# The Effects of Headache on Clinical Measures of Neurocognitive Function

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**Objective:** To examine effects of preseason baseline headache and posttraumatic headache (PTH) on neurocognitive function.

**Design:** Retrospective repeated measures study with headache groups formed regarding baseline headache score (0 = negative headache; 1–6 = positive headache) and day 1 postinjury headache score (0 = no headache; 1–2 = mild headache; 3–6 = moderate–severe headache).

**Setting:** Clinical athletic training setting and sports medicine research laboratory.

**Participants:** High-school and collegiate athletes with a concussion.

**Independent Variables:** Preseason baseline headache, PTH, test-day.

**Main Outcome Measures:** A Graded Symptom Checklist (GSC) was used to assess symptoms. The Automated Neuropsychological Assessment Metrics (ANAM) and the Standardized Assessment of Concussion (SAC) were used to assess neuropsychological function and mental status. The Balance Error Scoring System (BESS) was used to assess postural stability.

**Results:** Both baseline headache groups displayed a higher symptom endorsement and higher symptom severity at day 1 postinjury and improved by day 7 postinjury. The positive headache group reported an even greater increase in symptom severity and presence ($P < 0.05$). ANAM revealed deficits in both groups 1 day postinjury. All PTH headache groups displayed a difference in symptom number and severity with the increase being magnified by headache severity ($P < 0.05$). Individuals reporting moderate–severe PTH displayed increased deficits subacutely but improved by 5–7 days postinjury on overall neuropsychological performance, reaction time, and working memory ($P < 0.05$). Deficits were observed for all ANAM measures except simple reaction time 1 (SRT 1) and match to sample subacutely and improved over time ($P < 0.05$). The SAC yielded an interaction ($P < 0.05$) for baseline headache. The BESS yielded no significant findings.

**Conclusions:** Clinicians should consider headache when assessing concussion and during preseason baseline assessments because headache may affect symptom presence and other clinical measures of concussion.

**Key Words:** headache, concussion, balance, cognition

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## INTRODUCTION

Sport-related concussion may result in a cluster of symptoms ranging from headache to dizziness. Headache is a hallmark symptom of concussion and occurs in up to 86% of concussed individuals. Loss of consciousness (LOC) and posttraumatic amnesia (PTA) are frequently used as indicators of concussion, although LOC occurs in only 9% of cases and PTA occurs in only 23%.[1] Despite being the most common concussive symptom, posttraumatic headache (PTH) is often dismissed. Although common, there is a limited understanding about the relevance of headache and its relationship to return-to-play (RTP) decisions. Current literature suggests a relationship between neuropsychological test scores, prevalence of symptoms, and the presence and severity of PTH.[2] Posttraumatic migraine (PTM) may also affect clinical measures. Individuals suffering from PTH with PTM features may suffer greater neurocognitive deficits compared to a PTH group without those features and when compared to a nonheadache group.[3]

These studies indicate important effects of PTH on clinical measures of concussion; however, the presence and effects of preinjury headache, whether episodic or chronic, have not been thoroughly investigated. Because of a limited number of studies, understanding of the effects of both PTH and preinjury headache on postinjury measures of concussion is incomplete. Many factors play a role, including type and severity of headache, number of previous concussions, severity of previous concussions, location/severity of impact, and severity of current injury. Literature suggests all of these factors play a role in the manifestation and duration of symptoms. Guskiewicz[4] reported that 30% of individuals suffering from 3 or more concussions reported symptoms lasting longer than 1 week following injury compared to 14.6% of individuals with history of only 1 previous concussion. One of few studies involving headache and concussion reported an increase in number of symptoms and

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severity in individuals with a higher PTH score.[2] Although literature suggests a relationship between number of previous concussions, symptom frequency, and other clinical measures, the role of headache in this relationship is less understood.

Clinical outcome measures provide clinicians with valuable information. Certain symptoms may signal disruption in brain function. Because headache is the most common of these symptoms, its effects on clinical measures are important to clinicians in management and RTP decisions. Headache is also commonly experienced within healthy individuals, which may underemphasize the clinical significance of headache during postinjury evaluations. Therefore, the primary purpose of this study was to examine the effects of PTH and preinjury assessment headache on clinical measures of concussion. Our secondary purpose was to examine the association between both preinjury assessment headache and PTH with frequency of previous concussion.

