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The Persistent Influence of Concussive Injuries on Cognitive Control and Neuroelectric Function

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Context: Increasing attention is being paid to the deleterious effects of sport-related concussion on cognitive and brain health.

Objective: To evaluate the influence of concussion incurred during early life on the cognitive control and neuroelectric function of young adults.

Design: Cross-sectional study.

Setting: Research laboratory.

Patients or Other Participants: Forty young adults were separated into groups according to concussive history (0 or 1+). Participants incurred all injuries during sport and recreation before the age of 18 years and were an average of 7.1 ± 4.0 years from injury at the time of the study.

Intervention(s): All participants completed a 3-stimulus oddball task, a numeric switch task, and a modified flanker task during which event-related potentials and behavioral measures were collected.

Main Outcome Measure(s): Reaction time, response accuracy, and electroencephalographic activity.

Results: Compared with control participants, the concussion group exhibited decreased P3 amplitude during target detection within the oddball task and during the heterogeneous condition of the switch task. The concussion group also displayed increased N2 amplitude during the heterogeneous version of the switch task. Concussion history was associated with response accuracy during the flanker task.

Conclusions: People with a history of concussion may demonstrate persistent decrements in neurocognitive function, as evidenced by decreased response accuracy, deficits in the allocation of attentional resources, and increased stimulus-response conflict during tasks requiring variable amounts of cognitive control. Neuroelectric measures of cognitive control may be uniquely sensitive to the persistent and selective decrements of concussion.

Key Words: concussions, inhibition, mental flexibility, attention, P3, N2

Key Points

- Seven years after concussion, participants displayed disrupted higher-order neurocognition in the form of chronically impaired attention, working memory, inhibition, and interference control.
- The observed deficits in attention and conflict monitoring were only evident when cognitive demands were increased. Subtle deficits may remain unrecognized with other types of testing.

During the past decade, increased research efforts have been dedicated toward understanding the causes of brain and cognitive dysfunction stemming from concussive injuries. Concussions have been described as a "silent epidemic" by the Centers for Disease Control and Prevention,[1] and estimates of the incidence in the United States range from a conservative 300 000 per year[2] in 1997 to the more recent estimate of nearly 4 million cases per year.[3] As 15%–20% of these injuries result from sport participation,[4] sport-related concussion represents a growing public health concern. *Concussion* can be defined as a complex pathophysiologic process affecting the brain that is caused by a direct blow or an impulsive force transmitted to the head.[5] Injured persons commonly display deficits in cognition, postural control, and symptoms,[6] so the cost to society is heavy, with known negative effects on academic[7–9] and vocational[10] performance. The annual economic burden of concussions approaches $17 billion in direct and indirect expenses in the United States,[11] warranting more investigation in the clinical and laboratory settings.

Based on clinical evaluations, injured people typically return to a preinjury level of functioning within 7 to 10 days,[12] a time frame mirroring the acute neurometabolic cascade associated with concussion.[13] Investigations of young adult athletes who have progressed past the acute stages of injury indicate normal performance on a variety of clinical tests,[14–17] leading to a general belief that concussion is a transient brain injury. However, the transient view of concussion has recently come into question, as a growing body of evidence illustrates numerous chronic nervous system dysfunctions and cognitive deficits stemming from these injuries.[18–28] Further, recent epidemiologic reports[16,29] reveal increased prevalence of mild cognitive impairment, dementia, and Alzheimer disease in retired contact-sport athletes. Evidence from these studies appears to diverge from the concept of concussion as a transient injury.

Given this divergence and the lack of a definitive diagnostic tool, identifying aspects of cognition that are sensitive to subtle concussion-related deficits during the postacute phase is warranted. Because concussive injuries are inherently difficult to assess[30] and result in a wide

variety of injury outcomes, assessing multiple aspects of cognitive functioning (eg, planning, memory, cognitive flexibility) may provide further insight into the nature and duration of these injuries.[31]

The aspects of cognitive functioning described by Aubry et al[31] fall under the domain of *cognitive control*, which designates a subset of goal-directed, self-regulatory operations involved in the selection, scheduling, and coordination of computational processes underlying perception, memory, and action.[32,33] Recent evaluations of the cognitive control of concussed persons demonstrate that tasks requiring this feature may be sensitive to detecting persistent cognitive deficits. For example, Pontifex et al[26] observed deficits in cognitive control, as indicated by decreased response accuracy during a modified flanker task, which requires variable amounts of inhibitory control (ie, an aspect of cognitive control) in previously concussed persons an average of 2.9 years after injury. Also using a flanker task, de Beaumont et al[20] observed decreased response accuracy in previously concussed persons an average of 34.7 years after injury relative to age-matched control participants. In addition, Ellemberg et al[22] noted deficits in a group of previously concussed athletes 6 months after injury during the Stroop color-word test, which further measures inhibitory control, and the Tower of London DX task, which requires planning, working memory, and cognitive flexibility. Together, these studies provide convergent evidence that tasks requiring various aspects of cognitive control may be well suited for examining the relationship between concussion history and prolonged cognitive dysfunction. However, further examination of task specificity appears necessary if future researchers are to adequately detail this relationship.

In addition to focusing on cognitive control, investigators have recently begun to incorporate sensitive measures of brain function in sport-related concussion research. Electroencephalography and event-related potentials (ERPs) in particular have emerged as valuable tools to evaluate covert neurocognitive deficits between stimulus engagement and response execution that stem from concussion. Therefore, electroencephalography may contribute to the development and refinement of differential diagnostic information for those with atypical clinical recovery after concussive injuries. The benefit of the ERP approach lies in its temporal sensitivity, which allows researchers to parse individual components in the stimulus-response relationship. Recent investigations[19,23,34–36] have demonstrated the efficacy of neuroelectric measures in detecting neurocognitive deficits associated with a concussion history. Beyond providing a unique method for researchers and clinicians to monitor enduring neurocognitive alterations, ERPs may serve as a measure of treatment effectiveness.

In particular, the P3 component has been of considerable interest in recent concussion research. The P3 component can be further divided into interrelated but distinct subcomponents, the P3b (P300) and P3a, which are differentiated by both the context in which they occur and scalp topography.[37] The P3b component, which is evoked in response to an infrequently occurring target stimulus, is believed to reflect the allocation of attentional resources (as indexed by component amplitude[37]) and stimulus classification and evaluation speed (as indexed by component latency[38,39]) and demonstrates a centroparietal maximum.[37] The P3a component, which is evoked in response to a distracter or novel stimulus, is believed to reflect the orienting of focal attention to such novel or distracting environmental stimuli and exhibits a frontocentral maximum.[37,40,41] Therefore, the P3 components can serve as valuable measures for researchers and clinicians to evaluate multiple aspects of cognition and brain function.

