

Motion Sickness Susceptibility and Baseline Vestibular and Ocular-Motor Performance in Adolescent Athletes

R. J. Elbin, PhD*; Anthony P. Kontos, PhD†; Alicia Sufrinko, PhD†; Mallory McElroy, MS*; Katie Stephenson-Brown, MS*; Samantha Mohler, MS*; Nathan R. D'Amico, MS, LAT, ATC*; Michael W. Collins, PhD†

*Office for Sport Concussion Research, Department of Health, Human Performance and Recreation, University of Arkansas, Fayetteville; †UPMC Sports Medicine Concussion Program-Department of Orthopaedic Surgery, University of Pittsburgh, PA

Context: High school athletes with a history of motion sickness susceptibility exhibit higher baseline vestibular and ocular-motor scores than those without a history of motion sickness susceptibility.

Objective: To examine the effects of motion sickness susceptibility on baseline vestibular and ocular-motor functioning, neurocognitive performance, and symptom scores.

Design: Cross-sectional study.

Setting: Preseason concussion testing.

Patients or Other Participants: A convenience sample of high school athletes ($N = 308$, age = 15.13 ± 1.21 years) involved in a variety of sports.

Main Outcome Measure(s): Vestibular/Ocular Motor Screening, computerized neurocognitive assessment, symptom scale, and Motion Sickness Susceptibility Questionnaire-Short Form (MSSQ-S).

Results: Participants were categorized into 3 groups based on a median split of the scores (eg, NONE, LOW, and HIGH). The LOW ($n = 95$) and HIGH ($n = 92$) groups (ie, MSSQ-S score

> 0) were 2.64 times more likely ($\chi^2_{1,257} = 7.94$, $P = .01$, 95% confidence interval = 1.32, 5.26) to have baseline Vestibular/Ocular Motor Screening scores larger than the clinical cutoffs for the NONE group ($n = 70$). No between-groups main effects were present for the NONE ($n = 52$), LOW ($n = 89$), and HIGH ($n = 90$) MSSQ-S groups for verbal ($F_{2,230} = .09$, $P = .91$, $\eta^2 = .001$) and visual ($F_{2,230} = .15$, $P = .86$, $\eta^2 = .001$) memory, processing speed ($F_{2,230} = .78$, $P = .46$, $\eta^2 = .007$), or reaction time ($F_{2,230} = 2.21$, $P = .11$, $\eta^2 = .002$). The HIGH group exhibited higher total baseline symptom scores than the LOW ($U = 3325.50$, $z = -1.99$, $P = .05$, $r = .15$) and NONE ($U = 1647.50$, $z = -2.83$, $P = .005$, $r = .24$) groups.

Conclusions: Motion sickness should be considered a preexisting risk factor that might influence specific domains of the baseline concussion assessment and postinjury management.

Key Words: VOMS, baseline testing, concussion

Key Points

- Preexisting motion sickness susceptibility influenced baseline vestibular and ocular-motor performance by and symptom reporting of adolescent athletes.
- Athletes who were highly susceptible to motion sickness reported more baseline affective concussion symptoms (eg, sadness, nervousness, feeling more emotional).
- Assessing motion sickness susceptibility should be an important part of the concussion evaluation.

Vestibular and ocular-motor assessments are emerging as a valuable component of the standard of care for patients with sport-related concussion (SRC). The vestibular and ocular-motor systems are a complex sensory brain network that provides a subjective sense of self-motion and orientation that is vital to integrating sensory input and generating appropriate and accurate behaviors.¹ Vestibular and ocular-motor impairment occurs in 60% to 80% of patients with SRC, and researchers²⁻⁴ have linked these impairments to protracted SRC recovery. Moreover, data from vestibular and ocular-motor assessments can be used to inform targeted treatment and rehabilitation strategies for individuals with SRC (eg, vestibular and ocular-motor therapy).

