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Electrophysiological abnormalities in well functioning multiple concussed athletes

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Abstract

Objective: The present study was aimed at characterizing the short- and long-term effects of multiple concussions using an electrophysiological approach.

Method: Participants for this study were recruited from college football teams. They included athletes who never sustained concussions compared to two groups of asymptomatic multiple concussed athletes, one that sustained their last concussion within the year and the other more than 2 years prior to testing. All participants were submitted to an auditory three-tone Oddball paradigm while event-related potentials (ERP) were recorded.

Results: Results from ERP recordings reveal significantly reduced P3a and P3b amplitudes in the recent concussed group in the three-tone task compared to control athletes. In contrast, athletes who sustained their concussions more than 2 years prior to testing had equivalent P3a and P3b amplitude to that of controls.

Conclusion: These findings suggest that, despite functioning normally in their daily lives, concussed athletes still show subtle neuronal changes in information processing. Thus, the persistence of sub-clinical abnormalities on ERP components despite normal overt functioning may indicate sub-optimal compensation in multiple concussed athletes and leave them vulnerable to subsequent concussions.

Keywords: *Event-related potentials, concussion, P3a, P3b, athletes*

Introduction

Traumatic brain injury (TBI) of all severities is a significant public health problem with an annual incidence between 180–500 per 100 000 [1, 2]. Among the various degrees of TBI, concussions or mild traumatic brain injuries (MTBI) constitute ~85% of all TBIs sustained in the USA [2]. Concussions and their multi-faceted repercussions are a major concern in the sphere of professional sports. Epidemiological studies indicate that there are 1.6–3.8 million sport-related concussions occurring in the USA annually [3] including 20% of all high school football players. These figures are likely an under-estimation given that many athletes and coaches often fail to recognize them, especially when there is no loss of consciousness (LOC) [4]. Moreover, athletes sometimes do not report concussions purposefully, fearing that they will be taken out of the competition or under-estimating the severity of injury [5].

According to the American Academy of Neurology (AAN), a concussion occurs when the impact causes an alteration in mental status that may or may not be accompanied by LOC [6]. It is defined as a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces [7, 8]. There has been a large amount of research investigating the effects of sport-related MTBI, but little is known about the pervasiveness of the effects. The majority of neuropsychological studies report recovery from the effects of a concussion between 2–10 days after the incident [9–12]. However, there is a growing body of evidence suggesting that there are

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cumulative effects of concussions that manifest as increased susceptibility to subsequent concussions as well as an increase in their severity [13–16]. Among them, a large epidemiological study conducted by Guskiewicz et al. [13] demonstrated that college-level athletes with a prior history of sports concussion are 1.5-times more likely to suffer a subsequent concussion. This risk factor increases to 2.5 for athletes who have a history of two concussions, while having sustained three or more sports concussions increases this acquired vulnerability by a factor of 3. In High School athletes, Collins et al. [15] have found that the increased vulnerability to subsequent sports concussions is 9-fold. Many authors have also suggested that a history of three or more concussions is associated with poorer outcome, which is in line with the contention that the effects of sports concussions are cumulative [13, 15, 17–20]. Indeed, associations have been established between recurrent concussions and more severe on-field symptoms [15], slower recovery [21] or worsening neuropsychological performances after a subsequent concussion [18]. The pervasiveness of these deficits still needs to be investigated.

In recent years, it has been shown that event-related potentials (ERP) constitute a brain investigation technique that is highly sensitive to detect deleterious sequelae of sports concussion on cognitive functions [20, 22–24]. Several studies using classic oddball paradigms have shown P3b waveform component changes in concussed athletes in the first few weeks after the injury. Among them, Dupuis et al. [22] have compared symptomatic concussed athletes to control athletes using a visual Oddball paradigm. They found that the intensity of self-reported post-concussion symptoms is negatively correlated with the amplitude of the P3b component. Similarly, Lavoie et al. [24] found that a visual oddball paradigm elicited significant P3b amplitude reduction in symptomatic athletes when compared to other athletes with no prior concussion history. Their study also revealed that asymptomatic concussed athletes tended to show reduced P3b component amplitude when contrasted with athletes who had no concussion history. In the same vein, Gosselin et al. [23] discovered that the amplitude of the P3b component is significantly attenuated in asymptomatic concussed athletes tested a few weeks after their injury on a dichotic listening oddball task. Athletes who sustained their last sport concussion years prior to testing also showed P3b component amplitude reductions [25, 26] and latency delays [20]. De Beaumont et al. [26] also reported significant P3b amplitude attenuation in asymptomatic varsity football players with a history of two or more sports concussions who had sustained their last sports concussion on average 3 years prior to testing when they performed a difficult visual search oddball paradigm.