Authors evaluating the persistent effects of concussion on neuroelectric indexes of cognition have observed decreased P3 amplitude[19,20,23] and increased P3 latency,[23] suggesting that concussive injuries may negatively affect attentional resource allocation and the speed of cognitive processing during environmental interactions. Further, this effect appears to endure: deficits have been observed in participants from approximately 3 years[19,23] to more than 34 years after injury.[20]

In addition to the P3 component, recent researchers[19,26,42] have also observed enduring concussion-related deficits in the neuroelectric correlates of conflict monitoring and adaptation during cognitive control performance. These results suggest that in addition to attentional resource allocation, concussive injuries may negatively affect indexes of conflict monitoring and adaptation. One neuroelectric index of conflict is the N2 component, which immediately precedes the P3 component. The frontocentral N2, observed during cognitive control tasks, has been linked to the conscious detection of deviance,[19,43] the mismatch of a stimulus with a mental template, and increased cognitive control over response inhibition.[44] Accordingly, N2 amplitudes are more negative during conditions of greater conflict,[44,45] arising from competition between the execution and inhibition of a single response.[46] Thus, the N2 component can serve as a valuable index of stimulus-response conflict during cognitively demanding environmental transactions.

Accordingly, the goal of our study was to evaluate the chronic influence of concussion on cognitive and brain function using cognitive control tasks, which allowed us to measure neuroelectric function. We used tasks requiring cognitive flexibility, inhibitory control, and working memory. We hypothesized that, relative to uninjured control participants, those with a concussion history would demonstrate deficits in task performance (ie, reaction time and response accuracy) for conditions requiring the upregulation of cognitive control. Further, we predicted that participants with a concussion history would demonstrate a smaller P3 amplitude, reflecting deficits in the allocation of attentional resources during cognitive control operations relative to participants without a concussion history, and a longer P3 latency for those with a concussion history, indicating prolonged delays in the speed of cognitive processing. Last, we predicted that participants with a concussion history would demonstrate increased stimulus-response conflict, as evidenced by greater N2 amplitude relative to controls, during task conditions requiring the upregulation of cognitive control.

METHODS

Participants

Forty adults (14 women, 26 men) between the ages of 20 and 29 years (mean ± standard deviation age = 21.3 ± 2.4

years) were recruited from the east-central Illinois region and the general student body of the University of Illinois. Consistent with previous works,[18,19,26,29,47] we categorized participants based on self-reports of a concussion diagnosis. Each participant was screened for a concussion diagnosed by a medical practitioner (eg, physician, emergency medical technician, nurse). An affirmative answer placed the person into the concussion group. Information about loss of consciousness and posttraumatic amnesia was also collected. To reduce the likelihood that a participant with an undocumented concussion would be placed in the control group, additional questions were asked, including "following a blow to the head, have you experienced any concussion-like symptoms?," and a list of clinical diagnostic symptoms was provided.[5] Participants had to answer *no* and *none*, respectively, to both questions to be placed in the control group. Based on these criteria, 40 participants were enrolled in the study. Specifically, 19 participants were in the concussion group and 21 participants were in the nonconcussed group.

All participants in the concussion group sustained their injuries before age 18 during sport or recreation and were symptom free at the time of testing according to a 21-item symptom scale. Participants also completed a battery of health and demographic questionnaires and were screened for any comorbid conditions, including developmental or learning disorders, neuropsychiatric disorders (anxiety or depression), history of epilepsy, history of brain surgery, or history of alcohol or drug abuse. Other instruments addressed socioeconomic status; life satisfaction; and general, physical, and mental fatigue. Analysis revealed no difference between groups for any of the health or demographic variables (Table). All participants had normal or corrected-to-normal vision and provided written informed consent in accordance with the institutional review board (which approved the study) at the University of Illinois before testing.

Procedures

Participants completed a single testing session that lasted approximately 2.5 hours. They provided informed consent and then filled out the health and demographics screening questionnaires. Next, a trained experimenter administered the Kaufman Brief Intelligence Test (K-BIT; Pearson Education, Inc, San Antonio, TX) to estimate intelligence quotient (Table). Finally, participants were fitted with a 64-channel Quik-Cap (Compumedics Neuroscan, El Paso, TX) and completed a battery of cognitive and perceptual tasks, including a 3-stimulus oddball task, a numeric switch task, and a flanker task. Participants were then briefed on the purpose of the experiment and given remuneration of $15 per hour.

Tasks

Three-Stimulus Oddball Task. During a visual 3-stimulus oddball task, participants responded as quickly and accurately as possible, with a right thumb press only, to randomly occurring, infrequent target stimuli. Target stimuli were 5-cm tall, inverted white triangles that occurred with a probability of 0.12; nontarget stimuli were 5-cm tall, upright white triangles that occurred with a probability of 0.76. In addition to the target and nontarget stimuli, a novel stimulus (black and white checkerboard) occurred with a probability of 0.12. Two counterbalanced blocks of 200 trials were presented focally on a computer monitor at a distance of 1 m. All stimuli were presented on a black background for 100 milliseconds, with a 950-millisecond response window and a 2000-millisecond interstimulus interval.

Switch Task. Participants first completed 2 single-item tasks referred to as *homogeneous task conditions*. Specifically, they were presented with a series of numbers surrounded by a dashed box and asked to determine if the digit presented was odd or even. Participants pressed the left button on the response pad if the number was odd and the right button if the number was even. Next, a different homogeneous task was conducted in which a series of numbers surrounded by a solid box was presented and the participant was asked to determine if the digit presented was greater than or less than the number 5. Participants pressed the left button on the response pad if the number

Table. Demographic Data for Control and Concussion-History Groups[a]

Measure	Group	
	Control	Concussion
Age, y (mean ± SD)	21.1 ± 2.5	21.5 ± 2.4
Participants (men/women)	9/12	5/14
Time since injury, y (mean ± SD)	NA	7.1 ± 4.0
No. of concussions (frequency [%])	NA	1 (14) 2+ (5)
Loss of consciousness (No. [duration])	NA	11 (all <3 min)
Amnesia (no. [duration])	NA	12 (range, 1–3 h)
Kaufmann Brief Intelligence Test (range, 40–60; mean ± SD)	107.5 ± 7.4	104.7 ± 7.5
Education, y (mean ± SD)	14.2 ± 1.9	14.2 ± 1.3
Physical activity, h/wk (mean ± SD)	4.5 ± 1.7	4.1 ± 2.0
Socioeconomic status (range, 1–3; mean ± SD)	2.2 ± 0.5	2.1 ± .6
Hospital Anxiety and Depression Scale: anxiety (range, 0–21; mean ± SD)	7.4 ± 2.2	8.2 ± 3.7
Hospital Anxiety and Depression Scale: depression (range, 0–21; mean ± SD)	5.5 ± 2.2	5.4 ± 1.7
Satisfaction With Life Score (range, 5–35; mean ± SD)	27.2 ± 7.7	27.7 ± 4.6
General fatigue (range, 0–30; mean ± SD)	8.6 ± 2.7	9.4 ± 3.0
Physical fatigue (range, 0–30; mean ± SD)	6.2 ± 2.2	7.1 ± 3.0
Mental fatigue (range, 0–30; mean ± SD)	8.7 ± 3.4	9.6 ± 3.9

Abbreviation: NA indicates not applicable.

