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Sport-Related Concussion and Sensory Function in Young Adults

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Context: The long-term implications of concussive injuries for brain and cognitive health represent a growing concern in the public consciousness. As such, identifying measures sensitive to the subtle yet persistent effects of concussive injuries is warranted.

Objective: To investigate how concussion sustained early in life influences visual processing in young adults. We predicted that young adults with a history of concussion would show decreased sensory processing, as noted by a reduction in P1 event-related potential component amplitude.

Design: Cross-sectional study.

Setting: Research laboratory.

Patients or Other Participants: Thirty-six adults (18 with a history of concussion, 18 controls) between the ages of 20 and 28 years completed a pattern-reversal visual evoked potential task while event-related potentials were recorded.

Main Outcome Measure(s): The groups did not differ in any demographic variables (all P values $> .05$), yet those with a concussive history exhibited reduced P1 amplitude compared with the control participants ($P = .05$).

Conclusions: These results suggest that concussion history has a negative effect on visual processing in young adults. Further, upper-level neurocognitive deficits associated with concussion may, in part, result from less efficient downstream sensory capture.

Key Words: mild traumatic brain injuries, visual processing, event-related potentials, pattern-reversal visual evoked potentials

Key Points

- Visual processing and higher-level cognitive function were affected by concussion over the long term.
- The potential contributions of low-level sensory deficits to higher-order neurocognitive dysfunction after concussion should be studied.
- Event-related potentials have greater sensitivity than standard clinical tools and have the potential for clinical use.

The long-term and cumulative effects of concussive injuries represent a growing concern in the public consciousness. *Concussion* has been defined as "a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces."[1,2] Estimated incidence rates for this condition, described as a "silent epidemic" by the Centers for Disease Control and Prevention,[2–5] range from a conservative 300 000 per year[4–6] to a more liberal and recent estimate of 3.8 million cases in the United States annually.[7] Because 15% to 20% of these injuries result from sport participation,[8] sport-related concussion represents an increasing concern, not only in the public domain but also in clinical and research settings.

Based on clinical evaluations, concussed persons typically return to their preinjury level of functioning within 7 to 10 days of injury,[9,10] a time paralleling the acute neurometabolic cascade associated with concussion.[11,12] Indeed, several investigations[2,13–15] of young adult athletes with a concussion history indicate normal performance on a variety of clinical tests after the acute injury stage. However, more recent studies using highly sensitive assessment measures suggest that a multitude of chronic nervous system dysfunctions and cognitive deficits stem from concussive injuries.[16–28] Thus, the chronic, subclinical effects of concussion remain unclear, and measures sensitive to subtle and persistent deficits stemming from concussion are needed.

Electroencephalography, which records brain activity from electrodes placed on the scalp, has been extensively used to examine neuroelectric activity in normal and clinical populations for almost a century. More recently, event-related potentials (ERPs; patterns of neuroelectric activity that occur in preparation for or in response to an event) have emerged as a technique to provide insight into the neural processes underlying perception, memory, and action.[29] The ERPs may be obligatory responses (exogenous) to stimuli in the environment or may reflect higher-order cognitive processes (endogenous) that often require active consideration by a person.[30]

Over the past decade, electroencephalography and ERPs in particular have demonstrated the requisite sensitivity to detect subtle, covert deficits in neurocognitive function associated with concussion[17,23,27,31–33] (for review, see Broglio et al[29]). Although several groups have evaluated ERP components, such as the ERN, N2, and P3, to examine attention, perception, and memory, few authors[23,34–36] have evaluated the effects of concussion on neuroelectric indexes of sensory function. In particular, only 3 studies have evaluated the influence of concussion on visual-evoked

potentials (VEPs).[23,34,35] Findings from these studies suggest that for a significant portion of people, concussion may lead to chronic impairment in the neuroelectric correlates of visual processing.