The Vestibular/Ocular Motor Screening (VOMS) is a brief assessment that was developed to assess vestibular

and ocular-motor impairments and symptoms associated with SRC.⁴ To date, most investigators⁵⁻⁸ who used the VOMS applied posttest-only research designs in patient cohorts with SRC. However, other authors have used the VOMS as a baseline (ie, preinjury) measure to identify false-positives,⁹ document normative values,¹⁰ and examine prospective changes¹¹ in vestibular and ocular-motor performance. Although these studies advanced the knowledge base and underscored the importance of the vestibular and ocular-motor assessment after SRC, several clinical questions warrant continued study. Specifically, limited data are available on factors that influence vestibular and ocular-motor function in individuals with or without SRC. To date, sex and a history of migraines or headaches have been identified in the literature as influencing the vestibular

and ocular-motor function associated with SRC evaluation.^{9,10}

In recent studies, researchers have identified motion sickness as a risk factor for vestibular and ocular-motor dysfunction in individuals with or without SRC. Sufrinko et al⁶ compared vestibular impairment, in addition to neurocognitive and symptom scores, at 1 to 10 and 11 to 20 days postinjury between concussed adolescent athletes with and those without a history of motion sickness. At 1 to 10 days postconcussion, both groups of athletes reported similar levels of vestibular dysfunction and neurocognitive impairment. However, concussed athletes with a history of motion sickness endorsed more affective symptoms (eg, sadness, nervousness, feeling more emotional) than concussed athletes without a history of motion sickness. At 11 to 20 days postinjury, concussed athletes with a history of motion sickness were more likely to experience persistent vestibular impairment and greater total symptom severity than concussed athletes without a history of motion sickness. These findings were also supported by Kontos et al,⁹ who documented baseline VOMS scores below clinical cutoff values (ie, normal) in 89% of nonconcussed collegiate athletes; among the 11% who scored above clinical cutoff levels, 72% endorsed a history of motion sickness. These findings are likely attributable to a “sensory conflict” between the vestibular system and visual/somatosensory inputs (eg, riding in the back seat of a moving vehicle) that results in symptoms of motion sickness (eg, dizziness, nausea, vomiting, abdominal discomfort).¹² The relationship between motion sickness and vestibular dysfunction after SRC is in agreement with other studies of nonathletic patient populations.¹³

Despite these findings, a limited number of investigators have examined the influence of motion sickness on vestibular and ocular-motor functioning, in addition to neurocognitive and symptom scores, among adolescent athletes. The onset of motion sickness is typically in early adolescence; the condition persists into the teenage years and dissipates in adulthood.¹⁴ Because the vestibular system is not fully mature until middle to late adolescence,¹⁵ baseline motion sickness may be particularly relevant when making clinical management and treatment decisions for adolescents with SRC. Previous researchers⁹ obtained information on a history of motion sickness from the medical records, but these data are not always available for healthy athlete samples. Alternatively, motion sickness susceptibility can be assessed via the Motion Sickness Susceptibility Questionnaire-Short Form (MSSQ-S).¹⁶ The MSSQ-S has been used to assess motion sickness susceptibility in several age groups¹⁷ and in both vestibular¹⁸ and concussed patient populations.⁵ However, the MSSQ-S has not been used to examine motion sickness in healthy adolescent athletes. Moreover, no authors to date have addressed the effects of motion sickness susceptibility on vestibular and ocular-motor functioning in adolescent (eg, high school) athletes. Assessing motion sickness susceptibility in healthy adolescent athletes would extend the current knowledge base regarding the influence of motion sickness on SRC assessments, particularly vestibular-ocular measures, to a younger population. Moreover, this work would help explain abnormal baseline scores observed in adolescent athletes that should be considered when interpreting postinjury assessment data. The primary

purpose of our study was to examine the effects of motion sickness susceptibility on baseline vestibular and ocular-motor function in healthy adolescent athletes. A secondary purpose was to investigate the effects of motion sickness susceptibility on baseline neurocognitive performance and symptom scores in healthy adolescent athletes.