[a] No differences were demonstrated between groups for any demographic variable ($P > .05$).

was less than 5 and the right button if the number was greater than 5. The number 5 was not presented in either task. These 2 conditions were counterbalanced across participants.

After the homogeneous task, participants completed a heterogeneous task, which required them to switch randomly between the 2 previously learned rule sets, depending on whether the cue was a solid or dashed box. Performance decrements associated with the heterogeneous tasks relative to the homogeneous tasks are referred to as *global switch costs*, whereas performance decrements associated with switching between rule sets compared with repeating the same rule set during the heterogeneous task are referred to as *local switch costs*. During the heterogeneous task, the 2 rule sets alternated in an equally probable yet random fashion; 7 consecutive trials was the maximum number performed repeatedly for each rule set. Thus, all trials in the heterogeneous blocks were categorized as either switch or nonswitch conditions. For all conditions of the task, white numeric stimuli were presented focally on a black background for 200 milliseconds, with a 1950-millisecond response window and a 2000-millisecond intertrial interval. Participants completed 64 trials in each of the homogeneous conditions and 248 trials in the heterogeneous condition.

Flanker Task. During a modified flanker task,[48,49] participants responded as quickly and accurately as possible to the direction of a centrally presented arrow amid either congruous (eg, <<<<< or >>>>>) or incongruous (eg, <<><< or >><>>) flanking arrows. The incongruent condition requires the concurrent activation of both the correct response (elicited by the target) and the incorrect response (elicited by the flanking stimuli) before stimulus evaluation is complete. Thus, flanker tasks require greater amounts of interference control to inhibit flanking stimuli and provide the correct response.[50] Participants completed 2 blocks of 200 trials and were instructed to respond by making a right thumb press if the central arrow pointed to the right or a left thumb press if the central arrow pointed to the left. Trials were randomized across congruency conditions and presented with equally probable congruency and directionality. The stimuli were white arrows presented focally for 80 milliseconds on a black background with a variable interstimulus interval of 1100, 1300, or 1500 milliseconds and a response window of 1050 milliseconds.

Event-Related Potential Recordings. The electroencephalography activity was recorded from 64 electrode sites (FPz, Fz, FCz, Cz, CPz, Pz, POz, Oz, FP1/2, F7/5/3/1/2/4/6/8, FT7/8, FC3/1/2/4, T7/8, C5/3/1/2/4/6, M1/2, TP7/8, CB1/2, P7/5/3/1/2/4/6/8, PO7/5/3/4/6/8, O1/2) of the International 10–20 system[51] using a Neuroscan Quik-Cap referenced to averaged mastoids (M1, M2). The AFz served as the ground electrode, and impedances were less than 10 kΩ. Additional electrodes were placed above and below the left orbit and on the outer canthus of each eye to monitor electro-oculographic activity with a bipolar recording. Continuous data were digitized at a sampling rate of 500 Hz, amplified 500 times with a DC to 70-Hz filter and a 60-Hz notch filter using a Neuroscan Synamps 1 amplifier. Continuous data were corrected offline for electro-oculographic activity using a spatial Neuroscan filter. Epochs were created from –100 to 1000 milliseconds around the stimulus and baseline corrected using the 100-millisecond prestimulus period. Data were filtered using a zero-phase–shift 30-Hz (24 dB/octave) low-pass filter. Trials with a response error or artifact exceeding 75 μV were rejected. Average ERP waveforms were created for correct trials during the oddball, switch, and flanker tasks. The *P3* component was defined as the largest positive peak occurring within 300–700 milliseconds after stimulus onset. *Amplitude* was defined as the difference between the mean preresponse baseline and peak of maximum amplitude ±15 milliseconds, yielding a peak interval measure. Latency was measured in milliseconds from stimulus onset to maximum peak within the specified window. The *N2* component was defined as the largest negative peak occurring within 150–250 milliseconds after stimulus onset.

Statistical Analysis

Statistical analyses were completed using SPSS (version 19.0; SPSS Inc, Chicago, IL), and statistical significance was defined as $P < .05$. Behavioral data were analyzed using a 1-way analysis of variance (ANOVA) for the 3-stimulus oddball task, a 2 (concussed, nonconcussed) × 2 (congruent, incongruent) repeated-measures ANOVA for the flanker task, and a 2 (group) × 2 (condition: global switch, homogeneous or heterogeneous; local switch, switch or nonswitch) repeated-measures ANOVA for the switch task. All ANOVAs used the Greenhouse-Geisser correction. We used post hoc Bonferroni corrected *t* tests to evaluate significant interactions.

Neuroelectric data analysis was conducted on N2 and P3 component values (ie, amplitude, latency). The N2 component values for each participant were analyzed using electrode site FCz[52] and were submitted to similar factorial models as described earlier. We analyzed the P3 component values for each participant using the 6 midline electrode sites (Fz, FCz, Cz, CPz, Pz, POz) and similar factorial models as described earlier, with the addition of a site factor.

RESULTS

Task Performance

Three-Stimulus Oddball. Analysis of task performance (reaction time, response accuracy) failed to reveal group differences for target, distracter, or nontarget trials: all t_{38} values ≤ 1.3, all P values $\geq .20$.

Switch Task. For the global switch, omnibus analysis for reaction time revealed an effect of condition ($F_{1,38} = 374.05$, $P < .001$, $\eta^2 = .90$), with a longer reaction time for the heterogeneous condition ($\mu = 758.2 \pm 111.5$ milliseconds) than the homogeneous condition ($\mu = 456.8 \pm 63.8$ milliseconds). A condition effect was noted for response accuracy ($F_{1,38} = 13.83$, $P = .001$, $\eta^2 = .26$), with less accuracy for the heterogeneous condition ($\mu = 80.5 \pm 15.7$) relative to the homogeneous condition ($\mu = 89.6 \pm 63.8$). No group effects were apparent for reaction time ($F_{1,38} = 0.128$, $P = .72$, $\eta^2 = .01$) or response accuracy ($F_{1,38} = 0.511$, $P = .48$, $\eta^2 = .01$).

