International Journal of Psychophysiology 82 (2011) 16–23

Contents lists available at ScienceDirect

International Journal of Psychophysiology

journal homepage: www.elsevier.com/locate/ijpsycho

Review

A history of sport-related concussion on event-related brain potential correlates of cognition

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ARTICLE INFO

Article history:
Received 20 October 2010
Received in revised form 7 February 2011
Accepted 11 February 2011
Available online 19 February 2011

Keywords:
Mild traumatic brain injury
N1
P1
P3
ERN
Cognitive function
Athletes

ABSTRACT

Over the past decade, a growing body of research has detailed persistent changes to neuroelectric indices of cognition in amateur and professional athletes with a concussion history. Here, we review the relevant neuroelectric findings on this relationship while considering the duration from the last concussive event. Collectively, the findings support a negative relation of concussive injury to neuroelectric indices of brain health and cognition in the presence of normal clinical findings. The results suggest that event-related brain potentials are especially well-suited for identifying aspects of cognition that remain dysfunctional for an extended period of time, which are otherwise unidentified using standard neuropsychological tests. Such findings also suggest the need for additional research to fully elucidate the extent to which concussive injuries negatively impact brain health and cognition.

1. Introduction

Sport-related concussion has been described by the Center for Disease Control and Prevention as a 'silent epidemic' (Langlois et al., 2004) with traditional estimates suggesting that 300,000 cases occur in a given year (Center for Disease Control, 1997). More recent injury estimates suggest that nearly 4 million injuries occur on an annual basis (Langlois et al., 2006), which now includes those injuries that do not involve a loss of consciousness and those suspected to go unreported. Concussion is considered a type of mild traumatic brain injury (mTBI) with an economic impact approaching $17 billion in direct and indirect expenses annually (National Center for Injury Prevention and Control, 2001). Injured individuals often display deficits in cognitive functioning, postural control, and increased symptom reports (Broglio and Puetz, 2008) with known negative effects on academic (Moser and Schatz, 2002; Covassin et al., 2003; Moser et al., 2005) and job performance (Pelczar and Polityńska, 1997). In most instances, injured individuals return to a pre-injury level of functioning on clinical evaluations within seven to ten days of injury (McCrory et al., 2005), but the chronic effects of concussion remain unclear.

As the acute signs and symptoms of concussion represent functional changes in brain, rather than structural damage (Giza and Hovda, 2001), with traditional thought suggesting no long term effects to brain health and cognition. Indeed, several evaluations of young adult athletes with a concussion history, who have progressed beyond the acute stages of injury, indicate normal performance on a variety of clinical evaluations (Guskiewicz et al., 2002; Broglio et al., 2006; Collie et al., 2006; Iverson et al., 2006). These evaluations were conducted using standard clinical tests designed to evaluate large magnitude cognitive decrements associated with acute injury, the sensitivity of which to detect persistent subtle changes in cognitive functioning has been questioned (Broglio et al., 2006). Furthermore, recent evidence from former professional athletes suggests that cognitive health concerns emerge later in life in those with a concussion history. Indeed, rates of depression (1.5 fold increase; Guskiewicz et al., 2007), mild cognitive impairment, dementia (5 fold increase), and Alzheimer's disease (3 fold increase; Guskiewicz et al., 2005) are more common among ex-professional athletes and occur at a younger age than in the general population. Thus, it is apparent that more sensitive measures of cognitive functioning are needed to accurately identify and evaluate the long term effects of concussion in previously injured individuals, who have not yet demonstrated clinical pathologies.

To this end, electroencephalography (EEG), and event-related brain potentials (ERPs) in particular, have demonstrated the requisite sensitivity to better elucidate the existing literature gap between normal-appearing clinical performance and covert clinical pathologies that might underlie subtle deficits in cognitive performance. Therefore, the primary purpose of this review is to synthesize the current literature surrounding the persistent effects of concussion on

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doi:10.1016/j.ijpsycho.2011.02.010

long-term cognitive health as indexed by measures of the neuroelectric system, with a secondary focus on the duration from injury. Although a body of literature investigating traumatic brain injuries (i.e., mild, moderate, and severe) occurring under other circumstances exists, this review is limited to those events occurring within a sporting context. In addition, papers addressing the long-term effects of sub-concussive forces (e.g., soccer heading and boxing) were also excluded due to a lack of standardization in diagnostic criteria.

2. Event-related brain potentials

For almost a century, EEG has been extensively used to examine electrical activity associated with normal and abnormal brain function. More recently, ERPs have emerged as a technique to provide insight into the neural processes underlying perception, memory, and action. ERPs refer to patterns of neuroelectric activation that occur in preparation for, or in response to, an event. ERPs may be obligatory responses (exogenous) to stimuli in the environment or reflect higher-order cognitive processes (endogenous) that often require active consideration from the individual (Hugdahl et al., 1995). The benefit of the ERP approach lies in its temporal sensitivity, which affords researchers the ability to parse the stimulus–response relationship into its various component cognitive processes. That is, ERPs offer the requisite temporal resolution to determine which aspects of the stimulus–response relationship are influenced by some manipulation, providing a greater understanding of the mechanisms underlying overt behavior. Further, the ERP technique has been used for many years, and is one of the most well studied Psychophysiological techniques, producing a number of reliable and replicable components. However, ERPs have poor spatial resolution relative to other neuroimaging techniques, making source localization difficult. Other criticisms of this approach include distortion of the signal due to the distance of the electrodes from the neuroelectric source, and the fact that ERPs can only be recorded from specific groups of neurons with a specific orientation toward the scalp. Regardless of these shortcomings, ERP have remained a viable tool for investigating covert aspects of human information processing, and have been used to understand mTBI, even in the absence of clinical symptoms and overt cognitive deficits, which has quickly made them a valuable tool for the evaluation of sport-related concussion.