METHODS

Design and Participants

A cross-sectional research design was used for this study. High school athletes aged 13 to 18 years were recruited from an ongoing SRC research surveillance program in the central Midwest region of the United States. Athletes who completed a computerized neurocognitive assessment, symptom scale, vestibular-ocular motor screening, and a motion sickness susceptibility questionnaire during pre-season (ie, baseline) concussion testing were included in the study. Any athlete who endorsed English as a second language or had a learning disability, attention-deficit/hyperactivity disorder, or history of migraines or headaches was excluded from the study.

Instrumentation

Vestibular and Ocular-Motor Assessment. The VOMS has 9 components: (1) baseline symptoms, (2) smooth pursuits, (3) horizontal saccades, (4) vertical saccades, (5) horizontal vestibular-ocular reflex (VOR), (6) vertical VOR, (7) visual motion sensitivity (VMS), (8) near point of convergence (NPC) distance, and (9) convergence symptoms. Before the VOMS was administered, athletes rated their current headache, dizziness, nausea, and foggiess on a 10-point Likert scale (0 = none to 10 = severe). After completing each VOMS component, athletes rated these symptoms (headache, dizziness, nausea, and foggiess) again. The NPC distance was calculated as the average distance (cm) across 3 trials. The scoring sheet for the VOMS was published as online supplemental material in Mucha et al.⁴ Clinical cutoff scores for the VOMS were as follows: (1) a total symptom score of ≥ 2 for any VOMS item and (2) an average NPC distance of ≥ 5 cm. The VOMS has shown good internal consistency and sensitivity for identifying patients with concussion.^{2,4,8,9}

Neurocognitive and Symptom Assessment. The Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT) was used to measure neurocognitive impairment and symptoms. The ImPACT is a computer-based neurocognitive test battery composed of 6 tasks that yield 4 composite scores: verbal memory, visual memory, processing speed, and reaction time. Also included in ImPACT is the Post-Concussion Symptom Scale (PCSS), which is a 22-item, self-reported symptom inventory that results in a total symptom severity score. In addition, baseline symptom factors were calculated according to Kontos et al¹⁹: (1) cognitive sensory (sensitivity to light, sensitivity to noise, feeling slowed down, mentally foggy, difficulty concentrating, difficulty remembering, vision problems); (2) sleep arousal (fatigue, trouble falling asleep, sleeping less than usual, drowsiness); (3) vestibular somatic (headache, nausea, vomiting, balance, dizziness); and (4) affective (irritability, sadness, nervousness, feeling more emotional). The ImPACT also has a demographics section

Table 1. Vestibular/Ocular-Motor Screening (VOMS) Scores By Motion Sickness Susceptibility Group

VOMS Component	Motion Sickness Susceptibility Group, Mean ± SD (Median)		
	NONE (n = 70)	LOW (n = 95)	HIGH (n = 92)
Baseline VOMS symptoms	0.21 ± 0.59 (0.00)	0.49 ± 1.35 (0.00)	0.51 ± 1.09 (0.00)
Smooth pursuits	0.19 ± 0.62 (0.00)	0.47 ± 1.31 (0.00)	0.58 ± 1.49 (0.00)
Horizontal saccades	0.29 ± 0.68 (0.00)	0.74 ± 1.54 ^a (0.00)	0.82 ± 1.64 ^b (0.00)
Vertical saccades	0.24 ± 0.58 (0.00)	0.83 ± 1.60 ^b (0.00)	0.87 ± 2.02 (0.00)
Horizontal vestibular-ocular reflex	0.41 ± 0.84 (0.00)	1.27 ± 2.19 ^b (0.00)	1.10 ± 1.97 ^b (0.00)
Vertical vestibular-ocular reflex	0.36 ± 0.98 (0.00)	1.02 ± 1.95 ^b (0.00)	0.98 ± 2.17 ^b (0.00)
Visual motion sensitivity	0.41 ± 0.88 (0.00)	1.09 ± 2.12 (0.00)	1.11 ± 2.38 (0.00)
Near-point convergence distance, cm	3.42 ± 3.28 (0.00)	2.55 ± 2.65 (0.00)	2.59 ± 2.29 (0.00)
Near-point convergence, symptoms	0.17 ± 0.56 (2.33)	0.83 ± 1.86 ^b (2.17)	0.62 ± 1.28 ^b (2.29)

^a $P \leq .05$.