For the local switch, omnibus analysis for response accuracy demonstrated an effect of condition ($F_{1,38} = 53.81$, $P < .001$, $\eta^2 = .58$), with less accuracy for switch trials ($\mu = 76.9 \pm 9.9$) relative to nonswitch trials ($\mu =$

84.0 ± 10.5). Analysis for reaction time failed to reveal a condition effect ($F_{1,38} = 3.38$, $P = .07$, $\eta^2 = .01$) or any group effects for reaction time ($F_{1,38} = 0.10$, $P = .75$, $\eta^2 = .001$) or response accuracy ($F_{1,38} = 0.15$, $P = .69$, $\eta^2 = .01$).

Flanker Task. Omnibus analysis of reaction-time latency revealed an effect of congruency ($F_{1,38} = 20.9$, $P = .001$, $\eta^2 = .35$), indicating that participants had shorter reaction times for congruent ($\mu = 386.5 \pm 36.5$ milliseconds) relative to incongruent ($\mu = 408.5 \pm 47.0$ milliseconds) trials. Response accuracy analyses revealed a main effect of group ($F_{1,38} = 4.03$, $P = .05$, $\varepsilon = .09$; Figure 1), reflecting less accuracy in the participants with a concussion history ($\mu = 82.9 \pm 13.2$) compared with control participants ($\mu = 87.2 \pm 9.4$). Further, we observed a congruency effect ($F_{1,38} = 17.01$, $P < .001$, $\eta^2 = .30$), indicating greater accuracy for congruent ($\mu = 87.76/\sigma = 9.35$) relative to incongruent ($\mu = 84.25/\sigma = 12.23$) trials.

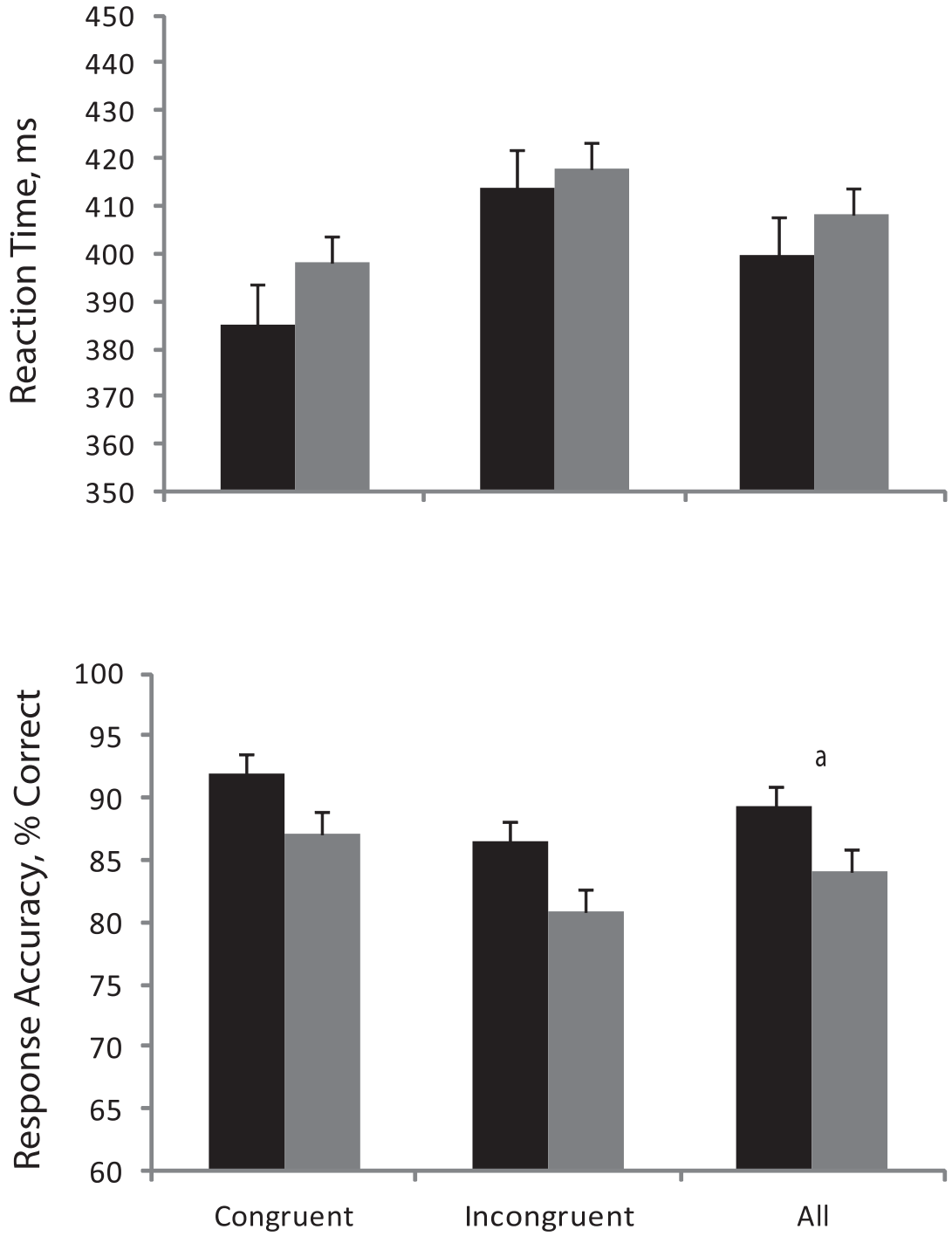

Figure 1. Response time and accuracy for the concussion (black bars) and control (gray bars) groups for all conditions of the flanker task.
[a] Indicates statistical significance ($P < .05$).

The N2 Component

Three-Stimulus Oddball Task. Analysis of N2 amplitude and latency failed to reveal an effect for group or for any condition of the task: all t_{38} values ≤ 2.41, all P values $\geq .16$.

Switch Task: Global Switch. For the global switch, omnibus analysis of N2 amplitude showed no effect for group ($F_{1,38} = .07$, $P = .78$, $\eta^2 = .002$) but did show an effect for condition ($F_{1,38} = 8.16$, $P < .01$, $\eta^2 = .17$). However, this effect was superseded by a group X condition interaction ($F_{1,38} = 7.23$, $P = .01$, $\eta^2 = .16$). Using post hoc comparisons, we attributed the effect to the heterogeneous condition of the task (t_{38}, $P = .002$), indicating that participants with a concussion history exhibited increased N2 amplitude ($\mu = -3.9 \pm 1.9$ μV) compared with control participants ($\mu = -1.8 \pm 2.2$ μV).