Believed to reflect the functional integrity of the visual system, VEPs are electrophysiologic signals passively evoked in response to visual stimuli that demonstrate a parietal-occipital maximum.[37–40] Efficient visual processing and sensory integration are essential to day-to-day functioning[34,41]; however, the visual system of a concussed individual is typically unevaluated.[34] Thus, VEPs represent an underused and potentially valuable tool for evaluating and understanding sensory and nervous system dysfunction after injury.

One VEP paradigm of particular utility is the pattern-reversal task (PR-VEP). This task uses an inverting patterned stimulus to evoke an electrocortical waveform, which is characterized by a negative deflection at about 75 milliseconds (N75), followed by a positive deflection at about 100 milliseconds (P1).[42] The PR-VEP task is a standard in clinical research assessing central nervous system function[43] because of the high sensitivity, specificity, and intraindividual stability of PR-VEPs relative to VEPs elicited by other paradigms.[44,45] Specifically, the P1 elicited by this paradigm is less variable than the P1 components elicited by other paradigms, making it preferable for evaluating clinical populations.[40] For example, Sarnthein et al[45] observed a test-retest sensitivity of 95% and specificity of 99.7% for P1 component values. Further, Mellow et al,[46] evaluating binocular reproducibility in a single participant, observed test-retest coefficients of variation of 9% to 14% for P1 amplitude and 1% to 2% for P1 latency.

The P1 component is an exogenous or obligatory potential and is the first positive-going deflection after stimulus presentation (or inversion). The P1 is thought to reflect sensory processes such as gating, amplification, and preferential attention to sensory inputs.[38,47] Within the context of the PR-VEP paradigm, the P1 is believed to index the functioning of the geniculostriatal pathway,[39] which is thought to mediate visual processing. The P1 component values can provide important information to researchers and clinicians: reduced P1 amplitude may indicate neuronal atrophy,[48] and increased P1 latency may indicate slowed neural conduction within the visual pathways.[49]

To our knowledge, only one set of authors[23] has evaluated the P1 component in relation to sport-induced concussion by using a pattern-reversal task to elicit VEPs in young and middle-aged adults. Approximately one-third of the participants who reported a concussion history evidenced P1 deficits, as determined by clinical diagnostic criteria. Such findings suggest that concussion may negatively influence the P1 component in a subset of persons, but further investigation is warranted to clarify the nature of the relationship between concussive injuries and the P1 VEP. Accordingly, the purpose of our investigation was to assess the relationship of sport-related concussion on visual processing using a pattern-reversal paradigm.

METHODS

Participants

Thirty-eight adults (14 women, 24 men) between the ages of 20 and 29 years (21.3 ± 2.4 years) were recruited from the east-central Illinois region and the general student body of the University of Illinois at Urbana-Champaign. All participants were affiliated with the university and currently engaged in club-level or recreational athletics. All participants in the concussion group had sustained their injuries before the age of 18 years during sport and recreation and were symptom free at the time of testing. Consistent with previous works,[16,17,27,50,51] participants were categorized based on self-report of a medical diagnosis of concussion. In addition, information about injury characteristics, including the presence and duration of loss of consciousness and posttraumatic amnesia, was collected. To reduce the likelihood that a person with an undocumented concussion would be placed in the control group, an additional question asked "following a blow to the head, have you experienced any of the following symptoms?" with a list of common concussion symptoms used in clinical diagnostic interviews provided.[52] A participant had to answer *no* to both questions to be placed in the control group. Based on these criteria, 38 persons were enrolled in the study: 19 participants in the concussion group and 19 in the nonconcussed group. Participants also completed a battery of health and demographic questionnaires and were screened for any comorbid conditions. Exclusion criteria were history of a developmental or learning disorder, neuropsychiatric disorder, epilepsy, brain surgery, or alcohol or drug abuse or use of psychotropic medication. All participants had normal or corrected-to-normal vision and provided written informed consent in accordance with the university's institutional review board before testing. Demographic information is available in the Table.