## METHODS

### Subjects

Subjects were recruited for this study on the basis of participation in high school or collegiate basketball, football, lacrosse, or soccer at randomly selected high schools (110) and colleges (14) across the eastern United States. Subjects (age 16.65 ± 1.87 years) attended schools that employed a certified athletic trainer to oversee athletic health care. The certified athletic trainers were responsible for all data collection at their respective schools.

### Testing Protocol

This study involved data collected on 247 athletes with a concussion. All subjects completed preseason baseline testing and serial postinjury testing. Testing involved completion of the Graded Symptom Checklist (GSC), Standardized Assessment of Concussion (SAC), and Balance Error Scoring System (BESS) at baseline and on postinjury days 1, 3, and 7. Subjects completed Automated Neuropsychological Assessment Metrics (ANAM) testing during a subacute testing period (day 1–2 postinjury) and a prolonged testing period (day 5–7 postinjury).

Subjects were part of a larger 4-arm study; however, we only used 3 of the study arms in our study (Table 1). In relation to preseason headache, subjects were grouped by those with a positive baseline headache score (PHA) and those with a negative baseline headache score (NHA). For PTH scores, subjects were grouped by reported GSC headache score at day 1 postinjury. Those with a PTH score of 0 were placed in the no-headache group, those with a PTH score of 1–2 were placed in the mild PTH group, and those with a PTH score of 3–6 were placed in the moderate to severe PTH group.

## INSTRUMENTATION

### Graded Symptom Checklist

The GSC is a 7-point Likert scale that allows individuals to self-report and rate concussive symptoms. Current literature suggests this is a commonly used measure of symptomatology following sports-related concussion.[5,6] We used a 20-item checklist that included common symptoms associated with concussion. Total symptom score and total number of symptoms endorsed served as dependent variables.

### Automated Neuropsychological Assessment Metrics

For the purposes of this study, ANAM was used in assessing neuropsychological function. We used an overall composite score and the scores of 5 subtests. The 5 subtests in the study were simple reaction time (SRT), continuous performance test (CPT), math processing (MTH), Sternberg memory (STN), and match to sample (MTS), which assess various cognitive processes including reaction time, working memory, concentration, and mental processing. Subjects were tested on all 5 subtests with the SRT and CPT subtests being repeated at the end of the battery during each testing session.[7,8] We then calculated the composite *Z*-score, which represents overall neuropsychological performance, by adding the *Z*-scores from all ANAM subtest individual throughput scores (number of correct responses per minute). Composite scores and throughput score served as dependent variables.

### Standardized Assessment of Concussion

The SAC was used to assess mental status. This instrument assesses orientation, immediate memory, delayed recall, and concentration. The SAC has been shown to be sensitive and specific in detecting mental status deficits.[9–12] The total SAC score was used as a dependent variable.

### Balance Error Scoring System

The BESS, which has been shown to be a reliable and valid measure of postural stability[13,14] was used in our study. We used 6, 20-second trials of 3 different stances (double-leg, single-leg, tandem), performed of 2 different surfaces (firm, foam) with eyes closed. Individuals were asked to perform the single-leg task on the nondominant foot. Dominance was defined by which leg would be used to kick a ball.[13,14] Errors

**TABLE 1.** Breakdown of Study Arms

| Study Arm | Graded Symptom Checklist | Concussion History Questionnaire | Standardized Assessment of Concussion | Balance Error Scoring System | Automated Neuropsychological Assessment Metrics |
|---|---|---|---|---|---|
| 1 (n = 100) | X | X | X | X | — |
| 2 (n = 247) | X | X | — | — | — |
| 3 (n = 45) | X | X | X | X | X |

Part of a 4-arm study. Our study only used 3 arms.

were recorded if the individual lifted hands off of their iliac crest, abducted or flexed their hip to greater than 30 degrees, stepped, stumbled or fell, opened eyes, or remained out of the testing position for longer than 5 seconds. Total BESS error score was used as a dependent variable.