Omnibus analysis of N2 latency revealed a main effect for group ($F_{1,38} = 7.10$, $P = .01$, $\eta^2 = .16$), indicating that participants with a concussion history exhibited shorter N2 latency relative to control participants across both conditions of the task. Analysis failed to reveal an effect of condition ($F_{1,38} = .001$, $P = .97$, $\eta^2 = .001$).

Switch Task: Local Switch. Omnibus analysis of N2 amplitude revealed effects for group ($F_{1,38} = 4.58$, $P = .04$, $\eta^2 = .108$) and condition ($F_{1,38} = 4.68$, $P = .04$, $\eta^2 = .11$). However, these effects were superseded by a group X condition interaction ($F_{1,38} = 23.18$, $P < .001$, $\eta^2 = .38$). Post hoc comparisons localized the effect to nonswitch trials within the heterogeneous condition of the task ($t_{38} = 4.615$, $P < .001$), indicating that participants with a concussion history exhibited increased N2 amplitude ($\mu = -3.9 \pm 1.6$) during the nonswitch trials of the task compared with control participants ($\mu = -1.5 \pm 1.6$).

Omnibus analysis of N2 latency failed to reveal a main effect for group ($F_{1,38} = 0.63$, $P = .43$, $\eta^2 = .016$), but a main effect of condition was revealed ($F_{1,38} = 10.70$, $P < .001$, $\eta^2 = .22$). However, this effect was superseded by a group X condition interaction ($F_{1,38} = 18.93$, $P < .001$, $\eta^2 = .33$). Post hoc comparisons revealed that control participants exhibited shorter N2 latency during the nonswitch trials ($t_{38} = 2.70$, $P = .01$), and participants with a concussion history exhibited shorter N2 latency during the switch trials ($t_{38} = 3.12$, $P < .01$).

Flanker Task. Omnibus analyses of N2 amplitude and latency failed to reveal an effect for group ($F_{1,38} < 1.3$, $P > .25$, $\eta^2 \leq .03$) or condition ($F_{1,38} < 0.70$, $P \leq .40$, $\eta^2 \leq .01$).

The P3 Component

3-Stimulus Oddball Task. Omnibus analysis of P3 amplitude revealed a main effect of group during the target condition of the oddball task ($F_{1,38} = 6.14$, $P < .02$, $\eta^2 = .14$; Figure 2), indicating that participants with a concussion history exhibited smaller P3 amplitude during target detection than control participants. A main effect of site for the target condition was noted ($F_{1,38} = 25.47$, $P = .001$, $\eta^2 = .40$). Maximum amplitude occurred at the Pz site. The P3 latency analysis failed to reveal any effects for group ($F_{1,38} = 1.82$, $P = .18$, $\eta^2 = .04$) or site ($F_{1,38} = 2.32$, $P = .08$, $\eta^2 = .40$) during the target condition.

Analysis of the distracter and nontarget conditions failed to reveal an effect of group for amplitude or latency ($F_{1,38}$ values ≤ 2.34, P values $\geq .13$, η^2 values $\leq .32$). However, P3 amplitude for the distracter condition revealed an effect for site ($F_{1,38} = 9.38$, $P = .001$, $\eta^2 = .20$). Maximum amplitude occurred at the Cz site. The P3 latency failed to reveal an effect of site during the distracter condition ($F_{1,38} = 2.69$, $P = .09$, $\eta^2 = .06$).

Flanker Task. Omnibus analysis of P3 amplitude revealed that all participants displayed greater amplitudes during the incongruent relative to the congruent trials ($t_{39} = 2.58$, $P = .01$). A main effect was noted for site ($F_{1,38} = 25.11$, $P = .001$, $\eta^2 = .39$), indicating that maximum amplitude occurred at the CPz site. Amplitude analysis failed to reveal a main effect for group ($F_{1,38} = 0.76$, $P = .38$, $\eta^2 = .13$).

Analysis of P3 latency revealed a main effect for congruency ($F_{1,38} = 15.26$, $P < .001$, $\eta^2 = .29$), indicating that all participants had shorter latency to congruent relative to incongruent stimuli. A main effect of site was seen ($F_{1,38} = 9.23$, $P < .001$, $\eta^2 = .19$), with the POz site exhibiting the shortest latency. No main effect for group was evident ($F_{1,38} = .004$, $P = .95$, $\eta^2 = .01$).

Switch Task: Global Switch. Omnibus analysis of P3 amplitude revealed a main effect for group ($F_{1,38} = 24.87$, $P = .001$, $\eta^2 = .39$; Figure 3), indicating that participants with a history of concussion exhibited decreased P3 amplitude across task conditions compared with control participants. A main effect for site was demonstrated ($F_{1,38} = 24.87$, $P < .001$, $\eta^2 = .39$). However, this effect was superseded by a condition X site interaction ($F_{1,38} = 12.32$, $P = .001$, $\eta^2 = .24$). Post hoc comparisons revealed effects at the Pz and POz sites (t_{38} values ≥ 4.72, $P \leq .003$), indicating that all participants demonstrated larger amplitudes during the heterogeneous condition than the homogeneous condition at these sites.

Analysis of P3 latency failed to reveal main effects for group, condition, or site ($F_{1,38}$ values ≤ 1.80, P values $\geq .36$, η^2 values $\leq .04$).

Switch Task: Local Switch. Omnibus analysis of P3 amplitude revealed a main effect of group ($F_{1,38} = 6.18$, $P = .01$, $\eta^2 = .14$). However, this effect was superseded by a group X condition interaction ($F_{1,38} = 6.61$, $P = .01$, $\eta^2 = .15$). Post hoc comparisons localized the effect to the switch condition ($t_{38} = 2.70$, $P = .02$), indicating that participants with a concussion history exhibited decreased P3 amplitude during switch trials compared with control participants. No such effect was observed for the nonswitch trials ($t_{38} = 2.15$, $P = .08$). Analysis further revealed a main effect for condition ($F_{1,38} = 19.71$, $P < .001$, $\eta^2 = .34$), indicating that all participants exhibited greater amplitude during the switch relative to the nonswitch condition. Analysis also revealed a main effect for site ($F_{1,38} = 9.38$, $P < .001$, $\eta^2 = .19$). However, this effect was superseded by a condition X site interaction ($F_{1,38} = 6.63$, $P < .01$, $\eta^2 = .15$). Post hoc comparisons revealed differences at the Cz, CPz, Pz, and POz sites (t_{38} values ≥ 3.72, P values $\leq .004$), indicating that all participants exhibited decreased P3 amplitude in the nonswitch relative to the switch condition.