P3a alterations resulting from a mTBI are not limited to the first few months [27] or even years post-injury [28, 29]. A recent study conducted by De Beaumont et al. [25] reported a significant P3a amplitude attenuation in healthy former university athletes who sustained their last sports concussion more than three decades prior to testing. To the authors' knowledge, no study to date has specifically assessed P3a changes in younger sports concussion patients. This is surprising considering that the P3a component has often been shown to be more sensitive to clinical status in various neurological conditions [30] than its P3b counterpart. In addition, because the P3a component is thought to be related to resistance to distraction, it may provide additional support to previous studies that argued that distractibility could be the central impairment associated with MTBI [28, 31].

The present study therefore sought to explore whether sports concussions induce similar long-term P3a amplitude reductions in young athletes as those found in asymptomatic MTBI patients. This study also intended to examine the sensitivity of both P3a and P3b components to detect pervasive cognitive function changes in asymptomatic multiple concussion athletes tested at different time points post-injury. It was hypothesized that the amplitude of both P3b and P3a components would be significantly reduced in asymptomatic athletes with a history of multiple concussions, but less so in the group of concussed athletes who sustained their last concussion more than 2 years prior to testing than in athletes that sustained their last injury within 1 year from the date of testing.

Methods

Participants

All athletes included in this study were male members of university varsity teams. They have been assigned to one of three groups each consisting of 10 participants: (1) control group (athletes from football, volleyball and basketball teams with no prior concussion history); (2) recent concussion group (tested between 5–12 months after the last concussion); and (3) late concussion group (tested between 22–60 months after the last concussion). The authors deliberately recruited varsity athletes who participated in sports that required similar skills as football players in an attempt to restrict inter-group variability. Similarly to football, basketball and volleyball are both fast-paced team sports that heavily solicit visuospatial attention. Athletes included in both concussion groups were part of

university football programs. All athletes included in the concussed groups reported having sustained at least two concussions. Group classification was based on medical documentation for concussions that occurred in University settings, while self-reported concussions that took place prior to their University years were assessed with a concussion history questionnaire. None of the participants reported experiencing post-concussion symptoms at the time of testing, as assessed with the Post-Concussion Symptoms Scale. No athletes were taking drugs or medication known to affect central nervous system function at the time of testing and exclusion criteria included prior history of neurological (i.e. epilepsy) or psychiatric illnesses (including depression). Recruitment was performed with the help of medical and/or coaching staff of participating varsity teams. Each participant provided written informed consent prior to testing. The protocol was approved by the Université de Montréal ethics committee.

Procedure

Participants came to the laboratory for a single 2.5-hour session. Testing included the administration of a concussion history questionnaire to obtain information about sports concussions that had occurred prior to their University years. In addition to the number of previous concussions (if any), further questions inquired about the description of the accident, the nature and duration of post-concussion severity markers according to AAN classification [6] (e.g. confusion, post-traumatic amnesia (PTA), LOC). All reported concussions were classified by a sports physician using the practice parameters of the American Academy of Neurology [32]. Hecht and Kent [33] found that self-reported concussions and medically documented concussions generally showed agreement. Participants were then administered the Post-Concussion Symptoms Scale to assess the presence/absence and intensity of reported symptoms at the time of testing. This questionnaire asks players to rate themselves on a scale from 0 (no symptom) to 6 (severe symptom) on a series of 19 common post-concussion symptoms for a total possible score of 114. Participants subsequently underwent a battery of neuropsychological tests formerly used by the National Football League [34]. Finally, athletes had to perform the three-tone auditory oddball paradigm used to record event-related potentials.