## Data Analyses

Data analyses were conducted using SPSS 13.0 (Chicago, Illinois). Scores on the BESS, SAC, and GSC investigating the effects of headache reported at baseline were analyzed using 2 × 4 repeated measures, mixed model analyses of variance (ANOVAs). The effects of PTH on these measures were analyzed using 3 × 3 repeated measures mixed model ANOVAs. The effects of headache reported at baseline on ANAM were analyzed using 2 × 3 repeated measures mixed model ANOVAs. Finally, 3 × 2 repeated measures mixed model ANOVAs were used to determine the effects of PTH on ANAM. Tukey post-hoc analyses comparing specific group means were performed on all significant interactions. The association between previous history of concussion and headache reported at baseline and between previous history of concussion and PTH was analyzed using 2 chi square tests of association. Level of significance ($P < 0.05$) was set a priori for all statistical analyses. Headache score was removed from all GSC analyses as it served as the classifying factor.

# RESULTS

We observed a significant relationship between preseason baseline headache group and concussion history ($\chi 2(3) = 14.48$, $P = 0.002$). A Crosstabs analysis suggested individuals who reported 3 or more previous concussions were more likely to report headache at baseline (Table 2). There was no significant association between PTH and a history of previous concussion. Seventy-six of the 106 subjects who reported headache at baseline also reported PTH at day 1 postinjury.

The next series of analyses examined the effects of headache reported at baseline on the GSC, the SAC, and the BESS. A repeated measures ANOVA revealed no significant effects of headache on the BESS. An interaction effect ($F_{3,267} = 3.25$, $P = 0.027$) and a main effect of day ($F_{3,267} = 5.18$, $P = 0.002$) were observed for the SAC. An interaction effect was also observed for symptom total score ($F_{3,675} = 3.41$, $P = 0.03$) and total number of symptoms endorsed ($F_{3,675} = 5.63$, $P = 0.024$) on the GSC. Tukey post-hoc analyses revealed that individuals reporting headache at baseline displayed an increase in symptom severity and presence when compared to the group reporting no headache at baseline on day 1 postinjury and had a similar symptom score by day 7 postinjury. A main effect of day was observed for symptom total score ($F_{1,225} = 77.75$, $P < 0.005$) and total number of symptoms endorsed ($F_{3,675} = 108.94$, $P < 0.005$) (Table 3), suggesting that all groups displayed a greater number of symptoms and an increase in severity at days 1 and 3 postinjury with symptoms resolving by day 7 postinjury.

Investigation of the effects of headache reported at baseline on neuropsychological performance (ANAM) revealed no significant interactions for any ANAM subtest, although a main effect of day was observed for all subtests ($P < 0.05$), suggesting a practice effect may have resulted in improved scores during serial testing.

The final series of analyses examined the effects of PTH on the GSC, the SAC, the BESS, and ANAM. A repeated measures ANOVA revealed no significant findings for the SAC or the BESS. However, interaction effects and a main effect of day were observed on the GSC for symptom total score ($F_{4,458} = 24.98$, $P < 0.005$) and total number of symptoms endorsed ($F_{4,458} = 20.88$, $P < 0.005$) (Table 4). All groups displayed an increase in symptom severity and presence at day 1 postinjury but improved to within normal range by day 7 postinjury. Tukey post-hoc analyses revealed the moderate–severe PTH group displayed an increase in symptoms and severity when compared to the other 2 groups across all days; the mild PTH group was significantly different than the no-PTH group only at day 1 postinjury for total symptoms endorsed and total symptom score.

Regarding total symptoms endorsed, an increased score was also found at day 3 postinjury for the mild PTH group compared to the no-PTH group. When considering the effect of PTH on neuropsychological performance, a repeated measures ANOVA revealed interactions for the ANAM composite *Z*-score ($F_{2,41} = 4.98$, $P = 0.01$) and the individual throughput scores for SRT 1 ($F_{2,40} = 3.36$, $P = 0.46$), Continuous Performance Test 1 (concentration and working memory) ($F_{2,41} = 3.89$, $P = 0.028$), Sternberg Memory Search (working memory) ($F_{2,39} = 3.67$, $P = 0.035$), SRT 2 ($F_{2,38} = 6.06$, $P = 0.005$), and Continuous Performance Test 2 ($F_{2,42} = 4.64$, $P = 0.015$). Tukey post-hoc analyses revealed these interactions to be a result of significant improvement in the severe PTH group's scores during the prolonged testing session. A main effect of day ($P < 0.05$) was observed for all throughput measures except matching to sample (Table 5) because all groups score lower at the subacute testing period and improved during the prolonged testing period.