Analysis of P3 latency failed to reveal main effects for group, condition, or site ($F_{1,38}$ values $\leq .92$, P values $\geq .33$, η^2 values $\leq .03$).

DISCUSSION

Our results suggest that sport-related concussion sustained during early life can have prolonged negative

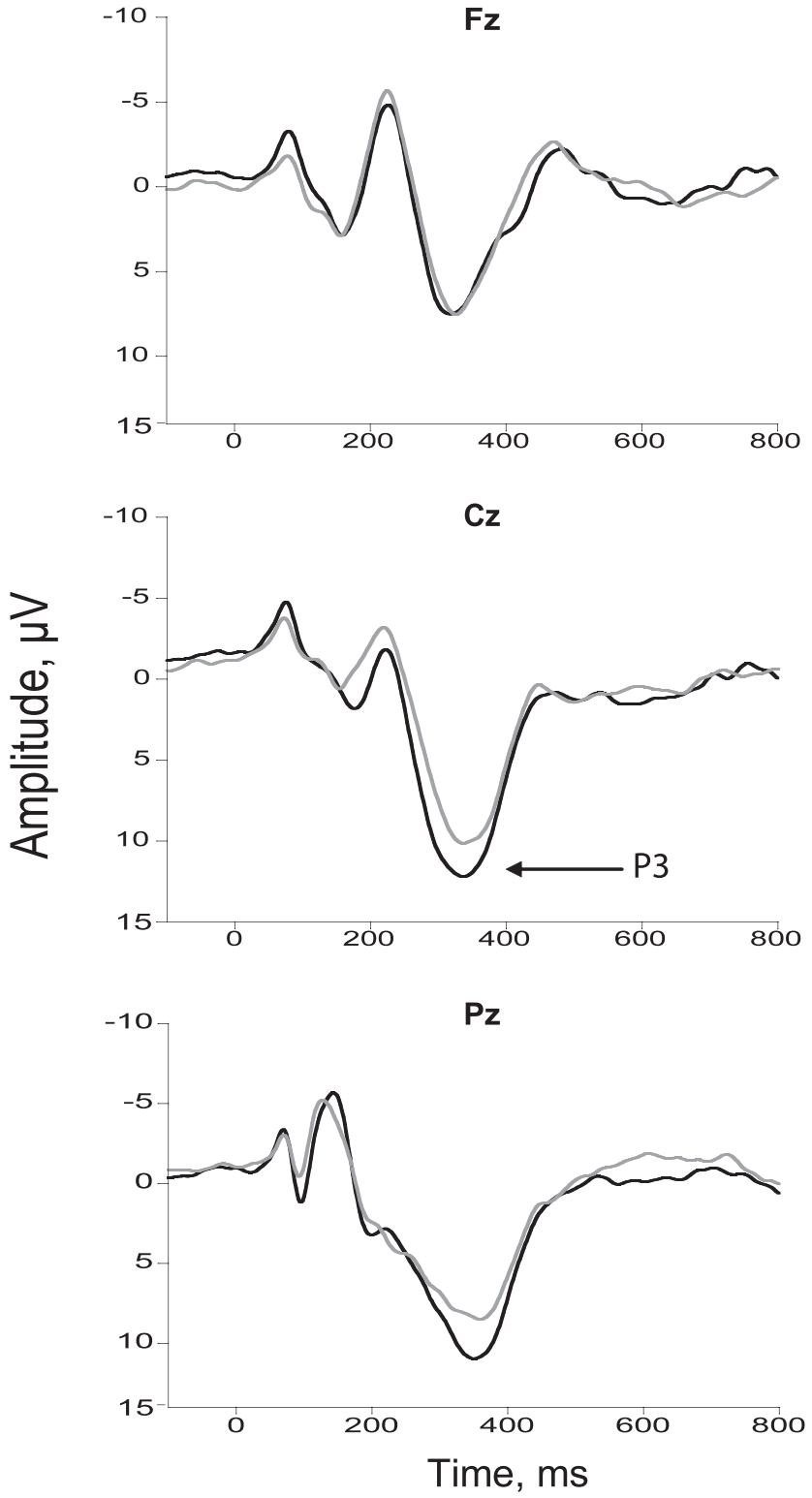

Figure 2. The P3 amplitudes for the concussion (gray lines) and control (black lines) groups at the Fz, Cz, and Pz sites for the target trials of the oddball task.

consequences for brain and cognitive function. Previously concussed young adults, an average of 7.2 years from injury, displayed overt cognitive and covert neural deficits relative to healthy and demographically equivalent control participants. Replicating previous findings,[20,26] participants in the concussion-history group exhibited chronic deficits in cognitive control, as shown by less accuracy on the flanker task, which requires the modulation of inhibitory control. Additionally, neuroelectric measures taken during the oddball and switch tasks demonstrated covert differences between participants with a history of concussion and control participants, implicating attention, conflict monitoring, and mental flexibility as target cognitive processes that may be especially vulnerable after concussion.