Although several components, such as the contingent negative variation (CNV), error-related negativity (ERN), and N2 have been evaluated in relation to sport-related concussion, the P3 component has captured considerable attention. Occurring about 300–800 ms following the presentation of a stimulus, the P3 (i.e., P300 of P3b) component is thought to reflect neural activity associated with the revision of the mental representation of the previous event (Donchin, 1981), such that the P3 is sensitive to the allocation of attentional resources during stimulus engagement (Polich, 2007). P3 timing, marked by its peak latency, is believed to index stimulus classification and evaluation speed independent of response selection and action (Duncan-Johnson, 1981; Verleger, 1997).

The P3 has been further divided into the P3a and P3b subcomponents, which represent related but distinct neuroelectric processes that are distinguished by their unique scalp distributions and the context in which they occur (Polich, 2007). Stimulus discrimination tasks, and in particular "oddball" paradigms, have been extensively used to elicit these subcomponents. That is, the presentation of an infrequent target stimulus amid a train of frequent non-target stimuli has been repeatedly demonstrated to elicit a pronounced P3b component, with a parietal scalp distribution (Polich, 2007). Conversely, the infrequent presentation of an unexpected, uninstructed stimulus during a similar train of non-target stimuli has been observed to elicit a P3a component, marked by a fronto-central scalp distribution and a relatively short latency (Polich, 2007). The P3a is thought to reflect the selection of stimulus information associated with attentional orienting (Knight, 1984; Kok, 2001; Rushby et al., 2005). Therefore, P3a amplitude is theorized to reflect greater focal attention toward the orientation of stimuli in the environment (Polich, 2007). Accordingly, the P3a and P3b, along with other ERP components, provide insight into select aspects of cognition that may be altered by the persistent effects of concussion. Through the use of these measures, inferences may be made regarding which aspects of cognition continue to present dysfunction, and strategies for cognitive interventions may be effectively formulated.

3. P3b

The initial work on the affect of sport-related concussion to ERP indices of cognitive dysfunction occurred approximately a decade ago with three publications appearing from two different Canadian laboratories (Dupuis et al., 2000; Gaetz et al., 2000; Gaetz and Weinberg, 2000). Gaetz and Weinberg (2000) compared younger (18–34 years) and middle age (35–55 years) adults with a concussion history to those with no injury history. Participants underwent a battery of tests, including visual and auditory oddball tasks in which ERPs were recorded. The results indicated that no differences existed as a function of age grouping, but that 40% of the participants with a history of concussion fell more than 2.5 standard deviations outside their normative database for the visual P3 (i.e., P3b), compared to 0% in the non-concussed group (Gaetz and Weinberg, 2000). A similar, yet smaller effect (10–20% above the 2.5 SD in the concussion history group based on age grouping) was observed for the auditory oddball task. More traditional means of (statistically) analyzing the P3 data indicated that the concussion history groups, regardless of age, had smaller P3 amplitude and longer P3 latency relative to the group of individuals without a history of concussion (Gaetz and Weinberg, 2000). These findings have been replicated in other laboratories as well (Gosselin et al., 2006). Based on contemporary theories of the P3 (Donchin and Coles, 1988; Polich, 2007), the findings suggest that a history of concussion is related to deficits in attentional resource allocation in the service of context updating and delays in cognitive processing speed during stimulus acquisition.

However, other publications (Dupuis et al., 2000; Gaetz et al., 2000) have provided only partial support for the P3 findings described above using similar oddball tasks. Dupuis et al. (2000) reported a decrease in P3 amplitude for previously concussed individuals who had symptoms at the time of testing (1.7 ± 2 months post injury) compared to a group of previously concussed individuals who were asymptomatic at the time of testing (9.8 ± 7.8 months post injury) and non-concussed individuals. No group differences in P3 latency were reported (Dupuis et al., 2000). Further, De Beaumont et al. (2007) reported smaller P3 amplitude for individuals with a history of multiple concussions relative to those with a single concussive event and those with no history of concussion. The data are especially informative as they controlled for time since injury in their analyses, suggesting that the cumulative effects of multiple concussions on the reduction in P3 amplitude are not merely functions of time since injury. Despite these lasting deficits in P3 amplitude, P3 latency differences were not observed between groups (De Beaumont et al., 2007).