**TABLE 2.** Number of Previous Concussions per Group (Last 7 Years)*

| | 0 | 1 | 2 | 3+ |
|---|---|---|---|---|
| NHG (n = 258) | 69.0% (177) | 23.0% (60) | 7.8% (20) | 0.3% (1) |
| PHG (n = 106) | 59.5% (63) | 24.5% (26) | 9.4% (10) | 6.6% (7) |

*$\chi^2 = 14.48$; df = 3; $P = 0.002$; percentages (%) are expressed as a proportion of subjects of each group.

NHG, negative preseason headache group; PHG, positive preseason headache group.

# DISCUSSION

## Preseason Baseline Headache

To our knowledge no study has examined effects of headache in nonconcussed athletes on clinical measures of concussion. Our study suggests that individuals reporting baseline headache may display an increased presence and severity of other symptoms. In regard to neuropsychological testing, there were no significant interactions found,

**TABLE 3.** Baseline Headache Means and Standard Deviations

| | Baseline | Day 1 Postinjury | Day 3 Postinjury | Day 7 Postinjury | Group Main Effect | Group X Day Interaction | Day Main Effect |
|---|---|---|---|---|---|---|---|
| Symptom total | | | | | | | |
| No headache at baseline | 1.62 (3.47) | 10.99 (12.98) | 5.55 (10.37) | 1.96 (6.48) | $F_{1,225} = 3.175$ | $F_{3,675} = 3.414$ | $F_{3,675} = 77.75$ |
| +Headache at baseline | 5.65 (6.41) | 13.56 (13.13) | 6.46 (9.92) | 1.60 (4.56) | $P = 0.076$ | $P = 0.033$ | $P < 0.005$ |
| | 2.74 (4.82) | 11.70 (13.04) | 5.80 (10.23) | 1.86 (5.99) | | | |
| Symptoms endorsed | | | | | | | |
| No headache at baseline | 0.91 (1.59) | 4.80 (4.11) | 2.77 (3.95) | 0.92 (2.39) | $F_{1,83} = 5.57$ | $F_{3,675} = 5.63$ | $F_{3,675} = 108.94$ |
| +Headache at baseline | 2.86 (2.84) | 5.76 (3.99) | 3.28 (3.56) | 0.83 (1.83) | $P = 0.019$ | $P = 0.024$ | $P < 0.005$ |
| | 1.45 (2.12) | 5.07 (4.09) | 2.92 (3.85) | 0.89 (2.24) | | | |
| SAC Total score | | | | | | | |
| No headache at baseline | 26.85 (1.64) | 26.62 (2.79) | 27.62 (1.72) | 26.08 (1.74) | $F_{1,89} = 0.283$ | $F_{3,267} = 3.250$ | $F_{3,267} = 5.18$ |
| +Headache at baseline | 27.35 (1.85) | 26.88 (1.79) | 26.73 (2.66) | 27.46 (1.66) | $P = 0.596$ | $P = 0.027$ | $P = 0.002$ |
| | 26.99 (1.71) | 26.69 (2.54) | 27.36 (2.59) | 27.90 (1.73) | | | |
| BESS Total score | | | | | | | |
| No headache at baseline | 11.85 (4.76) | 14.23 (7.11) | 11.13 (6.65) | 9.60 (5.66) | $F_{1,83} = 0.030$ | $F_{3,249} = 0.202$ | $F_{3,249} = 15.99$ |
| +Headache at baseline | 11.20 (4.27) | 14.08 (7.27) | 11.52 (4.58) | 9.24 (4.86) | $P = 0.863$ | $P = 0.869$ | $P < 0.005$ |
| | 11.66 (4.60) | 14.19 (7.12) | 11.25 (6.02) | 9.50 (5.42) | | | |

suggesting that headache at baseline may have little effect on this type of neuropsychological testing battery. All groups displayed deficits at the subacute testing session (days 1–2 postinjury) with improvements at the prolonged testing session (days 5–7 postinjury). This is consistent with literature (ie, deficits appear postinjury and begin to resolve over time). As a result of practice effects, individuals often demonstrate improvement in testing by day 7 postinjury.

Another important finding was the association between previous history of concussion and headache reported at baseline. Individuals suffering from 3 or more prior concussions were more likely to report headache at baseline than individuals with a history of less than 3 prior concussions. This finding is consistent with literature suggesting that after 3 or more prior concussions, long-term repercussions and a delayed recovery may be present.[4] This association parallels our finding, suggesting that individuals experiencing headache at baseline display an increase in symptoms and slower symptomatic recovery than individuals who do not report headache at the preseason baseline.