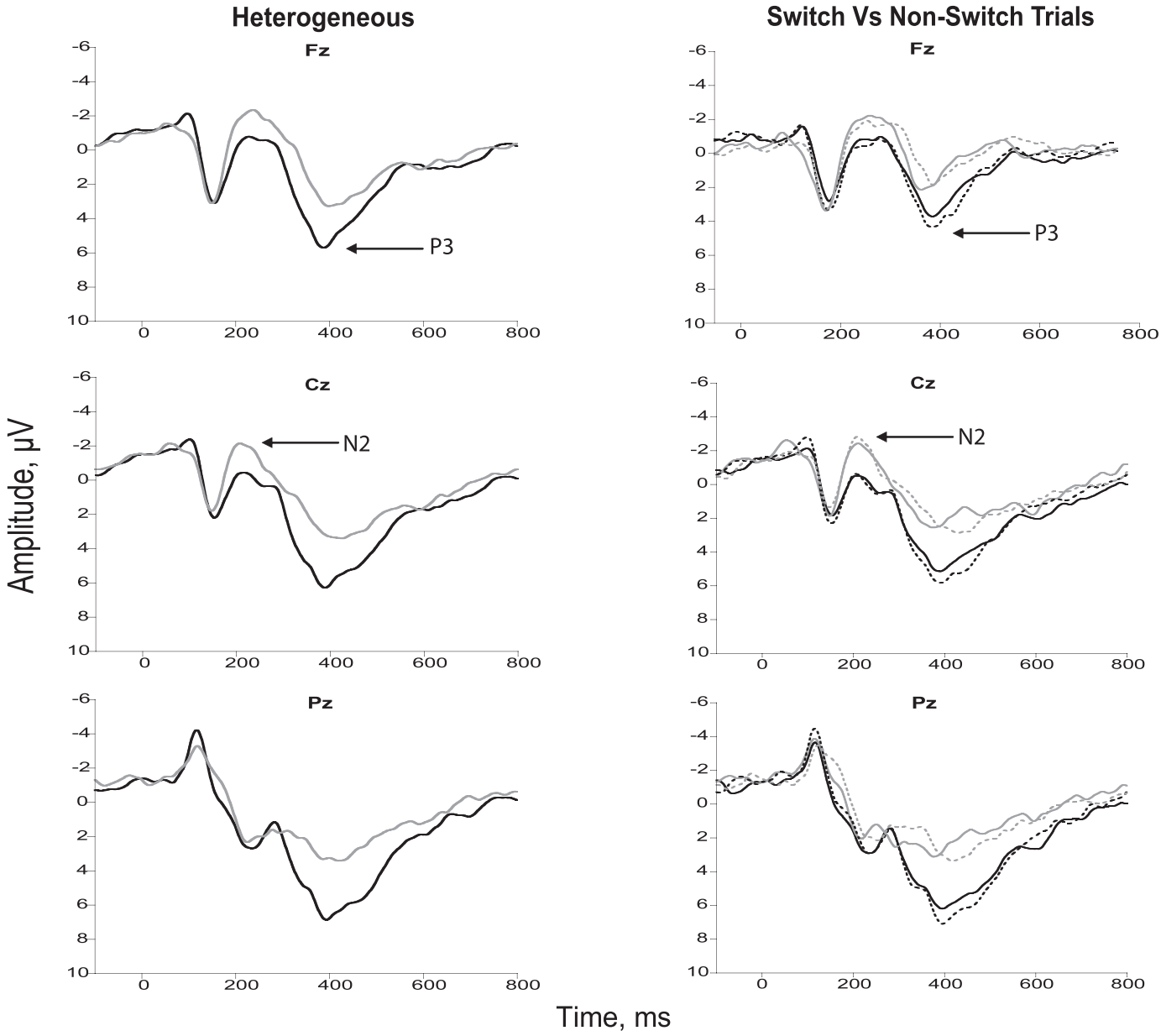

Figure 3. The N2 and P3 amplitudes for the concussion (gray lines) and control (black lines) groups at the Fz, FCz, Cz, CPz, and Pz sites for the heterogeneous condition of the switch task. Nonswitch (solid lines) and switch (dashed lines) refer to the various conditions of the heterogeneous task.

Collectively, the data support the use of cognitive control measures to better understand long-term deficits in cognition and brain health as a function of concussion.

Cognitive control relies on a distributed network of neural regions, including the dorsal and anterior cingulate cortex, dorsal and ventrolateral prefrontal cortex, superior parietal cortex (including the premotor and primary motor cortex), inferior temporal gyrus, and superior parietal lobule.[53] Thus, efficient cognitive control depends on successful intercortical and interhemispheric integration.[54] Concussive injuries have been associated with diffuse axonal injury and compromised white-matter integrity[55–58] and so may disrupt both the efficiency and speed of transmission between cortical areas. Recent research[57] indicates that the most frequently damaged white-matter structures in concussed persons are those subserving memory and cognitive control. Accordingly, investigators have observed persistent concussion-related deficits across various aspects of cognitive control, including prospective and working memory,[18,28,59] mental flexibility,[22] and interference and inhibition.[20,22,26] Thus, cognitive control functions and their neural generators appear particularly sensitive to the effects of sport-related concussion.

In our study, participants with a concussion history showed deficits in cognitive control, as reflected by decreased response accuracy during a flanker task. Flanker tasks require inhibitory control to filter irrelevant (and interfering) flanking stimuli and overcome proponent response tendencies to execute a correct response.[50] Previous authors evaluating formerly concussed persons have also witnessed less response accuracy relative to controls during flanker tasks. For example, Pontifex et al[26]

observed decreased response accuracy in previously concussed persons 3 years after injury, and de Beaumont et al[20] noted concussion-related deficits in flanker accuracy more than 3 decades after injury. In tandem with our results, these studies suggest that concussion can chronically impair the cognitive control processes underlying successful flanker performance. Beyond flanker tasks, evidence of concussion-related deficits in interference control and inhibition is beginning to accumulate. For example, Ellemberg et al[22] demonstrated concussion-related deficits during a Stroop color-word task in female athletes 6 months after injury. Recent evaluations using transcranial magnetic stimulation and electromyography have revealed concussion-related deficits in intercortical inhibition persisting for years[27,60] to decades[21] after injury, providing convergent evidence that concussive injuries can chronically compromise a person's inhibitory control.

Because the ability to reliably inhibit irrelevant information and actions is central to the voluntary control of behavior,[61,62] concussion-related deficits in inhibitory control may have far-reaching consequences. For example, concussion-related deficits in vocational and academic performance,[7,8,10] as well as the increased prevalence of neurobehavioral disorders associated with concussion,[63,64] may stem in part from deficits in inhibitory control. Recent research[65,66] implicates the inferior frontal cortex and the frontosubthalamic circuit as the critical neural architecture underlying inhibitory and interference control, regardless of the aspect of the stimulus-response interaction (ie, cognitive or behavioral inhibition, reactive or proactive interference). Supporting this assertion are multiple studies[67–70] evaluating patients with damage or dysfunction to the inferior frontal cortex and the frontosubthalamic circuit. Patients in these studies consistently demonstrated enduring behavioral and neurologic deficits during environmental and task conditions requiring inhibition and interference control. Given this evidence, it is plausible that the structural or functional integrity in 1 or more of these areas is also compromised in persons with less severe forms of traumatic brain injury. Future neuroimaging research is well positioned to evaluate the relationship of mild traumatic brain injury and the structural and functional integrity of areas supporting inhibitory and interference control.

In addition to overt cognitive deficits, persons with a history of concussion also displayed covert neuroelectric deficits. These neuroelectric deficits were evidenced by reduced P3 amplitude and increased N2 amplitude relative to control participants. More specifically, persons with a history of concussion showed reduced P3 amplitude relative to control participants during the oddball and switch tasks. Previous researchers[19,20,23,26,71] have observed persistent concussion-related deficits in P3 latency and amplitude across multiple sensory modalities and oddball paradigms. Thus, our study replicated previous findings of persistent neuroelectric deficits in persons with a history of concussion during oddball tasks. Specifically, persons who were more than 7 years after injury demonstrated decreased P3b amplitude relative to control participants during target presentation, adding to the body of evidence indicating that concussive injuries can lead to persistent deficits in attentional resource allocation during stimulus acquisition.