Recently, Di Russo (Di Russo and Spinelli, 2010) evaluated executive control function in professional boxers, who had experienced at least one boxing loss by knockout (mean 3.1 knockouts), indicating at least one concussion, albeit additional injuries may have occurred. Professional fencers and non-athlete controls were recruited as controls, but concussion history was not reported. Participants executed a Go/No-Go task to evaluate the P3b response, wherein the boxers demonstrated suppressed amplitude and increased latency during the Go portion of the task compared to both control groups. In addition, latency was increased relative to the controls during the No-Go portion of the task, which required

response inhibition for successful task performance. Collectively, these data suggest a decrease in the capacity to allocate attention resources, delays in processing contextual information, and poorer capability to inhibit 'pre-potent' responses as a function of a history of concussions and the sub-concussive blows that regularly occur during boxing participation. However, the relationship between the number of concussions and neuroelectric indices of cognitive deficits was not examined, indicating that this question remains open.

Alternatively, Gaetz et al. (2000) compared four groups of participants who had either never sustained a concussion, or had a history of 1, 2, or 3 or more concussions, and were at least six months removed from their last injury. They reported no group differences in P3 amplitude, but significantly longer P3 latency in the group with 3 or more concussions relative to the group who had never been injured (Gaetz et al., 2000). No differences were observed for those who had sustained 1 or 2 concussions. Accordingly, findings across these studies are inconsistent, with some supporting a detrimental relation of concussion history to cognitive processing speed (i.e., longer P3 latencies), and others indicating reductions in attentional resource allocation (i.e., smaller P3 amplitude). It should be noted that all studies had relatively small sample sizes and differences in the time since the last concussive event differed across studies. Regardless, it is clear that concussion has a lasting effect on the neuroelectric system that extends beyond the acute injury stage.

Interestingly though, several reports have demonstrated a relationship between self-reported symptoms and the P3 component. Specifically, Dupuis et al. (2000) indicated that participants in their symptomatic concussion group exhibited a negative correlation between P3 amplitude and symptomatology. That is, those reporting a higher number and greater severity of symptoms generated smaller P3 amplitude. No such relationship was observed for the number of concussions, severity of the most recent concussion, or time from the last injury (Dupuis et al., 2000). Parallel findings were reported by Gosselin et al. (2006), who observed a negative relation between self-reported headache and difficulty concentrating with P3 amplitude. Again, no such relationship was observed for the number of concussions, the severity of the previous injuries, loss of consciousness, and the time from the previous concussive event (Gosselin et al., 2006). Further, Gaetz et al. (2000) reported a positive correlation between P3 latency and self-reported post-concussion symptoms of 'memory problems' and 'taking longer to think'. Similar findings were reported by Lavoie et al. (2004) in which symptomatic athletes with a history of concussion had smaller P3 amplitudes than previously concussed asymptomatic athletes and never concussed control athletes. Importantly, negative correlations were observed between the severity of post concussion symptoms as well as the time since the last concussive event and P3 amplitude (Lavoie et al., 2004). Collectively, these findings suggest that the P3 component may serve as an objective index for chronic cognitive dysfunction associated with concussion history, with some evidence indicating that it may also reflect specific symptoms reported by the individual. Future research clearly needs to advance these preliminary findings to determine the nature and the extent of the relationship between the P3 component and prolonged symptoms experienced following a concussive event.

4. P3a

Broglio et al. (2009) examined a history of sport-related concussion on the P3a and P3b components in a sample of 90 intercolligate club and recreational athletes ($M = 19.7 \pm 1.3$ years), of whom 46 had a prior history of concussion (3.4 years post-injury). Participants completed a standard sport specific neurocognitive assessment (i.e., ImPACT) and then performed a visual novelty oddball task, while their ERPs were recorded. Similar to other previous reports (Broglio et al., 2006; Collie et al., 2006; Iverson et al., 2006) the ImPACT battery was not successful in discerning prolonged cognitive dysfunction associated with concussion history. However, corroborating earlier P3b studies, a reduction in P3 amplitude was observed for those with a history of concussion relative to those who had never sustained a brain injury. Interestingly, no such effect was observed for the P3a component, suggesting that while persistent cognitive dysfunction related to the allocation of attentional resources (i.e., P3b amplitude) remained more than three years after injury, neuroelectric measures reflecting the attentional system, which governs orienting, were intact (Broglio et al., 2009). Such a finding suggests selectivity in the prolonged effects of concussion history on brain health and cognition in young adults.

However, other researchers have observed a different pattern of findings, indicating a lack of consensus relative to the selectivity of concussion history on cognition. Specifically, De Beaumont et al. (2009) examined concussion history in a group of older adults ($M = 60.8 \pm 5.2$ years) who had sustained their last concussion approximately 30 years ($M_{age} = 26.1 \pm 9.2$ years) prior to testing, and compared them with a group of age-matched adults ($M = 59.9 \pm 9.1$ years) who had never been concussed. Participants performed neuropsychological tests, followed by an auditory three stimulus oddball task. Results indicated performance deficits on the neuropsychological tests and modified flanker task in the concussion group, suggesting impaired memory and inhibitory control. Importantly, the groups did not differ in their mental status, as equivocal group differences were noted on the mini mental status exam. Further, concussed athletes demonstrated significantly smaller P3a and P3b amplitude, along with longer peak latencies for these components, relative to the no-injury history group (De Beaumont et al., 2009). This pattern of findings suggests general deficits in cognitive function, given that deficits were observed for components of cognition related to the allocation of attentional resources (P3b amplitude), the orienting of attention (P3a), cognitive processing speed (P3 latency), as well as measures of cognitive control (response accuracy to neuropsychological battery).