The neuropsychological battery included the Hopkins Verbal Learning Test (verbal memory and learning); Color trails, parts 1 and 2 (visual search and inhibition); Controlled Oral Word Association Test (verbal fluency); Symbol Digit Modalities Test (processing speed and non-verbal incidental memory); Pennsylvania State University (PSU) Cancellation Task (processing speed and visual attention); Brief Visuospatial Memory Test (non-verbal memory and learning); and an orientation test. Response accuracy (Hits/Total number of trials), reaction time (time taken to complete a task), total omissions (number of missed target stimuli) and false alarms (Total number of erroneous responses to non-target stimuli) were computed for each test when appropriate. Reliability, validity and sensitivity of neuropsychological tests have been demonstrated to assess specific cognitive areas associated with MTBI in the general population [35, 36] (refer to the following book for a more detailed description of these tests [37]). This neuropsychological assessment procedure was kept constant across participants and test administration was performed by a trained neuropsychology student.

ERP paradigm

The three-tone auditory oddball paradigm used in the present design is inspired by an experiment conducted by Comerchero and Polich [38]. This paradigm consisted of three different sounds: frequent, rare target and rare deviant tones. The frequent tone (1700 Hz, 80 dB) was presented in 80% of the trials while the rare target (2000 Hz, 80 dB) and the rare deviant (4000 Hz, 90 dB) tones were each presented in 10% of trials. Participants were asked to press a button on a key pad with the thumb of their dominant hand when they heard the target tone. The inter-stimulus interval (ISI) was held constant at 2 seconds and the duration of each stimulus was 70 ms (10 ms rise and fall). A total of 400 stimuli were presented free field via two loudspeakers (Yamaha, model YST-M20DSP), separated by 90 cm and located at 115 cm in front of the subject and fixed at head level. Stimulus presentation was carried out using the STIM system from Neuroscan (Neurosoft, Inc. Sterling, USA).

EEG recordings

EEG was recorded using Ag/AgCl electrodes mounted on a cap according to the 10/20 system [39–41]. Recording sites included: FP1, FP2, Fz, F3, F4, F7, F8, FCz, Fc3, FC4, Cz, C3, C4, T7, T8, CPz, CP3, CP4, Pz, P3, P4, TP7, TP8, P7, P8, Oz, O1 and O2. A ground electrode was included in the montage and the nose was used as a reference electrode. The impedance was kept below 5 kΩ throughout the entire experimental procedure. EOG was recorded with four Ag/AgCl electrodes placed on the external canthi and on infra-/supra-orbital regions with the impedance kept under 8 kΩ.

EEG was digitized at 1024 Hz and band-pass filtered at 0.01–100 Hz during the recordings. The Neuroscan NuAmps system (Neurosoft, Inc. Sterling, USA) was used to amplify the signal.

ERP calculation

The EEG signal was epoched between −200 to 700 ms for all conditions, 0 corresponding to the stimulus onset. Epochs were corrected for ocular movements with a procedure developed by Gratton et al. [42]. EEG epochs contaminated by electrical artefacts above 100 mV were automatically rejected from further analyses while remaining EEG epochs were band pass filtered at 1–30 Hz (24 dB/octave) and baseline corrected relative to the activity recorded during the 200 ms that immediately preceded stimulus onset. These procedures were performed using BrainVision Analyzer software (Brain products, Inc., Germany, version 1.4).

Further ERP analyses were computed on averaged ERP waveforms for each experimental condition. The P3b component was computed from averaged brain activity recorded at centro-parietal electrode sites (Cz, CPz and Pz) when the rare target stimulus was presented. The P3a brain response was obtained from averaged brain activity recorded at central electrode sites (FCz, Cz, CPz) when the rare deviant tone was presented. Averaged brain activity for frequent stimuli was recorded from electrodes located on the midline (Fz, FCz, Cz, CPz and Pz).

The P3b size was then quantified as the mean amplitude during the 250–450 ms time window post-stimulus onset while the size of the P3a was obtained from the mean amplitude recorded during the 200–400 ms time window that followed stimulus onset. Latencies were calculated by taking the most positive sample point recorded within the predefined time window for both P3a and P3b components.

Statistical analyses

ERP and behavioural data, neuropsychological test scores and demographic information were subjected to standard descriptive statistics. Two between-subject ANOVAs were computed for each P3a and P3b components according to their amplitude and latency. These analyses were carried out on SPSS 13 for Windows (SPSS inc., Illinois). Tukey tests were used as post-hoc measures.