**TABLE 4.** Posttraumatic Headache Clinical Measures Scores Means and Standard Deviations

| | Day 1 Postinjury | Day 3 Postinjury | Day 7 Postinjury | Group Main Effect | Group X Day Interaction | Day Main Effect |
|---|---|---|---|---|---|---|
| Symptom total | | | | | | |
| No PTH | 2.32 (3.42) | 1.19 (3.94) | .43 (2.21) | $F_{2,229} = 29.75$ | $F_{4,458} = 4.98$ | $F_{2,458} = 100.21$ |
| Mild PTH | 8.07 (8.27) | 3.35 (5.43) | .81 (2.06) | $P < 0.005$ | $P < 0.005$ | $P < 0.005$ |
| Moderate–Severe PTH | 19.16 (15.23) | 10.02 (13.43) | 3.32 (8.35) | | | |
| | 11.88 (13.31) | 5.90 (10.40) | 1.86 (5.94) | | | |
| Symptoms endorsed | | | | | | |
| No PTH | 1.53 (2.12) | 0.66 (1.76) | 0.21 (0.93) | $F_{1,83} = 5.57$ | $F_{4,458} = 20.878$ | $F_{2,458} = 62.96$ |
| Mild PTH | 4.31 (3.41) | 2.25 (2.83) | 0.58 (1.46) | $P = 0.019$ | $P < 0.005$ | $P < 0.005$ |
| Moderate–Severe PTH | 7.28 (3.98) | 4.51 (4.58) | 1.42 (2.90) | | | |
| | 5.08 (4.11) | 2.94 (3.89) | .88 (2.22) | | | |
| SAC total score | | | | | | |
| No PTH | 27.50 (1.75) | 27.56 (1.37) | 28.25 (1.57) | $F_{2,89} = 0.283$ | $F_{4,180} = 0.673$ | $F_{2,180} = 8.46$ |
| Mild PTH | 26.44 (2.99) | 27.22 (1.72) | 27.94 (1.97) | $P = 0.596$ | $P = 0.596$ | $P = 0.001$ |
| Moderate–Severe PTH | 26.63 (2.32) | 27.46 (2.50) | 27.63 (1.58) | | | |
| | 26.71 (2.53) | 27.39 (2.04) | 27.86 (1.73) | | | |
| BESS total score | | | | | | |
| No PTH | 10.71 (5.23) | 9.86 (4.47) | 8.86 (3.37) | $F_{2,84} = 0.030$ | $F_{4,168} = 1.37$ | $F_{2,168} = 2.12$ |
| Mild PTH | 13.31 (6.15) | 10.03 (3.40) | 8.06 (5.54) | $P = 0.05$ | $P = 0.252$ | $P < 0.005$ |
| Moderate–Severe PTH | 16.02 (7.76) | 12.41 (7.27) | 10.66 (5.62) | | | |
| | 14.17 (7.04) | 11.13 (6.01) | 9.41 (5.38) | | | |

PTH, posttraumatic headache; SAC, Standardized Assessment of Concussion; BESS, Balance Error Scoring System.