Still other studies have observed deficits in P3a amplitude, without concomitant changes in latency (Theriault et al., 2009). Specifically, asymptomatic athletes who had sustained multiple concussions were compared with athletes with no history of concussion. Concussed athletes were further divided into groups based on those who had sustained their last concussion within the previous year and those who had sustained their last concussion more than two years prior. In response to an auditory oddball task, recently concussed athletes (i.e., within the previous year) exhibited smaller P3a amplitude compared to the non-concussed athletes. Those who had sustained their concussion more than two years earlier demonstrated a non-significant trend for smaller P3a amplitude relative to non-concussed athletes (Theriault et al., 2009). A similar effect was realized for P3b, but it should be noted that component amplitudes were averaged from overlapping electrodes (P3a: average of FCz, Cz, and CPz; and P3b: average of Cz, CPz, and Pz), so the specificity of the two components is unclear.

Despite the interesting findings of these studies (De Beaumont et al., 2009; Broglio et al., 2009; Theriault et al., 2009), conflicting results were obtained, with the exception of P3b, indicating that a clear picture has not emerged relative to other aspects of cognition, namely attentional orienting (as reflected by the P3a component). As such, this remains an open question in this literature. However, it is also clear that multiple differences in experimental protocols and study populations may have led to these conflicting outcomes. Another possibility though, is that cognitive dysfunction related to attentional orienting may not be pronounced in young adults who have sustained a brain injury. But, over the course of the lifespan such an injury may exacerbate cognitive declines associated with normal aging resulting in clinical pathologies during later life. Where, on the other hand, processes subserving the allocation of attentional resources are more profoundly affected by brain injury,

demonstrating notable changes during earlier periods of the lifespan, which remain dysfunctional later in life.

5. Sensory evoked potentials

Exogenous (i.e., obligatory) evoked potentials such as the brainstem auditory evoked potential (BAEP), P1, and N1 reflect automated sensory processing involved in the early stages of perception. The BAEP, first discovered by Jewett (1970), reflects basic neuronal function in the brainstem and peripheral nervous system (Jewett and Williston, 1971). The P1 and N1 are closely related exogenous components associated with sensory processing. The P1 is thought to reflect sensory gating, preferential attention to sensory inputs (Key et al., 2005), and the inhibition of irrelevant sensory information (Waldo et al., 1992). The P1 also may reflect the process of amplifying the signal to noise ratio in selective attention (Hillyard and Muente, 1984; Hillyard and Anllo-Vento, 1998; Hillyard et al., 1998) given that enhanced P1 amplitudes are observed in relation to attended stimuli (Awh et al., 2000). The N1 is an exogenous ERP component thought to reflect the discrimination and encoding of basic stimulus properties (Vogel and Luck, 2000). Similar to the P1, N1 amplitudes are enhanced in response to attended stimuli (Knight et al., 1981; Mangun, 1995; Awh et al., 2000) and may also reflect an amplification of the signal to noise ratio during selective attention processes (Hillyard and Muente, 1984; Hillyard and Anllo-Vento, 1998; Hillyard et al., 1998).

Among the earliest studies to examine ERP correlates of sport-related concussion, Gaetz and Weinberg (2000) compared neurocognitive function in previously concussed and non-concussed athletes across multiple ages (see the P3b section above for details). The researchers used an extensive battery of tasks to elicit several ERP components. In particular, the BAEP was elicited by a click in the stimulated ear and a white noise mask in the non-stimulated ear. The researchers used traditional statistical analyses as well as clinical, qualitative criteria to evaluate their results. According to their clinical criteria, 10% of concussed participants' aged 18–34 years had BAEP values beyond the normal range compared to 2.2% of their age-matched non-concussed peers. Interestingly, none of the participants' aged 35–55 years in either the concussed or control groups had abnormal BAEP values. Despite these descriptive clinical observations, traditional statistical analyses did not yield significant differences for the BAEP component for younger or middle aged participants, suggesting that sport-related concussion does not detrimentally affect basic auditory/brainstem processes.

In addition to the BAEP, Gaetz and Weinberg (2000) evaluated participants' P1 component, which was elicited via a pattern reversal paradigm (i.e., checkerboard). Again, researchers used both traditional and clinical criteria to evaluate their results, with 30% of concussed participants aged 18–34 years displaying abnormal P1 latencies, compared to none of their non-concussed peers. Similar to the younger concussed participants, 30% of the middle aged concussed participants displayed abnormal P1 latencies compared to only 5% of control participants. Despite these descriptive clinical observations, traditional experimental analyses again did not yield significant differences for the P1 component for the younger participants. However, middle aged concussed participants demonstrated significantly longer P1 latencies compared to their non-concussed peers, suggesting that concussions experienced during younger adulthood may lead to belabored sensory gating and preferential attention in middle age (Gaetz and Weinberg, 2000).

