NEURAL PROCESSING FUNCTION FOLLOWING CONCUSSION: A NARRATIVE REVIEW OF THE P300 EVENT-RELATED POTENTIAL COMPONENT

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ABSTRACT

Background: Mild traumatic brain injury (mTBI), or concussion, represents a significant public health concern. Current diagnostic practice relies on clinical tests and symptom reporting, as standard neuroimaging is unable to detect the microstructural changes of injury. This diagnostic gap creates challenging decision making for clinicians regarding diagnosis and medical clearance. The P300 event-related potential (ERP) measured via electroencephalography, is the most extensively studied neurophysiological measure in current concussion literature. Previous studies demonstrate changes in amplitude and latency reflecting functional deficits in the patient population.

Objective: To summarize the evidence for how the P300 ERP component differs following concussion. We also summarized how the P300 changes throughout concussion recovery, and investigated whether or not differences exist between populations and mechanism of injury.

Methods: A systematic literature search was conducted across Medline, PsychInfo, and Embase databases. Following title and abstract screening and full text review, 19 original studies were included, published between 1988 and 2025, across pediatric and adult cohorts. Studies were eligible if they assessed the auditory P300 ERP in a concussion population compared to baseline measurements or healthy controls.

Results: P300 amplitude reduction was the most common finding, reported as significant across 10 of the 19 studies. This reduction was most consistently detected in sport-related concussion populations. P300 latency was less consistently altered, with 9 studies showing no significant change compared to controls. Prolonged latency was most reliably seen across the acute civilian concussion population, and chronically in blast-injured military cohorts. Two studies showed amplitude increases. Substantial variability in concussion definition, study design, ERP methodology, and population traits limited cross study comparison.

Conclusion: The P300 ERP demonstrates potential as an objective, portable, and more accessible tool to supplement current clinical protocols. P300 abnormalities frequently persisted beyond symptom recovery and clinical clearance, which challenges the adequacy of symptom based return to activity protocols. The P300 shows promise as a prognostic marker of recovery trajectory and treatment monitoring tool rather than a standalone diagnostic marker. Future research should prioritize standardization of ERP evaluation tools, longitudinal studies with pre-injury assessments in the non-sport population, and investigation of P300 within a broader multimodal biomarker framework.

TABLE OF CONTENTS

INTRODUCTION

Concussion is a mild traumatic brain injury (mTBI) which represents a significant public health concern. Of all traumatic brain injuries (TBI), concussion, represents 80% of cases. Some estimates report approximately 42 million people worldwide suffer a concussion every year (1). While this number is high, the true incidence may be underestimated, as diagnosis is difficult and subjective, leading to underdiagnosis. Thus, underreporting poses a barrier to timely medical care (2,3). In Canada alone, over 400,000 people 12 years and older have experienced a concussion in their lifetime (4). While definitions vary, concussions are commonly referred to as a mild cranial trauma resulting in physiological disruption of brain function with minimal or no loss of consciousness. Patients typically present as GCS 13-15, with negative results on standard diagnostic imaging, and may present with functional deficits. The mechanism of injury consists of contact to the head or body leading to external force transmission to the brain (5). Sport-related concussions (SRC) have received recent research focus, with one study suggesting that approximately 1 in 9 youth athletes experience an SRC yearly (6). Concussions also occur beyond sport, including other mechanisms of injury (MOI) such as motor vehicle accidents, military service with blast injuries, domestic settings, and workplace settings (7–9). While most people recover within weeks of concussion, 30% of adults and 40% of youth experience prolonged symptoms after 30 days of initial injury (2,7). While the prognosis of concussion is quite good relative to moderate and severe cases (0.1% mortality for concussion), persistent symptoms may require targeted treatment to facilitate recovery (10,11).

Following concussion, impairments may arise impacting functional, cognitive, and emotional health. These impairments can dysregulate attention, concentration, and information processing (10,11). Underlying neural impairments may persist beyond symptom resolution,

which is clinically relevant for premature return to activity without full recovery (2). In concussion seen in the military from blast injuries, persistent post-concussion symptoms (PPCS) have been documented years after the initial injury (8). Thus, both the acute and chronic patient population burden the healthcare system, requiring monitoring, treatment, and testing.