However, we found no group differences for P3a amplitude or latency. Such results are contrary to those of previous sport-related concussion studies that showed reductions in P3a amplitude,[26,36] suggesting that differences in study paradigms may explain the discrepant results. For example, previous authors used 3-stimulus paradigms that included novelty distracters, whereas we used an alerting distracter. These 2 types of distracters elicit different neuroelectric profiles (ie, scalp topographies), which may be differentially affected by clinical factors.[41] Therefore, the more frontally mediated[72,73] novelty P3a may be more sensitive to the effects of concussion than the more centrally mediated[37,41] alerting P3a. It is also possible that our findings simply reflect the heterogeneous nature of concussion injuries, particularly given the diverse manifestations of P3 deficits observed in previous concussion research. Regardless, our results further demonstrate the persistent negative effect of concussive injuries on neuroelectric function during attentional paradigms such as oddball tasks.

Our formerly concussed participants also displayed deficits in neuroelectric function during a switch task. To our knowledge, we are the first to demonstrate persistent concussion-related deficits in neuroelectric function using this task, extending the current body of knowledge by showing the sensitivity of mental flexibility paradigms to sport-related concussion. Specifically, we observed deficits in attentional resource allocation, as evidenced by smaller P3 amplitude, and increased stimulus-response conflict, as evidenced by increased N2 amplitude, during the heterogeneous condition of the task. Relative to the homogeneous conditions, which require control of a single rule set, heterogeneous or mixed rule sets conditions require sustained attention and cognitive control[74] to reliably execute a correct response while holding multiple rule sets in the contents of working memory. Relative to homogeneous conditions, heterogeneous conditions also require greater working memory to maintain multiple rule sets in a state of readiness and to track stimuli sequences.[75] In the current study, the P3 deficits associated with formerly concussed persons were further localized to the switch trials within the heterogeneous condition. Switch trials require both reactive and proactive interference control[76] and inhibition[77] to manage competing stimulus-response mappings[78] associated with multiple rule sets. Thus, concussion appears to negatively relate to a person's attentional processing when environmental conditions require increased working memory, proactive or reactive interference, and inhibitory control.

Although the pathophysiology underlying the neuroelectric deficits in attention is uncertain, the P3 component is believed to reflect the activation and circuit integrity in the frontal and temporoparietal areas.[40,79] White-matter tracts subserving attention and memory have been demonstrated to be particularly susceptible to damage and dysfunction after concussion.[55,57,58] Further, reduced functional connectivity in concussed persons has been observed during attention and working memory paradigms.[80,81] In addition, recent functional magnetic resonance imaging[82] demonstrated activation deficits during performance of an oddball task in the dorsolateral prefrontal cortex and superior parietal lobe in formerly concussed persons. Hence, concussive injuries may disrupt the structural or functional

integrity of areas responsible for P3 generation. As such, further research examining the causes of concussion-related P3 deficits is warranted.

In addition, participants with a history of concussion also evidenced increased stimulus-response conflict as reflected by greater N2 amplitude during the switch task. Thus, the current results add to a growing body of research[19,26,42] detailing deficits in conflict monitoring and adaptation after concussion. These deficits were further localized to the nonswitch trials of the task. Closer inspection of the data revealed that participants with a history of concussion had equivalent N2 amplitudes across both nonswitch and switch trials. This pattern of task switching is similar to that observed in older adults[83] and suggests that persons with a concussion history experience an indiscriminate and generalized increase in stimulus-response conflict irrespective of trial demands. Analysis of N2 latency revealed that, relative to control participants, formerly concussed persons displayed a shorter latency that did not vary across heterogeneous task conditions. However, further analysis revealed that although formerly concussed persons had shorter N2 latency during switch trials, they exhibited increased N2 latency during nonswitch trials relative to controls. Such a pattern of results was unexpected and demonstrates the need for further investigation to clarify the relationship of concussive injuries to the processes subsumed by the N2.

Lastly, it should be noted that, contrary to our predictions, no group differences in P3 or N2 amplitude were observed during the flanker task. This may be explained by task settings, as the flanker task used an unusually brief stimulus duration. This 80-millisecond duration elicited overt behavioral findings but potentially at the cost of covert neuroelectric findings. Brief stimulus presentation times can dampen ERP amplitude,[41] potentially obviating neuroelectric differences during the flanker task. Similarly, the relatively long presentation time (200 milliseconds) and intertrial interval (2000 milliseconds) used during the switch task engendered covert but not overt differences. Given the current pattern of results, future researchers should diligently examine task settings because they may influence the ability to detect subtle yet persistent neurocognitive deficits.

CONCLUSIONS

Our findings add to a growing body of research detailing the persistent deficits in brain and cognitive function stemming from sport-related concussion. They suggest that concussive injuries can disrupt fundamental elements of higher-order neurocognition by chronically impairing attention, working memory, inhibition, and interference control. Further, the observed neuroelectric deficits in attention and conflict monitoring emerged only when cognitive demands increased (ie, during conditions requiring the upregulation of interference and inhibitory control). This is a key point, as subtle yet persistent neurocognitive deficits stemming from concussion may go unrecognized in the absence of highly sensitive tasks and neuroimaging tools.

Although the findings documented here are subtle in nature, they may have implications for athletic performance and clinical practice. As previously noted (Duncan et al,[43] Sloubonov et al[84]), ERPs may prove useful for identifying, targeting, and evaluating interventions in persons with atypical recovery rates. Further, the superior temporal specificity of ERPs relative to other neuroimaging measures and overt behavioral responses elicited from clinical testing may enable us to better understand which aspects of neurocognition are affected by concussion in a noninvasive and increasingly affordable manner. Specific to current and previous results, cognitive tasks targeting attention allocation (P3) and response inhibition (N2) may prove beneficial for remediation or slowing the proposed cognitive declines that may be associated with the injury.

Some degree of caution, however, should be taken when interpreting the current results because of methodologic considerations such as the cross-sectional design and reliance upon self-reported physician diagnosis of concussion. Although not ideal, our method of screening for concussion has been successfully used in laboratory[19,26] and epidemiologic studies,[29,47] and strong agreement has been observed between self-reported medical diagnosis of concussion and medical record documentation in college-aged athletes.[85] Unfortunately, reliance upon self-reported concussion for injuries sustained before college in college-age participants is common.[19,21,26,29,47,60] Future researchers will benefit from record access, baseline testing, and longitudinal design. In addition, given the cross-sectional design of the current study and the time elapsed between injuries and testing, it is possible that some preexisting difference or unmeasured variable contributed to the observed differences in brain and cognitive function; however, given the demographic equivalence between groups, this is unlikely. Despite these methodologic shortcomings, the current findings help to further our understanding of the nature, breadth, and duration of neurocognitive deficits stemming from concussion.

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