Results

Clinical characteristics and neuropsychological results

Table I shows the demographic and clinical characteristics of the three groups. None of the groups differed according to age ($F(2, 27) = 0.33$; $p > 0.05$) and level of education ($F(2, 27) = 0.062$; $p > 0.05$). Furthermore, recent and late groups of concussed athletes did not differ in terms of number of concussions ($t(1, 18) = 0.967$; $p > 0.05$), post-concussion symptoms reported ($t(1, 18) = 1.669$; $p > 0.05$) and the severity of the last injury ($t(1, 18) = 1.411$; $p > 0.05$), as graded according to American Academy of Neurology (AAN) criteria [6]. Groups of concussed athletes did not differ in terms of the number of experienced LOC ($t(1, 18) = 0.632$; $p > 0.05$) or PTA ($t(1, 18) = 0.647$; $p > 0.05$) consecutive to sports concussion.

Table I. Demographic characteristics for all groups and clinical characteristics for both recent and late concussed groups.

	Control group	Recent group	Late group
n	10	10	10
Age (years)	22.1 (1.4)	22.6 (1.5)	22.9 (3.3)
Education (years)	16.4 (1.4)	16.4 (1.3)	16.2 (1.8)
No of concussions	–	2.9 (1.1)	2.5 (0.7)
Time elapsed since last concussion (months)	–	9.1 (2.0)	33.2 (15.4)
LOC*	–	0.5 (0.9)	0.3 (0.5)
PTA*	–	0.9 (0.7)	0.7 (0.8)

Values are given as mean (standard deviation).
*LOC and PTA scores reflect the averaged number of occurrences per participant from each concussion group.

Neuropsychological results are presented in Table II. The level of performance was equivalent across groups on each neuropsychological test used to assess various cognitive functions.

Three-tone oddball task: Behavioural measures

No group differences were found on behavioural measures of reaction time ($F(2, 27) = 0.854$; $p > 0.05$), accuracy ($F(2, 27) = 0.645$; $p > 0.05$), number of omissions ($F(2, 27) = 0.596$; $p > 0.05$) or false alarms ($F(2, 27) = 1.921$; $p > 0.05$). Refer to Table III.

Three-tone oddball task: ERP measures

Figure 1 depicts grand average waveforms for each group when presented: (a) frequent stimuli, (b) rare target stimuli (eliciting the P3b component) and (c) rare deviant stimuli (eliciting the P3a component). A significant between-group effect on the mean P3a amplitude (Controls (8.21, SD 3.75 μV); Early concussion group (4.15, SD 1.56 μV); and Late concussion group (5.80, SD 2.67 μV)) was found from pooled electrodes ($F(2, 27) = 5.326$, $p < 0.05$). Subsequent Tukey post-hoc analyses revealed that the mean amplitude of the P3a component was

Table II. Neuropsychological tests results.

Neuropsychological tests results	Controls	Recent	Late	*F*	*p*
Hopkins–total recall	29.7 (1.7)	29.5 (3.7)	28.8 (5.4)	0.10	>0.05
Hopkins–delayed recall	10.8 (1.0)	9.8 (1.1)	10.5 (1.2)	2.01	>0.05
Symbol Digit Modalities test	56.6 (13.6)	58.4 (5.0)	66.5 (16.2)	1.35	>0.05
Verbal Fluency–total number of words	33.4 (9.0)	36.6 (7.6)	33.8 (5.9)	0.42	>0.05
PSU Cancellation test–total	47.6 (10.2)	48.4 (10.1)	46.8 (14.0)	0.39	>0.05
PSU Cancellation test–Omissions	2.6 (2.5)	3.8 (3.2)	2.7 (3.7)	0.44	>0.05
Brief Visual Memory Test–Total	31.6 (3.3)	28.9 (4.0)	29.3 (2.7)	1.68	>0.05
Brief Visual Memory Test–Delayed recall (Maximum score = 12)	11.9 (0.3)	11.2 (1.1)	12 (0)	3.11	>0.05
Color Trail A–Time (s)	29.0 (9.5)	28.4 (6.8)	24.4 (6.8)	0.70	>0.05
Color Trail B–Time (s)	66.7 (9.5)	57.7 (13.2)	54.0 (12.5)	1.24	>0.05
Post-Concussion Scale	10.8 (13.1)	8.2 (7.8)	2.7 (2.4)	1.35	>0.05
Orientation test (maximum score = 9)	9 (0)	8.6 (0.5)	8.7 (0.5)	3.00	>0.05

Values are expressed as means (standard deviation).