History and clinical assessment supports the clinical diagnosis of concussion (5,7,9,12). Regular imaging modalities such as CT and MRI are not useful for these patients outside of ruling out more severe brain injuries, as these tools do not detect changes following concussion (9,12). Thus, diagnosis relies on clinical judgement, where a clinician interprets signs, self-reported symptoms, and mechanism of injury (5). This approach is subjective (3), placing a burden on physicians and physician assistants to make critical management decisions, such as return to work/sport/activity, without objective markers of recovery. Moreover, secondary concussive impact before complete recovery is associated with second impact syndrome, which can cause severe cerebral hemorrhage, which is in some instances fatal (13). Thus, recognition and removal from risk following concussion is critical. In many contexts, symptoms may not be reported honestly, which further complicates diagnosis and medical clearance decision making. Thus, the lack of objective measures of concussion pathophysiology to support diagnosis represents a critical clinical gap (12). Ongoing research is investigating potential objective biomarkers to supplement clinical diagnosis of concussion, including measurement of blood-based inflammatory biomarkers (14,15), cerebrovascular function using ultrasonography (16), and neurophysiological function using electroencephalography (EEG) (17).

One promising approach for objectively measuring neurophysiological dysfunction following concussion are event-related potentials (ERP). ERPs use electroencephalography (EEG) to assess neural activity in response to a time-locked stimulus. The responses are labelled

by their polarity and latency following a stimulus (17–19). These objective measurements of neural processing underlie neurophysiological function, which may allow practitioners to detect subtle disruptions following concussion (4,11). Research suggests that subjects with a prior history of concussion, and exposure to accumulated sub-concussive impacts may also experience changes in the latency and amplitude of ERPs (21), which may indicate persistent neuro-physical changes (16,22,23). Practical barriers such as cost, accessibility, and signal processing complexity have limited clinical ERP assessment following concussion (20). Recent advances have leveraged machine learning and portable EEG to make ERP assessments of concussion more clinically feasible (24). The Neurocatch platform, for instance, can rapidly extract "brain vital signs" (such as the P300), which can be used in both clinical and athletic settings, and is a Canadian company (25). It has been suggested that the detection of underlying cognitive changes can support the traditional neuropsychological assessments done with a concussion diagnosis (18,25).

Among the ERP components evaluated in relation to concussion, the most extensively investigated is the P300, a positive neuroelectric deflection occurring approximately 300 milliseconds after a stimulus. EEG electrodes are placed on the scalp in a standardized framework using anatomical landmarks. Electrodes are named by their region, such as frontal (Fz), parietal (Pz), and central (Cz). The P300 is often elicited using a two-tone oddball paradigm, where participants detect a deviant target tone embedded in a sequence of standard tones (26). Depending on the task, ERP responses may reflect sensory, cognitive, and/or motor processing in response to auditory or visual stimuli. Thus, the amplitude and the latency of the P300 ERP component may be informative in evaluating changes in these neural processing capacities post-concussion (26,27). The amplitude, measured in microvolts, of the P300 reflects

the neural recruitment in response to the stimulus (28). The changes in latency reflect processing speed and efficiency (2). The P300 can be further subdivided into two components. An earlier component, the P3a, which reflects orienting responses to novel stimuli, and the later component, the P3b, reflects cognitive processing (7,27).

Concussion is associated with transient neurometabolic deficits, that while not detectable on standard imaging, may impair neural processing (29). Thus, the P300 ERP component might be a clinically useful objective measure of concussion-related impairment that could support diagnosis and medical clearance decision making. However, the influence of concussion on the P300 ERP component has yet to be summarised. Thus, the purpose of this narrative review is to summarize the evidence of auditory P300 ERP changes following concussion when compared to healthy controls.

METHODOLOGY

A systematic literature search was performed across the following electronic databases: Medline, PsychInfo, and Embase. The search strategy and terms used are detailed in Appendix A. Seed studies were selected *a priori* to ensure search strategy was effective at capturing manuscripts of interest (20,30). All search results were uploaded to the Covidence systematic review platform for screening.