In another experiment evaluating ERP correlates of sport-related concussion, Gosselin et al. (2006) compared the N1 component in previously concussed and never concussed athletes. Dichotic standard and deviant tones were presented within the context of a modified auditory oddball task to elicit an ERP. Previously concussed participants were bifurcated according to the presence/absence of symptoms at the time of testing. Symptomatic participants were an average of 15.1 ($\pm$16.6) weeks post injury, and asymptomatic participants were an average of 5.3 ($\pm$3.1) weeks post injury (Gosselin et al., 2006). Participants' performance was equivalent on all neuropsychological tests, save for the digit symbol modalities test. Smaller N1 component amplitudes were observed for both symptomatic and asymptomatic previously concussed participants in the unattended channel compared to the non-concussed group. This suggests that a history of concussion, regardless of the presence of symptoms, relates to persistent decrements in the encoding of basic auditory stimulus properties. Interestingly, the difference occurred in the unattended channel suggesting that concussion may lead to long-term decrements in the ability to process unattended sensory information.

Collectively, such a pattern of findings is difficult to interpret and further study is needed to better elucidate potential differences in these sensory evoked potentials. However, the data provide some preliminary evidence to suggest that sport-related concussions deleteriously affect the speed at which middle age participants encode basic stimulus features. That is, concussions experienced during younger adulthood may have a delayed onset of sensory dysfunction, or may be exacerbated during aging leading to the ability to detect such dysfunction later in the lifespan. Again, such speculation would require replication to draw this conclusion with greater certainty.

6. ERN

The error-related negativity (ERN; also referred to as the Ne) is a response-locked ERP component that has recently been investigated with considerable interest, (Falkenstein et al., 1991; Gehring et al., 1993; Scheffers et al., 1996; Luu et al., 2000; Ridderinkhof et al., 2002; van Veen and Carter, 2002; Yeung et al., 2004). The ERN refers to a negative component that peaks approximately 80–100 ms after an unintended response (Falkenstein et al., 2000; Holroyd and Coles, 2002), and neuroimaging research (Carter et al., 1998; Dehaene et al., 1994; Miltner et al., 1997; van Veen and Carter, 2002) suggests that it is generated in the dorsal anterior cingulate cortex. This component is theorized to reflect action monitoring processes following the evaluation of erroneous behaviors to initiate top–down compensatory processes, thus ensuring correct action on subsequent trials (Holroyd and Coles, 2002; Yeung, 2004).

Employing a modified flanker task to examine aspects of cognitive control, Pontifex et al. (2009) evaluated the ERN in individuals with a history of concussions ($M = 1.7$) and never before concussed participants. Concussed participants were an average of 2.9 years from their last concussive injury. Interestingly, concussed participants did not differ in behavioral task performance relative to control participants on a neuropsychological test battery (i.e., ImPACT). However, while performing the flanker task, participants with a history of concussion demonstrated significantly smaller ERN amplitudes relative to those participants who had never sustained a concussion. This finding suggests that the ability to recognize errors or evaluate response conflict is compromised in young adults with a concussion history. Interestingly, Pearson correlations indicated a negative association between the number of concussive incidents and the size of the ERN potential, with an increased number of injuries related to progressively smaller ERN amplitude (Pontifex et al., 2009). Additionally, concussed participants evidenced decreased response accuracy and increased flanker interference relative to control participants, suggesting decrements in the management of perceptual and response interference. Taken together, these findings suggest that both the ability to evaluate response conflict (i.e., error recognition) and the flexibly to adjust subsequent behavior are compromised in individuals with a concussion history. Thus, neurocognitive decrements in cognitive control of action monitoring appear to endure for several years after the last concussive incident. In addition, these data

further demonstrate the sensitivity of neuroelectric measurement to detect subtle neurocognitive decrements in cognition that appear to be linearly related to the number of previous injuries.

7. Movement potentials

The contingent negative variation (CNV) is an endogenous ERP component characterized by a large negative shift reflecting response anticipation that occurs between a warning stimulus (S1) and an imperative stimulus (S2; Walter et al., 1964; Tecce, 1972; Tecce and Cattanach, 1993; Bressler and Ding, 2006). The component has been further decomposed into two subcomponents: initial CNV (iCNV) and terminal CNV (tCNV; Loveless and Sanford, 1974; Birbaumer et al., 1990). The iCNV is the early portion of the component and has been associated with the processing of information associated with the warning stimulus, while the later occurring tCNV has been associated with anticipation and response preparation for the imperative stimulus (Brunia, 1988; Rockstroh et al., 1993).