**TABLE 5.** Posttraumatic Headache ANAM Throughput Scores Means and Standard Deviations

| | Subacute Postinjury | Prolonged Postinjury | Group Main Effect | Group X Day Interaction | Day Main Effect |
|---|---|---|---|---|---|
| Composite Z | | | | | |
| No PTH | 1.60 (5.69) | 4.74 (6.33) | $F_{1,41} = 0.368$ | $F_{2,41} = 4.98$ | $F_{1,41} = 25.343$ |
| Mild PTH | 1.39 (5.24) | 3.44 (5.49) | $P = .694$ | $P = 0.012$ | $P < 0.005$ |
| Moderate–Severe PTH | −2.23 (6.93) | 5.25 (4.80) | | | |
| | −0.05 (5.93) | 4.45 (5.34) | | | |
| SRT 1 | | | | | |
| No PTH | 241.43 (47.59) | 235.94 (51.85) | $F_{1,40} = 0.029$ | $F_{2,40} = 3.36$ | $F_{1,40} = 3.59$ |
| Mild PTH | 233.99 (47.46) | 243.14 (26.95) | $P = 0.969$ | $P = 0.045$ | $P = 0.065$ |
| Moderate–Severe PTH | 224.63 (39.70) | 257.75 (36.49) | | | |
| | 230.63 (43.20) | 249.10 (36.60) | | | |
| CPT 1 | | | | | |
| No PTH | 112.36 (27.65) | 130.31 (16.15) | $F_{1,41} = 0.324$ | $F_{2,41} = 3.89$ | $F_{1,41} = 23.11$ |
| Mild PTH | 109.46 (19.19) | 122.43 (17.75) | $P = 0.725$ | $P = 0.028$ | $P < 0.005$ |
| Moderate–Severe PTH | 95.39 (28.87) | 133.78 (26.63) | | | |
| | 103.21 (29.09) | 129.10 (22.44) | | | |
| Math | | | | | |
| No PTH | 24.48 (5.86) | 27.35 (7.95) | $F_{1,42} = 0.188$ | $F_{2,42} = 0.928$ | $F_{1,42} = 18.19$ |
| Mild PTH | 22.82 (7.87) | 29.61 (17.09) | $P = 0.829$ | $P = 0.403$ | $P < 0.005$ |
| Mod-Severe PTH | 20.88 (4.64) | 28.39 (5.63) | | | |
| | 22.21 (6.19) | 28.64 (11.17) | | | |
| Match to sample | | | | | |
| No PTH | 41.83 (8.81) | 42.76 (11.67) | $F_{1,42} = 0.583$ | $F_{2,42=} 3.19$ | $F_{1,42} = 3.84$ |
| Mild PTH | 41.54 (10.18) | 42.36 (14.12) | $P = 0.562$ | $P = 0.051$ | $P = 0.057$ |
| Moderate–Severe PTH | 33.34 (10.21) | 44.22 (14.71) | | | |
| | 37.77 (10.61) | 43.30 (13.74) | | | |
| Sternberg | | | | | |
| No PTH | 77.46 (15.54) | 99.65 (41.89) | $F_{1,39} = 1.35$ | $F_{2,39} = 3.67$ | $F_{1,39} = 10.76$ |
| Mild PTH | 82.47 (15.73) | 81.52 (20.19) | $P = 0.270$ | $P = 0.035$ | $P = 0.002$ |
| Mod-Severe PTH | 66.09 (23.94) | 85.71 (17.29) | | | |
| | 73.84 (21.08) | 86.54 (23.95) | | | |
| SRT 2 | | | | | |
| No PTH | 239.99 (43.41) | 236.60 (41.29) | $F_{138} = 0.082$ | $F_{238} = 6.06$ | $F_{1,38} = 5.60$ |
| Mild PTH | 230.42 (38.81) | 235.01 (37.70) | $P = 0.922$ | $P = 0.005$ | $P = 0.023$ |
| Moderate–Severe PTH | 210.10 (55.65) | 253..57 (35.05) | | | |
| | 223.86 (48.08) | 243.02 (37.37) | | | |
| CPT 2 | | | | | |
| No PTH | 111.85 (26.55) | 130.29 (19.87) | $F_{1,42} = 0.259$ | $F_{2,42} = 4.64$ | $F_{1,42} = 27.75$ |
| Mild PTH | 111.46 (22.16) | 121.72 (22.72) | $P = .758$ | $P = 0.015$ | $P < 0.005$ |
| Moderate–Severe PTH | 97.64 (27.96) | 131.86 (21.13) | | | |
| | 105.08 (26.16) | 127.83 (21.59) | | | |

PTH, posttraumatic headache; Z, Z-score; SRT, simple reaction time; CPT, continuous performance test.

## Posttraumatic Headache

We also examined the effects of posttraumatic headache on these same measures. We found that individuals reporting PTH reported an increased number and severity of symptoms when compared to individuals not reporting headache. This finding was magnified by severity, suggesting that headache severity may indicate increased acute dysfunction following concussive injury. We also found that individuals reporting moderate to severe PTH performed more poorly on mental processing and reaction time tasks than the other groups at day 1 postinjury, but displayed significant improvement by day 7 postinjury. Although this finding may seem ambiguous, individuals with moderate to severe PTH were evaluated more frequently because they reported more symptoms, leading to these individuals completing testing more often than individuals in the other groups and therefore increasing the practice effects. These results suggest that the increased neurocognitive deficits in these individuals were likely a result of both the headache and the concussive injury.