Procedures

Each participant completed a single testing session in which he or she provided informed consent, underwent a preliminary medical screening, and was fitted with a 64-channel Quik-Cap (Compumedics Neuroscan, Charlotte, NC). The person then completed a pattern-reversal task as part of a larger battery of tasks. Upon completion of all tasks, the participant was briefed on the purpose of the experiment and was given remuneration of $15 per hour.

Event-Related Potential Recording and Reduction

The electroencephalographic activity was recorded from 64 silver/silver chloride electrode sites of the international 10–10 system.[53] Ongoing electroencephalographic activity

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

Measure	Group	
	Control	Concussion
Age, y (mean ± SD)	21.5 ± 2.8	21.6 ± 2.6
Participants (men/women)	11/7	13/5
Years since injury	NA	6.7 ± 3.9
Loss of consciousness (No. [duration]}	NA	7 (∼1–3 min)
Amnesia (No. [duration])	NA	11 (1–3 h)
Education, y	14.2 ± 1.9	14.2 ± 1.3
Socioeconomic status (range = 1–3; mean ± SD)	2.1 ± 0.5	2.1 ± 0.6

Abbreviation: NA indicates not applicable.

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

was referenced to averaged mastoids (M1, M2), with AFz serving as the ground electrode. All 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 direct current to 70-Hz filter and a 60-Hz notch filter using an amplifier (model Synamps; Compumedics Neuroscan). Continuous data were corrected offline for electro-oculographic activity using a spatial filter (model 2003; Compumedics Neuroscan) to perform a principle component analysis to determine the major components that characterized the electro-oculographic artifact among all channels and then reconstruct the original channels without the artifact. Epochs were created from –100 to 250 milliseconds and were baseline corrected using the 100-millisecond preresponse period. Data were filtered using a zero-phase–shift 1-Hz (24 dB/octave) to 12-Hz (24 dB/octave) band-pass filter. Trials exceeding ±75 μV were considered artifact and rejected. However, after we filtered and inspected the data, we retained all trials for averaging.

The *P1 component* was defined as the largest positive-going peak occurring at 50–150 milliseconds after stimulus inversion. *Amplitude* was measured as the difference between the mean preresponse baseline and peak of maximum amplitude ±15 milliseconds, yielding a peak interval measure. Peak interval measures allow a mean amplitude window to be fixed around the peak, affording better characterization of the data than either a peak or area measure alone. *Latency* was measured in milliseconds from stimulus onset to maximum peak within the specified window. Two participants (1 from each group) were identified as outliers (3+ SDs) and excluded, leaving a total of 36 participants (18 per group) for grand averaging.

Stimuli

The stimuli were presented using a computer (model Optiplex 960; Dell Inc, Round Robin, TX) and were viewed on a 12-×15-in (30.48-×38.1-cm) monitor (model SyncMaster 915; Samsung, Seoul, South Korea) with a 60-Hz refresh rate and a screen resolution of 800×600 pixels. The task was generated through the perceptual-contrast program of the Stim2 system (Compumedics Neuroscan). The program presented a high-contrast (50 cd/m^2, luminance = 98%), 2-color (black, white) checkerboard, which reversed the spatial position of the image at a rate of 1 Hz or 2 cycles per second and maintained a fixed illumination during inversions. The checkerboard was oriented horizontally and composed of 6 rows and 6 columns, for a total of 36 checks.

Participants sat in a light, sound, and electrically attenuated chamber approximately 3.28 ft (1 m) from the monitor and were instructed to maintain stable binocular fixation on a cross located in the center of the screen. Stimuli were projected in a rectangular field at a visual angle of 20.1°. Each participant viewed 128 trials (ie, 64 sweeps). For more information regarding standards and procedures for visual electrophysiology in clinical research, see Odom et al.[40]

Statistical Analysis

The VEP component values for each participant were analyzed using an independent-samples *t* test comparing the P1 component values at site Oz. In addition, bivariate correlations were conducted to identify any relationship between P1 component values, time since injury, number of injuries, and loss of consciousness.