To date, only two studies have examined CNV correlates of sport-related concussion (Gaetz and Weinberg, 2000, Gaetz et al., 2000). Specifically, Gaetz and Weinberg (2000) compared neurocognitive function in previously concussed and non-concussed athletes across multiple ages (see the P3b section above for more details) and used visual and auditory stimulus pairings to elicit the CNV. Researchers employed both clinical and statistical criteria to evaluate their results. The clinical results indicated that 40% of younger (18–34 years) concussed participants produced visual CNV responses beyond the normal range, as opposed to 10% of younger control participants. Further, statistical analyses revealed significant differences between younger concussed and control participants, suggesting that sport-related concussion negatively affected processes related to cueing, motor anticipation, and response preparation in younger adults (Gaetz and Weinberg, 2000). For middle aged (35–55 years) adults, 10% of concussed participants produced visual CNV responses beyond the normal range, as opposed to 5% of control participants. Statistical analyses did not reveal any significant differences in CNV responses for middle aged participants. Perhaps sport-related concussion negatively affects cueing, anticipation, and response preparation processes in younger, but not middle aged adults (Gaetz and Weinberg, 2000). However, one consideration is that the mean time from injury for the middle aged group was skewed by a single participant, who was only one month post injury. With this exception removed, middle aged participants were significantly further from their last injury (42.4 vs. 37.2 months) than the young adult group. It is possible that this period of time was sufficient for the resolution of concussion effects on the CNV component, or perhaps concussion affects on motor processes are more difficult to detect in middle aged participants given that the CNV decreases with age (Dirnberger et al., 2010; Gajewskia et al., 2010). Further study is necessary to fully clarify the pattern of findings observed.

Examining CNV correlates of sport-related concussions, Gaetz et al. (2000) compared the neurocognitive function of young athletes with a history of 1, 2, or 3 or more concussions with control participants. All participants were at least six months from their last injury, and researchers used a visual stimulus pairing to elicit the CNV. Analyses failed to reveal any significant differences between concussed and non-concussed participants, regardless of the number of concussions, suggesting that a history of multiple concussions does not negatively affect response preparation processes of young athletes (Gaetz et al., 2000). Taken together, the implications of these findings remain unclear, with one study suggesting that sport-related concussion may negatively affect neuroelectric processes underlying young adult's response preparation, and the other suggesting no such relationship. Thus, these findings should be interpreted cautiously, and further research is needed to better understand the relation of sport-related concussion to processes subserving motor preparation.

The inconsistent nature of concussive injuries suggests that the impaired areas may not be limited to brain regions subserving cognitive function. Indeed, in the acute stage of injury several investigations have indicated changes to postural control (Guskiewicz et al., 2001; Guskiewicz et al., 1997). Thus, recording and evaluating the neuroelectric system related to motor control may provide novel insight into the long-term effects of injury. Motor-related cortical potentials (MRCPs) are one such signal and are comprised of three components: Bereitshafts potential, motor potentials, and motor monitoring potentials. Collectively, these signals are thought to represent cortical activity involved in the initiation and control of motor activity. Slobounov et al. (2002) have used a more comprehensive ERP approach to examine the effects of concussion on movement. That is, they dissected the preparatory period prior to a voluntary movement to examine differences in the *Bereitshaftspotential* (BP), which has been found to reflect neural activation associated with the early stages of motor preparation. The negativity seen just prior to the onset of a movement, termed motor potentials (MP), reflects neural activation associated with the later stages of motor preparation (Slobounov et al., 2002). Furthermore, the movement monitoring potential (MMP), the slow negative potential thought to reflect the ongoing monitoring of persistent task-related movement, was also evaluated in six individuals with a history of a single sport-related concussion, occurring 10–20 months previously (Slobounov et al., 2002). These findings were compared to those from six never-before concussed individuals. All participants were asked to press a load cell with their dominant index finger at 25% or 50% of their maximum voluntary contraction (MVC), such that force production would increase at a constant, gradual rate while visual feedback regarding their current force level was provided.

Slobounov et al. (2002) observed no group differences during the 25% MVC task. However, during the 50% MVC task, the concussed group exhibited less negativity (i.e., smaller amplitude) across all three movement potentials (i.e., BP, MP, and MMP) compared to the non-concussed group. Further, whereas the non-concussed group had larger amplitude from the 25% to 50% MVC, no such increase was observed for the concussed group, who failed to exhibit a BP during the 50% MVC (Slobounov et al., 2002). Behaviorally, deficits were also observed for the concussed group, with decreased accuracy during the 50% MVC condition. The findings suggest that transient functional changes in neural networks underlying motor control and coordination are observed for a prolonged period following a concussive injury, with specific dysfunction related to the planning, initiation, and monitoring of voluntary movements (Slobounov et al., 2002). A follow-up study using a within-participants design corroborated these deficits across the three motor potentials in concussed athletes 3, 10, and 30 days post injury relative to baseline, indicating that dysfunction of motor control and coordination exists long after individuals are cleared to resume sport participation based on standard neurocognitive assessments (Slobounov et al., 2005).

8. Conclusion

The current review was intended to provide a description of previous works that utilized ERP measures to elucidate the persistent effects of concussion on cognitive dysfunction. As noted throughout this review, a number of investigators have used the ERP approach because of its capacity to identify covert changes in cognitive processes that occur in the time between stimulus engagement and response execution. That is, ERPs afford the opportunity to better understand which aspects of cognition are affected by concussion due to their superior temporal specificity relative to other neuroimaging measures and overt behavioral responses elicited from clinical testing. However, the ERP technique also has a number of shortcomings, which may limit its feasibility for assessing sport-related concussion acutely. Specifically, EEG instrumentation is expensive and requires

an individual with advanced training to not only collect data, but also reduce and analyze the collected signal. Further, EEG preparation requires a significant amount of time, negating its use in the athletic environment where rapid decision making is often necessary. Given that ERPs reflect both exogenous and endogenous aspects of cognition, dedicated space that is void of extraneous stimuli is necessary to maximize signal quality. Such costs and space demands are not easily obtained in most sport settings. Alternatively, the EEG approach is better suited for return-to-play decision making and an examination of the persistent effects of concussion given its requisite sensitivity in identifying deficits that otherwise go unnoticed using routine task performance measures.