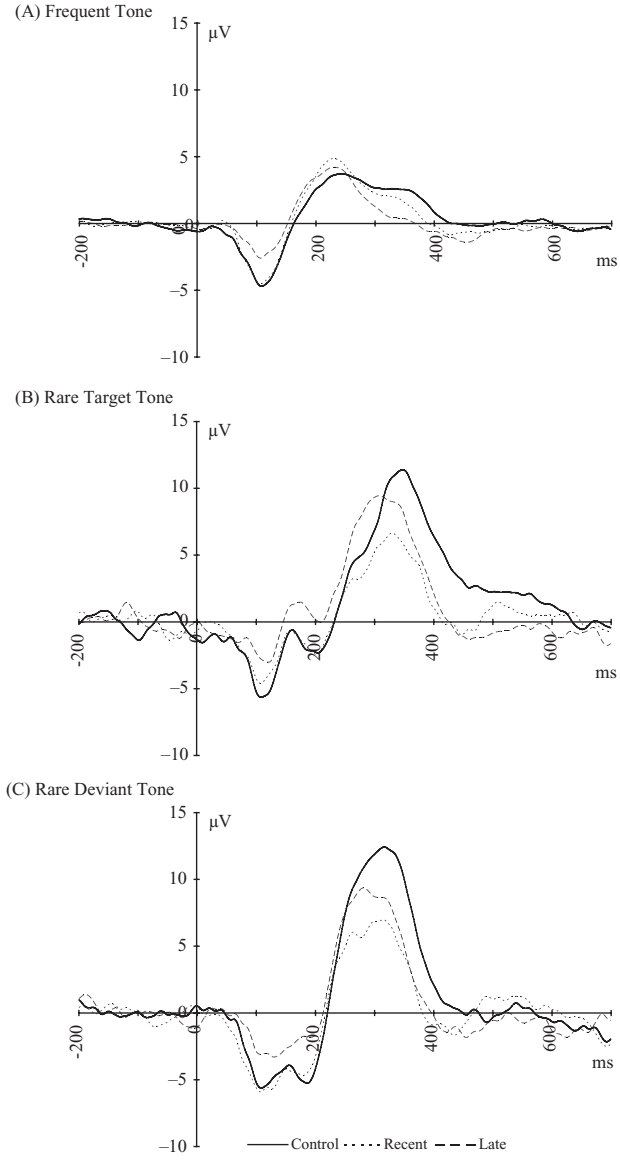

Figure 1. Three-tone Oddball.

Table III. Behavioural data for three tone Oddball task.

Parameters	Control	Recent	Late
Reaction time (ms)	615.2 (88.2)	568.3 (139.8)	550.4 (109.7)
Accuracy (%)	95.4 (8.5)	91.3 (16.6)	96.6 (3.0)
Omission (n)	1.8 (3.4)	3.2 (6.7)	1.1 (1.1)
Commission (n)	2.5 (2.0)	2.1 (2.1)	4.9 (5.3)

Values are given as mean (standard deviation).

significantly attenuated in the recent concussion group when contrasted with that of the control group ($p < 0.05$). The late concussion group also tended to be different from the control group, although it did not reach statistical significance ($p < 0.13$). A significant between-group effect on the mean P3b amplitude (Controls (7.14, SD 4.18 μV); Early concussion group (3.36, SD 1.90 μV); and Late concussion group (5.48, SD 2.20 μV)) was also found ($F(2, 27) = 4.165$, $p < 0.05$). Once again, post-hoc comparisons revealed a significant difference between the recent concussion group and normal controls ($p < 0.05$) while a similar comparison between the late concussion and control groups was not significant ($p > 0.05$). Interestingly, post-hoc analyses computed between late and recent concussion groups were not significant for either P3a or P3b components amplitude. None of the between-group comparisons computed on the latency of either the P3a (Controls (314, SD 30 ms); Early concussion group (285, SD 31 ms); and Late concussion group (293, SD 32 ms)) ($F(2, 27) = 2.317$; $p > 0.05$) or P3b (Controls (345, SD 20 ms); Early concussion group (331, SD 33 ms); and Late concussion group (318, SD 44 ms)) ($F(2, 27) = 1.507$; $p > 0.05$) components revealed to be significant.