^b Different from NONE ($P \leq .01$).

= 2693.50, $z = -2.48$, $P = .01$, $r = .19$), and NPC symptoms ($U = 2754.50$, $z = -2.73$, $P = .006$, $r = .21$). No post hoc differences were evident between the LOW and HIGH MSSQ-S groups for horizontal saccades ($U = 4188.50$, $z = -0.59$, $P = .56$), vertical saccades ($U = 4162.50$, $z = -0.69$, $P = .49$), horizontal VOR ($U = 4360.00$, $z = -0.03$, $P = .98$), vertical VOR ($U = 4165.50$, $z = -0.66$, $P = .51$), and NPC symptoms ($U = 4321.50$, $z = -0.17$, $P = .87$; Table 1). We found no between-groups main effects for VOMS baseline symptoms ($\chi^2_{2,257} = 3.39$, $P = .18$), smooth pursuits ($\chi^2_{2,257} = 3.42$, $P = .18$), VMS ($\chi^2_{2,257} = 4.26$, $P = .12$), or NPC distance ($\chi^2_{2,257} = 2.74$, $P = .26$). In addition, participants with any motion sickness susceptibility (ie, LOW or HIGH group) indicated on the MSSQ-S (ie, a MSSQ-S score > 0) were 2.64 times more likely ($\chi^2_{1,257} = 7.94$, $P = .01$, 95% CI = 1.32, 5.26) to exhibit VOMS scores higher than the clinical cutoffs (ie, a VOMS total score on any component ≥ 2)⁴ at baseline than participants with no motion sickness (ie, NONE group).

A Comparison of Baseline Neurocognitive Performance and Symptoms Among Motion Sickness Susceptibility Groups

A total of 231 participants completed the baseline MSSQ-S, ImPACT, and symptom measures. Participants were categorized into NONE ($n = 52$), LOW ($n = 89$), and HIGH ($n = 90$) groups based on MSSQ-S scores above and below the median (7.07; excluding a score of zero). The MSSQ-S groups were not different in age ($F_{2,231} = 1.98$, $P = .14$, $\eta^2 = .02$) or concussion history ($F_{2,231} = 2.14$, $P = .12$, $\eta^2 = .02$). Females comprised 19% (10/52), 17% (15/89), and 28% (25/90) of the participants in the NONE, LOW, and HIGH groups, respectively. No sex differences were demonstrated when we compared the numbers of males and females in the NONE and HIGH ($\chi^2_{1,137} = 1.40$, $P = .24$), NONE and LOW ($\chi^2_{1,146} = .11$, $P = .74$), or LOW and HIGH ($\chi^2_{1,179} = 3.24$, $P = .07$) groups.

One-way ANOVAs revealed no between-groups main effects for the NONE ($n = 52$), LOW ($n = 89$), and HIGH ($n = 90$) MSSQ-S groups for verbal memory ($F_{2,230} = .09$, $P = .91$, $\eta^2 = .001$), visual memory ($F_{2,230} = .15$, $P = .86$, $\eta^2 = .001$), processing speed ($F_{2,230} = .78$, $P = .46$, $\eta^2 = .007$), or reaction time ($F_{2,230} = 2.21$, $P = .11$, $\eta^2 = .002$). The Kruskal-Wallis tests identified differences among MSSQ-S groups for total symptoms ($\chi^2_{2,231} = 8.87$, $P = .01$) and the cognitive-sensory symptom factor ($\chi^2_{2,230} = 7.30$, $P = .03$). Post hoc Mann-Whitney U tests indicated that the HIGH