Regardless, the findings, summarized in Table 1, from multiple investigations suggest moderate to large decreases in ERP components related to stimulus acquisition (e.g., P3 amplitude), cognitive processing speed (i.e., P3 latency), action monitoring (i.e., ERN), and motor control and coordination (i.e., BP, MP, and MMP) in the presence of normal performance on a variety of clinical assessments. These prolonged changes suggest that concussed individuals do not allocate the same level of attentional resources toward aspects of their environment, nor are they capable of initiating and monitoring their actions in a similar manner to those without a prior history of injury. In addition, the changes brought about by concussion appear related to the number of concussions sustained by the individual and the time from injury. That is, ERP deficits appear to increase in line with the number of concussions sustained, while a reduction in deficits occurs as the time from injury increases.

How these persistent changes in the neuroelectric system relate to the underlying pathophysiology of injury is not entirely clear. Animal models have demonstrated cerebral cell death within 72 h of mTBI

Table 1
Summary of published ERP and EE experiments. Effect sizes were calculated based on the values presented in the respective manuscripts as an estimate of concussion's impact on the various indices of brain function.

Author	Sample population	Time from last injury	Method	Significant findings
Broglio et al. (2009)	Intercollegiate and recreational athletes: Concussed ($m=20$ yrs; $m=1.7$ concussions); Controls ($m=19.4$ yrs)	$m=2.9$ yrs	Novelty oddball	Concussed participants had smaller N2 ($d=2.89$), P3a ($d=0.39$), and P3b ($d=2.31$) amplitudes compared to controls.
De Beaumont et al. (2007)	College football players: Controls ($m=22.5$ yrs); 1 concussion ($m=23$ yrs); 2+ concussions ($m=23.5$ yrs, $m=2.8$ concussions)	1 concussion $m=4.7$ yrs; 2+ concussions $m=2.6$ yrs	Visual search task	Participants suffering multiple concussions demonstrated smaller P3 amplitudes than participants with 1 concussion ($d\leq2.71$)
De Beaumont et al. (2009)	Concussed former athletes ($m=61$ yrs), controls ($m=59$ yrs).	$m=34.7$ yrs	Auditory oddball	Concussed participants had longer P3a latencies ($d=0.68$) and amplitude ($d=0.39$), and P3b amplitudes ($d=0.52$) compared to controls.
Di Russo and Spinelli (2010)	Professional boxers (n=12, $m=28.1$ yrs), professional fencers (n=12, $m=26.3$ yrs), student-controls (n=12, $m=25.8$ yrs)	*n/a*	Go/No-Go	Boxers demonstrated suppressed P3b amplitude ($d\leq3.7$) and increased latency ($d\leq2.7$) during the Go task and increased latency ($d\leq3.9$) during the No-Go task
Dupuis et al. (2000)	3 groups of college athletes (m age=21.5): control, asymptomatic and symptomatic.	Asymptomatic, $m=9.75$ months; Symptomatic, $m=1.7$ months.	Visual oddball	Symptomatic participants demonstrated overall smaller P3 amplitudes compared to asymptomatic and control participants ($d=1.02$).
Gaetz and Weinberg (2000)	Contact Athletes divided into groups by age [18–34 ($m=25.5$) and 35–55 ($m=42.4$)] and compared to controls	Age 18–34 ($m=3.1$ yrs); 35–55 ($m=3.13$ yrs)	Visual oddball tasks: word, shape, and number; Auditory oddball task	Concussed participants of all ages demonstrated longer P3 latencies during all visual oddball tasks ($d\leq7.68$).
Gaetz et al. (2000)	High school Hockey players grouped by number of concussion: 0, 1, 2, or 3+	All subjects were >6 months post-injury; 3+ group $m=13.2$ months	Visual oddball task	3+ concussed participants demonstrated longer P3 latencies than control group ($d=1.21$)
Gosselin et al. (2006)	Professional, semi-professional and collegiate hockey and football players: control ($m=22.0$ yrs);asymptomatic concussed ($m=26.1$ yrs; $m=3.5$ concussions); and symptomatic concussed ($m=25.7$ years; $m=5.1$ concussions)	Asymptomatic, $m=5.3$ weeks; Symptomatic, $m=15.1$ weeks	Auditory oddball	Symptomatic athletes demonstrated smaller P2 amplitude ($d=0.74$) than controls; greater P3 amplitudes and longer P3 latencies were present in the concussed groups compared to the control group ($d=$n/a).
Lavoie et al. (2004)	Student athletes (18–26 years) grouped into control, asymptomatic(m concussion=2.6), and symptomatic (m concussion=3.2)	Asymptomatic ($m=9.9$ months); Symptomatic ($m=1.7$ months)	Modified visual oddball	Symptomatic participants demonstrated smaller P3 amplitude compared asymptomatic ($d=2.69$) and controls ($d=1.72$) participants
Pontifex et al. (2009)	Intercollegiate and recreational athletes: Concussed ($m=19.9$ yrs; $m=1.7$ concussions); Controls ($m=19.4$ yrs)	$m=2.9$ yrs	Modified flanker task	ERN (Ne) on error trials was smaller for concussed participants compared to controls ($d=3.11$).
Slobounov et al. (2002)	Collegiate athletes: Concussed ($m=22.3$ yrs) and Controls (n/a)	10–20 months	EEG during 25 and 50% maximum voluntary contraction (MVC) of the index finger	Concussed athletes had significantly smaller amplitudes during the 50% MVC for BP ($d=0.92$ to 1.10), MP ($d=1.07$ to 1.19) and MMP ($d=1.10$ to 1.22)
Slobounov et al. (2005)	Collegiate athletes: Concussed (n/a)	Evaluations at baseline and days 3, 10, and 30 post-concussion.	EEG taken during 3 static and 1 dynamic posture	Significant differences (BP, MP, and MMP) between baseline and post-injury days ($d=$n/a)
Theriault et al. (2009)	Varsity high school athletes: 3 groups: Control ($m=22.1$ yrs); Recent concussion ($m=22.6$ yrs; $m=2.9$ concussions); Late concussion ($m=22.9$ yrs; $m=2.5$ concussions).	Recent concussion $m=9.1$ months; Late concussion $m=33.2$ months	Auditory oddball	Recent concussed participants displayed smaller P3a ($d=1.41$) and P3b ($d=1.16$) amplitudes compared to controls. The Late concussion group displayed smaller P3a ($d=0.74$) and P3b ($d=0.50$) amplitudes compared to controls. Late concussion group participants also displayed large P3b amplitudes ($d=1.03$), than those in the recent concussion group.

