 10.3	−1.06 ± 9.3	0.815	−1.64 ± 12.7	0.809	0.80 ± 9.5	0.020
Mean change Diastolic BP	−6.99 ± 9.6	−6.87 ± 7.7	0.870	−9.84 ± 10.6	0.043	−4.34 ± 7.9	**0.001**
Mean change HR	16.1 ± 12.6	13.4 ± 13.8	0.026	16.8 ± 11.7	0.191	13.03 ± 13.6	**0.010**
Dizziness on Standing	4.2 % (n = 9)	36.7% (n = 109)	< **0.001**	34.6% (n = 9)	0.698	5.1% (n = 15)	0.650
Met Standard OH Criteria	5.1% (n = 11)	3.4% (n = 10)	0.323	7.7% (n = 2)	0.683	7.4% (n = 22)	0.300
Supine Pulse Pressure	48.0 ± 11.0	48.2 ± 12.1	0.856	53.6 ± 10.0	0.020	48.1 ± 10.2	0.914
Standing Pulse Pressure	42.3 ± 11.6	42.6 ± 12.0	0.759	45.4 ± 10.6	0.229	42.1 ± 14.8	0.921

VOR = Vestibulo-Ocular Reflex; BP = blood pressure in mmHg; HR = heart rate in bpm; OH: orthostatic hypotension

[1] = *p*-value of comparison between Control Group and Concussion Group's first visit

[2] = *p*-value of comparison between Prolonged Recovery Group's first visit and Concussion Group's first visit

[3] = *p*-value of comparison between Control Group and Concussion Group's last visit.

Table 3.

Demographics and physical assessment findings for Females and Males in Concussion Group at first visit

	Concussion Female (n = 123)	**Concussion Male (n = 174)**	***p*-value**
Age (years)	15.03 ± 1.6	15.06 ± 1.7	0.882
Body Mass Index (kgm^{-2})	22.35 ± 4.4	22.37 ± 4.6	0.980
Days since injury	6.46 ± 3.0	5.64 ± 3.0	0.021
Initial Visit symptom severity *	37.65 ± 19.9	28.40 ± 18.6	**0.001**
Days to recovery	54.6 ± 44.4	40.4 ± 39.0	**0.004**
Abnormal Tandem Gait	35.8% (n = 44)	40.8% (n = 71)	0.381
Abnormal Horizontal VOR	30.9% (n = 38)	35.1% (n = 61)	0.453
Supine Systolic BP	115.2 ± 11.4	119.2 ± 12.4	**0.005**
Supine Diastolic BP	69.8 ± 7.9	68.4 ± 7.3	0.124
Supine HR	69.0 ± 12.7	65.4 ± 11.7	0.012
Standing Systolic BP	116.5 ± 10.3	119.5 ± 15.4	0.064
Standing Diastolic BP	76.3 ± 8.3	75.6 ± 7.2	0.475
Standing HR	81.5 ± 15.0	79.4 ± 13.5	0.205
Symptoms on Postural Change	35.0% (n = 43)	38.0% (n = 66)	0.601
Met Standard OH Criteria	6.5% (n = 8)	1.2% (n = 2)	0.012

*: symptom severity on Post-Concussion Symptom Scale (max = 132); VOR = Vestibulo-Ocular Reflex; BP = blood pressure in mmHg; HR = heart rate in bpm