RESULTS

The P1 ERP

Independent-samples *t* tests revealed a main effect for group ($t_{34} = 2.01$, $P = .05$), indicating that participants with a history of concussion had smaller P1 amplitudes (2.19 ± 1.6 μv) than control participants (3.18 ± 1.2 μv; Figure). Analysis of P1 latency failed to reveal an effect of group ($t_{34} = 0.58$, $P = .57$).

Correlations

No significant relationship was observed between P1 amplitude and time from last injury ($R = –0.03$, $P = .92$) or the number of injuries incurred ($R = 0.20$, $P = .41$). No relationship was noted between P1 latency and time from last injury ($R = 0.34$, $P = .17$) or the number of injuries incurred ($R = –0.24$, $P = .35$). Lastly, analysis also failed to reveal any relationship between loss of consciousness and P1 amplitude ($R = 0.33$, $P = .18$) or P1 latency ($R = 0.06$, $P = .81$).

DISCUSSION

Our results suggest that sport-related concussion sustained during early life may have long-term negative consequences on visual processing. Previously concussed young adults, who were an average of 6.7 years from their last injury, demonstrated reduced P1 amplitude. Interestingly, this reduction in amplitude was not related to time since injury or the number of injuries. Future authors using causal designs will be better positioned to evaluate if a single concussive injury is sufficient to produce subtle yet enduring deficits in visual processing. The current P1 finding furthers our understanding of the relationship between concussion and visual processing and adds to the extant body of knowledge on concussion and neuroelectric function. It also adds to the findings of previous research,[23,35] which suggest a neuroelectric basis for concussion-related deficits in visual processing and perception.[11,22,41] Thus, converging evidence indicates that concussive injuries not only have a negative relationship with higher-order neurocognitive functioning[18,23,27,31] but also with lower-level sensory and perceptual processing as well.[11,22,23,35,36,41]

Given the increasing incidence of concussions among athletes and military personnel and the resulting societal effects, it is important to understand the specific deficits and time course associated with concussion. Our results have clear human performance implications in athletic and military settings, where sensory and perceptual integrity is critical for successful environmental transactions and avoiding injury. However, the implications are also broader in that there may be a lower-level sensory contribution to the higher-order neurocognitive deficits associated with

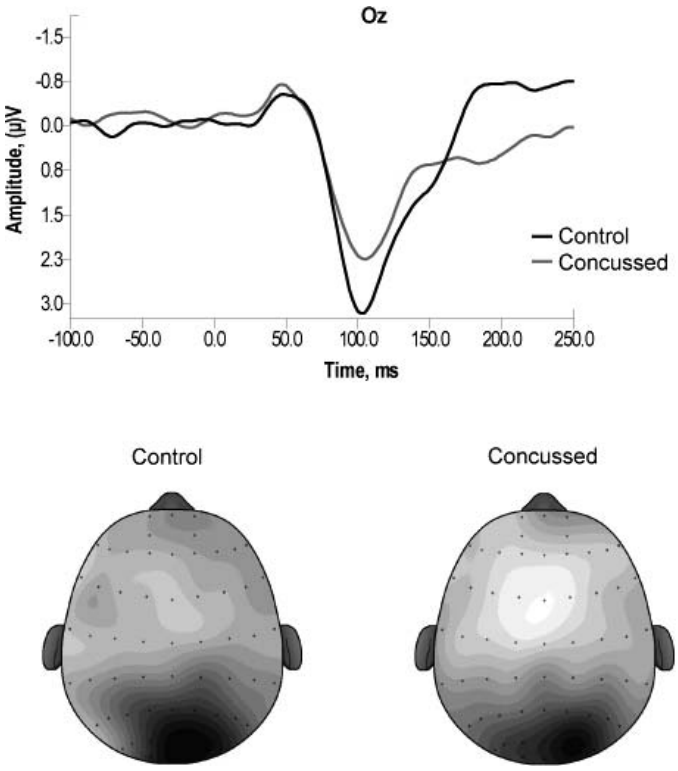

Figure. The P1 amplitudes in concussed and control participants.

concussion. Thus, higher-level neurocognitive deficits associated with concussion may be, in part, a result of less effective sensory capture. This is probably most applicable to deficits observed during environmental transactions requiring high levels of sensory discrimination or inhibition.