group had higher total baseline symptom scores than the LOW ($U = 3325.50$, $z = -1.99$, $P = .05$, $r = .15$) and NONE ($U = 1647.50$, $z = -2.83$, $P = .005$, $r = .24$) groups. The HIGH MSSQ-S group displayed higher symptom scores for the baseline cognitive-sensory symptom factor than the NONE group ($U = 1732.00$, $z = -2.61$, $P = .009$, $r = .22$). No other significant post hoc comparisons were present for the cognitive-sensory factor (P values $> .05$). Sleep-arousal ($\chi^2_{2,231} = 4.86$, $P = .08$), vestibular-somatic ($\chi^2_{2,231} = 3.18$, $P = .20$), and affective ($\chi^2_{2,231} = 1.88$, $P = .39$) symptoms did not differ among groups (Table 2).

DISCUSSION

The primary purpose of our study was to examine the effects of motion sickness susceptibility on baseline vestibular, ocular-motor, neurocognitive, and symptom scores among healthy adolescent athletes. Athletes who endorsed low or high motion sickness susceptibility demonstrated higher baseline scores on the VOMS and were more likely to exhibit vestibular and ocular-motor symptoms and impairments that were greater than established clinical cutoffs. Although motion sickness susceptibility did not influence baseline neurocognitive scores, athletes with high motion sickness susceptibility demonstrated more total baseline symptoms and cognitive-sensory symptoms. Documenting the effects of motion sickness susceptibility on vestibular and ocular-motor function in an adolescent sample extends previous research⁹ conducted on older college-aged cohorts. Moreover, these findings may explain abnormal baseline VOMS scores in adolescent-aged athletes and should be taken into consideration when interpreting postconcussion VOMS performance.

Our findings are in agreement with previous literature describing patients with both SRC and vestibular impairments. In the current study, adolescent athletes with motion sickness susceptibility had a 2.64 times greater likelihood of VOMS scores over the clinical cutoffs and higher VOMS scores on all components except vertical VOR and VMS. Similarly, Kontos et al⁹ reported a 7.73 times greater likelihood for VOMS scores exceeding clinical cutoffs in collegiate athletes with a history of motion sickness. Collectively, these findings from athletic populations support previous results of research involving patients with vestibular impairments¹⁸ and further establish the relationship between motion sickness and vestibular function.

Motion sickness susceptibility did not influence baseline neurocognitive performance but did affect baseline symp-

Table 2. Baseline Neurocognitive Scores, Total Baseline Symptoms, and Baseline Symptom Factors by Motion Sickness Susceptibility Group

Measure	Motion Sickness Susceptibility Group, Mean ± SD (Median)		
	NONE (n = 52)	LOW (n = 89)	HIGH (n = 90)
Neurocognitive			
Verbal memory, %	86.71 ± 9.70 (88.00)	87.40 ± 9.20 (89.00)	87.23 ± 9.45 (87.23)
Visual memory, %	78.88 ± 12.87 (79.50)	78.51 ± 12.82 (78.00)	77.81 ± 10.61 (78.00)
Processing speed, s	37.43 ± 5.66 (37.67)	36.31 ± 6.69 (36.17)	36.04 ± 6.89 (35.68)
Reaction time, s	0.59 ± 0.07 (0.58)	0.61 ± 0.08 (0.60)	0.62 ± 0.08 (0.62)
Symptoms			
Total	3.37 ± 5.29 (1.50)	4.89 ± 8.04 (2.00)	5.96 ± 6.59 (4.00) ^{a,b}
Cognitive-sensory	0.88 ± 1.76 (0.00)	1.69 ± 3.36 (0.00)	1.82 ± 2.42 (1.00) ^{a,b}
Sleep-arousal	1.31 ± 2.05 (0.00)	1.53 ± 2.39 (0.00)	2.03 ± 2.47 (1.00)
Vestibular-somatic	0.37 ± 1.30 (0.00)	0.47 ± 1.41 (0.00)	0.52 ± 1.25 (0.00)
Affective	0.71 ± 1.64 (0.00)	0.91 ± 2.04 (0.00)	1.32 ± 2.51 (0.00)

^a Different from NONE ($P \leq .05$).