yrs = years: d = Cohen's d:, n/a = data unavailable.

(Tashlykov et al., 2007) and neuron atrophy and death occur in humans following TBI (Maxwell et al., 2003). Thus, it is possible that the suppressed electrophysiology seen here is related to post-injury cell morbidity and mortality. Regardless of the cellular mechanism, the effects of concussion can persist well beyond the acute stage of injury and may extend for years or decades. As such, concussion can no longer be thought of as a transient injury void of long term effects.

Contemporary reports indicate that multiple concussions may bring about increased rates of mild cognitive impairment, depression, and early onset Alzheimer's disease in former professional athletes (Guskiewicz et al., 2005, 2007), but caution is necessary in generalizing these results. That is, a mere 1600 athletes play professional football each year and have sustained a 10+ year career (i.e., interscholastic, college, and professional) of sub-concussive and concussive blows that preceded these outcomes. More importantly, nearly 1.2 million interscholastic football athletes take to the field each year, the vast majority of which will not continue beyond high school. It is not presently known if these pathologies reported in the former professional athletes will be present in individuals following a 3 or a 4 year high school football career.

The gradual deterioration in cognitive functioning that occurs with age appears to accelerate with brain injury (see Mansel et al., 2010 for review) and autopsy reports attribute the declines to brain scarring (Omalu et al., 2005, 2006). No author reported that neuroelectric deficits associated with concussion were associated with clinical pathologies in their sample populations, but the findings of the research summarized herein clearly indicate a change in brain function that is associated with concussion. The abundant cognitive reserve of the young adult brain likely compensates for these changes (Satz, 1993). For example, higher levels of education and pre-injury intelligence quotient scores correlate negatively with post-injury symptom duration (Dawson et al., 2007) and those with lower cognitive reserve show greater declines on cognitive assessments immediately following brain injury (Ropacki and Elias, 2003). Thus, as normal cognitive decline occurs with age, the coupled effect of injury severity and quantity coupled will be a likely factor in the development of clinical pathologies.

As such, the clinical neuropsychological tests typically used to evaluate cognitive function beyond the acute stage of injury should be interpreted cautiously, as these instruments were not designed to detect subtle, persistent changes in cognitive function (Iverson et al., 2006). Rather, these tools serve the purpose of evaluating large magnitude changes in information processing, planning, memory, and mental flexibility (Aubry et al., 2002). Although we did not complete a comprehensive review of neuropsychological tests and their ability to detect subtle persistent changes in cognition, we speculate that following injury there is a rapid functional recovery in which compensatory mechanisms such as the adoption of new strategies and/or functional reorganization via brain plasticity allow the athlete to perform normally on standard clinical assessments. This is followed by a more prolonged neuronal recovery during which time subtle deficits in cognitive functioning are present, but not apparent in standard clinical concussion assessment tools. More sophisticated instrumentation, such as ERPs, clearly has the ability to detect more subtle ongoing changes, although the 'real world' implications of these changes in late life are not yet known. Thus, additional investigations involving longitudinal designs using both ERP and clinically relevant measures are needed to better clarify these changes.

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