As all participants in the current study were also involved in a larger study assessing higher-level neurocognitive deficits stemming from concussion, we decided to further investigate this hypothesis by exploring the relationship between P1 component values and neuroelectric and behavioral performance during a task requiring sensory-mediated response inhibition (Erickson flanker task). We found that P1 amplitude and the neuroelectric correlates of attention (P3 amplitude and latency) were significantly related for the control group but not for formerly concussed participants. Furthermore, for formerly concussed persons, P1 amplitude was inversely related to the number of errors of commission: those committing the greatest number of errors during the flanker task demonstrated the lowest P1 amplitudes. This effect was not observed in control participants.

Together, these results suggest that less effective sensory capture may have contributed to the higher-level deficits in attentional resource allocation and inhibition observed in these people.[54] Thus, persistent, low-level deficits in sensory and perception function may be contributing to the increasingly robust findings of degraded attention allocation[17–19,23,33,54] and inhibitory control[19,27,54] in those with a concussion history. However, future research using factorial designs is needed to further describe this relationship.

The current results add to but are in partial contrast to those obtained by Gaetz and Weinberg.[23] Several factors may have contributed to the disparity in results across studies, including binocular versus monocular testing, different stimulus inversion rates, and different experimental group sizes. Further, we used traditional research-based statistics to assess our data, whereas Gaetz and Weinberg[23] used clinical diagnostic criteria of ± 2.5 standard deviations. Future researchers should be diligent in addressing such methodologic issues to clarify differences across studies.

CONCLUSIONS

Our findings add to a growing body of research on long-term deficits stemming from sport-related concussion and suggest that visual processing and higher-level cognitive function are vulnerable to injury. The current results also provide additional support for the use of VEP and ERP paradigms in concussion research and an impetus for further investigation of the potential contribution of low-level sensory deficits to higher-order neurocognitive dysfunction. Furthermore, although ERPs have primarily been used in research settings to identify specific impairments after traumatic brain injury, they can be used by clinicians to better understand injury severity, evaluate cognitive training gains, and contribute to novel rehabilitative approaches.[55] Future investigators using multimodal paradigms will help elucidate the nature, breadth, and duration of neurocognitive deficits stemming from concussive injuries.

Finally, it should be noted that our study was not without limitations. First, it is possible that some unobserved variable or preexisting group difference is contributing to the current group differences in neuroelectric function. However, all participants had normal or corrected-to-normal vision and were free of neurologic disease history, and the groups did not differ in age, education, or socioeconomic status, thus reducing the likelihood of preexisting differences. Future researchers using baseline testing and longitudinal designs will be able to further minimize the likelihood of a priori group differences. In addition, caution is necessary when interpreting the results because of methodologic considerations, such as a relatively small sample size, correlational design, and reliance on self-reported physician diagnosis of concussion. Strong agreement has been observed[56] between self-reported medical diagnosis of concussion and medical record documentation in college-aged athletes, and moderate reliability has been observed[57] between the numbers of self-reported concussions in former contact athletes questioned a decade apart. Further, the current method of screening for concussion has been used successfully in previous laboratory[17,27,54] and epidemiologic studies.[49,50] However, self-reported information, such as loss of consciousness and posttraumatic amnesia duration, should be interpreted cautiously and may account for the lack of significant relationships between injury characteristics and P1 component values. Future research would benefit from medical record access, baseline testing, and longitudinal designs. Despite these methodologic shortcomings, our findings add to the understudied area of nervous system function after concussion and suggest that concussive injuries have long-term implications for brain health and sensory function.

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