^b Different from LOW ($P \leq .05$).

tom reports on the PCSS. To date, the literature investigating the relationship between motion sickness susceptibility and baseline neurocognitive performance and symptoms is scant. In one of the few studies that examined motion sickness, neurocognitive function, and symptoms, Sufrinko et al⁶ reported similar neurocognitive performance among concussed athletes with high or no motion sickness susceptibility. In addition, concussed athletes with high motion sickness susceptibility exhibited higher affective concussion symptoms (eg, sadness, nervousness, feeling more emotional) at 1 week postinjury than concussed athletes with no motion sickness susceptibility. The increased baseline symptoms described by our athletes with high levels of motion sickness susceptibility versus those with low or no motion sickness susceptibility was consistent with the findings of Sufrinko et al.⁶ Moreover, these results could be attributed to the increased symptom awareness (ie, hypervigilance) or somatization or both that are linked to increased symptom reporting.²⁸ Motion sickness susceptibility symptoms and cognitive-sensory symptoms may overlap, which could influence postconcussion management. More specifically, concussed athletes with high motion sickness susceptibility may endorse more cognitive-sensory symptoms (ie, a positive relationship), which emphasizes the need for clinicians to consider motion sickness susceptibility when interpreting symptom reports and making postconcussion management decisions.

STRENGTHS AND LIMITATIONS

For our cross-sectional study, a large sample of adolescent athletes completed a series of concussion-related outcome measures and a motion sensitivity susceptibility questionnaire. However, most of these items were self-reported and limited by the bias inherent in these types of measures, including assumptions about effort, accuracy, and honesty with regard to participant responses. In addition, timing, maturation, and other biases commonly affect self-reported data and may have influenced the findings. Further, the MSSQ-S is not a diagnostic evaluation for motion sensitivity, which may have led to a larger number of highly motion-sensitive athletes in the current sample. The cross-sectional design and participant attrition did not allow for an examination of changes across the measures we addressed because not all participants who

completed the MSSQ-S completed the vestibular/ocular-motor, neurocognitive, and symptom outcome measures. In addition, the order in which these measures were administered was not randomized. An order effect could have exacerbated or changed symptom reporting on the VOMS and PCSS due to symptom provocation associated with completing a vestibular/ocular-motor screening followed by a computer-based visual test. Future authors should counterbalance administration order to control for this threat to internal validity. Moreover, researchers should investigate larger samples that include more females to address the potential interaction between sex and motion sickness susceptibility on vestibular/ocular-motor, neurocognitive, and symptom assessments used to evaluate concussion.

CONCLUSIONS

To our knowledge, we are the first to examine the influence of motion sickness susceptibility on vestibular and ocular-motor and related concussion outcomes in a large sample of healthy athletes at baseline. Athletes with any motion sickness sensitivity reported more vestibular and ocular-motor symptoms and impairment versus athletes without motion sickness sensitivity. These findings support the importance of considering preexisting modifying or risk factors such as motion sickness susceptibility that might influence specific domains of baseline and postinjury performance and symptoms. The MSSQ-S may be a useful clinical tool for identifying athletes with preexisting vestibular and ocular-motor symptoms and impairment. This information should be considered by clinicians when interpreting baseline as well as postinjury concussion data.

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Address correspondence to R. J. Elbin, PhD, Office for Sport Concussion Research, Department of Health, Human Performance and Recreation, University of Arkansas, 155 Stadium Drive, Fayetteville, AR 72701. Address e-mail to rjelbin@uark.edu.