## Headache, Concussion, and Pain

Current literature suggests pain, including headache, affects concentration, mental processing, and reaction time.

Our study reinforces these findings because PTH significantly affected mental processing and the reaction time measures. These findings support literature suggesting PTH is a sign of incomplete recovery and may be an indicator of other neurocognitive deficits.[2] Because headache often precedes other postconcussive symptoms, the neurocognitive and other aspects of postconcussion syndrome may be at least, in part, the result of the headache.[15] Whereas some investigators have observed neuropsychological impairment in cases of PTH, there are no published studies that help to explain the potential sources of these deficits. Many confounding factors, including pain, actual brain injury or other causes may contribute to neuropsychological impairment.[16]

Athletes with posttraumatic headache in our study scored worse on postinjury assessments when compared to those without posttraumatic headache. We found significant effects of PTH on neuropsychological measures but no significant effects of headache reported at baseline on these same measures, suggesting that the differences may be a result of the combination of headache presence and concussion. The observed differences may best be explained by the notion that headache, concussion, and general pain may all have some effect on neurocognitive function.

### Limitations

The present study is not without limitations. The number of males greatly outnumbered females in our study, which may have lead to skewed data, especially in relation to headache because females in general are more likely to experience headache. Additionally, we only tested out to day 7 postinjury. Although many of the symptoms of concussions resolve by day 7 postinjury, many do not resolve until day 10 postinjury or beyond. Additionally, several athletic trainers participating in the study conducted their testing at different sites, which may have resulted in some variability in testing procedures and timing. We believe the greatest limitation of our study was the small subset of individuals completing the ANAM testing battery at all times outlined in the study protocol. In some cases, this lead to a small sample size, decreased power, and an unbalanced group design that may have resulted in an inability to identify true group differences.

### Clinical Implications

Headache is common in both athletes and nonathletes, supporting a need for a better understanding of its effects on clinical measures of concussion and neuropsychological assessment measures. Our study suggests that individuals suffering from headache at baseline, as well as individuals reporting headache at day 1 postinjury, display an increased number of symptoms and an increase in intensity of symptoms initially following injury and throughout the course of recovery, reinforcing the current head trauma literature emphasizing the importance of being asymptomatic before returning to play. With regard to PTH and symptoms, headache may represent an incomplete recovery following injury. Our study only followed subjects out to postinjury day 7, and previous literature has only investigated out to day 10. Future research should investigate these effects further from time of injury and the effects of headache in a healthy, physically active population. Studies should include both no-headache and headache test sessions to isolate the effects of headache on these clinical measures. Finally, our study reinforces the suggestion by other researchers (Randolph 2005, McCrea 2005) that neuropsychological testing should be used to measure recovery only after complete symptom resolution.[17,18] If the athlete is asymptomatic, the neuropsychological test results would provide clinicians with more valuable information regarding neurocognitive abilities of the individual, independent of symptoms that may be influencing the assessment.

## CONCLUSIONS

Although other concussion studies have examined common clinical measures, few have examined the effects of headache on those measures. Current research suggests that headache may affect an individual's performance on neurocognitive tasks. These studies display an increase in postconcussive symptoms and deficits in neuropsychological test scores in individuals reporting PTH.[2,3] Our findings are consistent with those of Collins et al[2] and Mihalik et al[3] because individuals reporting PTH display an increase in the incidence and severity of symptoms and increased neurocognitive deficits. There has been some contemplation in the literature regarding the role of headache in RTP decisions. Our study suggests that individuals with PTH demonstrate increased neurocognitive deficits following concussion and that headache may be an indicator of decreased neurologic function and/or may be a causative factor in the increased deficits.

As clinicians, we should be mindful of headache presence in our healthy and unhealthy athletes both preinjury and postinjury because the presence of a headache apparently affects cognitive function to some degree. These findings may cause clinicians to rethink the practice of conducting preseason baseline assessments during preseason camp, when headaches may be present as a result of dehydration, heat, or overexertion and therefore confounding the results of the baseline testing measures. Clinicians should consider these findings when interpreting clinical measures and ask whether an individual's headache (presence and severity) could be negatively influencing the cognitive abilities clinical measures are designed to assess. Clinicians should be especially mindful of headache in athletes with a concussion because it is a sign of incomplete recovery and impairment in neurologic function.

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