Pearson correlations were computed between P3a/P3b components amplitude and concussion history information of concussed football players. The number of concussions sustained and the number of occurrences of LOC or PTA did not correlate with either P3a or P3b components amplitude.

Discussion

The present results partially confirm the hypothesis as it was found that asymptomatic multiply concussed athletes who sustained their last concussion between 5–12 months prior to testing show significant P3a and P3b amplitude reductions. This is consistent with other studies that showed P300 components changes in concussed athletes or MTBI patients weeks [22–24] and even years post-injury [26, 28, 31, 43]. On the other hand, athletes who sustained their last concussion between 22–60 months prior to testing did not differ from controls on the amplitude of both P3a and P3b components. The present study therefore suggests that the suppressing effect of sports concussion on both P3a and P3b amplitude is transient. To the authors' knowledge, this is the first controlled study—i.e. groups being equivalent according to age, education, symptoms reported at the time of testing and level of competition—to systematically delineate the effect of time since the injury in young asymptomatic athletes with a history of sports concussion on both P3a and P3b components.

The P3b amplitude reduction found in athletes who sustained their last concussion between 5–12 months prior to testing is thought to reflect deficits in memory updating [44] as lower P3b amplitude is associated with worse performance on memory tasks [45, 46]. However, the performance of both groups of concussed athletes on classic neuropsychological tests of memory, namely the Hopkins Verbal Learning Test and the Brief Visuospatial Memory Test, did not differ from that of controls. In the same vein, the P3a amplitude reduction found in concussed athletes tested from 5–12 months post-injury may reflect reduced frontal lobe function efficiency particularly affecting one's ability to shift attentional resources to novel stimuli [30, 47–49]. However, neuropsychological tests of attention and executive functions used in this study (Color trails, parts 1 and 2; Controlled Oral Word Association Test; Symbol Digit Modalities Test; Pennsylvania State University Cancellation Task) revealed equivalent performance scores across the three experimental groups. This is consistent with previous neuropsychological studies of sports concussion that typically demonstrate normal performance scores on neuropsychological tests when tested beyond 10–14 days post-injury [50]. The present study adds to mounting evidence suggesting that electrophysiology represents a particularly sensitive brain investigation tool to detect pervasive cognitive functions alterations [18, 23, 24, 26, 52] months post-injury that typically go unnoticed on classic neuropsychological tests [9, 53, 54]. These P3a and P3b amplitude attenuations are found in athletes who do not report experiencing memory or attention impairments and who can still function at fairly high levels despite their concussion history. The clinical implications of these electrophysiological anomalies are further magnified in older athletes with concussions who experience P3a/P3b amplitude reductions that significantly correlate with performance decrements on neuropsychological tests of memory and executive functions. Taken together, these findings suggest that these electrophysiological markers of memory and executive functions are particularly useful to longitudinally assess cognitive functions as they appear to be sensitive to both the aging process and sports concussions.

In addition to deepening the current understanding on the long-term effects of sports concussions on cognitive functions, one of the main objectives of electrophysiological studies such as this one, which purposefully include standard neuropsychological tests, is to highlight the limited sensitivity of gold standard neuropsychological tests over that of more refined brain investigation techniques such as ERPs. This is particularly important if one is to raise awareness on the long-term sequelae of sports concussions which have long represented a silent epidemic.

The present study showed persistent P3a/P3b amplitude reductions in asymptomatic concussed athletes tested within 1 year of their last injury that had subsided in a group of concussed athletes who had sustained their last sports concussion from 22 months up to 60 months prior to testing. These findings provide preliminary evidence that recovery of cognitive functions typically follows a rather slow, progressive course that can be assessed longitudinally with standard event-related potentials recordings elicited by a three-tone oddball paradigm. Future studies should attempt to replicate these findings in a wider sample of concussed athletes presenting with various concussion history characteristics.

Finally, having to rely on some occasions on concussion history self-reports as opposed to medical records to address consequences of sports concussion that occurred years prior to testing is not optimal. Prospective studies conducted with young athletes followed longitudinally are therefore

required to validate the residual effects of concussions observed in the present study.

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