The inclusion criteria for this review involved studies that: 1) assess the auditory P300 ERP, 2) were written in English, 3) have concussion or mild traumatic brain injury as the mechanism of injury, 4) included patients who were currently symptomatic at time of assessment, and 5) were original article where post injury evaluations are compared to pre-injury baseline or control values. Eligible study designs included randomized control trial, cohort study,

case-control, prospective, and retrospective case series. Articles that were excluded were review articles, commentaries, letters to the editor, abstracts, and dissertations. Any research performed on animals as well as any research where the majority of participants had severe or acquired brain injury were excluded. An exclusion hierarchy was used to exclude studies based on the following hierarchy: abstract only, no English, no humans, no original data, not EEG, not auditory ERP, not mTBI/concussion, no comparison, and dissertations.

Prior to title and abstract screening, MGN and JDN independently screened 50 articles to establish inter-rater agreement. A 90% agreement on the sample articles was achieved before continuing. Title and abstract screening was performed independently on the Covidence platform. Full text screening was then conducted by MGN and JDN. Conflicts were resolved using a third expert opinion.

Once the studies were screened, data extraction was performed on included studies to extract title; authors; year of publication; study design; duration; location; sex, age, and number of participants; inclusion/exclusion criteria used; mechanism of injury; event related potential outcomes assessed; comparison groups; results to inform this review.

RESULTS

The initial search yielded 2040 papers to title and abstract screen. Title and abstract screening eliminated 1872 studies, with 178 texts remaining. Of the 178 full texts that were screened, 143 were excluded. Of the remaining 35 studies, 19 studies evaluated the auditory P300 event related potential component in a concussed population and were included in the following capstone. The papers reviewed were published from 10 countries, spanning from 1988 to 2025, with 11 of the studies taking place over the last decade. The papers evaluated mainly

adult populations, with 3 studies including pediatric participants. Studies were grouped based on mechanism of injury and timing, including acute SRC (5 studies), acute and subacute concussion (8 studies) and chronic/persistent post-concussion symptoms (6 studies). Six studies were categorised as non-sport/mixed cause which included motor vehicle accidents, bicycle accidents, falls, and general head trauma (7,9,26,31–33). Two studies specifically investigated road traffic accidents (RTA) (11,28). One study included mine blast related concussion (8). Finally, all studies investigating chronic concussed populations were mixed and/or unspecified MOI. P300 outcomes varied between studies. All studies used an auditory oddball paradigm to generate the ERP responses. Results of these studies' evaluation of the auditory P300 following concussion can be seen in Appendix B.

Sport-Related Concussion (SRC)

Five studies examined the P300 ERP in the sport concussion population with a mix between youth and adult cohorts. The most consistent finding was a significant reduction in the P300 amplitude, with no significant latency increases or decreases generally found between concussed and healthy controls. In concussed adolescents approximately 20 days post injury, Ruiter et al.'s cross sectional study demonstrated significantly lower P300 amplitude, and no significant change in latency compared to controls (34). Similarly, Gosselin et al.'s cross-sectional study demonstrated significantly reduced P300 amplitude in both symptomatic and asymptomatic concussed athletes compared to healthy controls. No significant differences were observed between acute and subacute samples. P300 latency results were reported spatially over different electrodes: central (Cz), frontal (Fz) and parietal (Pz), and prolongation was found only to be significant over the central electrode (2). A cohort study by Clayton et al. evaluated 364

college and high school athletes at baseline and following an SRC in a longitudinal design over 4 sport seasons. They identified a significant decrease in P300 amplitude following concussion compared to the athlete's own baseline. P300 amplitude differences were observed in the acute phase and persisted despite clinical recovery in 38% of return to play athletes (RTP). No P300 latency changes were reported between timepoints (20). Additionally, a longitudinal cohort study by Mortazavi et al. similarly observed diminished P300 amplitude in the acute, RTP and persistent post-concussion symptom (PPCS) timepoints compared to pre-injury baseline. P300 latency did not significantly differ from pre-injury baseline at the acute or RTP stages. P300 latency was significantly reduced in the PPCS group, though this finding was noted to be potentially unreliable due to low signal amplitude by the author (35).

In contrast, a longitudinal cohort study by Fickling et al. reviewed a portable ERP framework of "brain vital signs" which standardises electrophysiological changes in amplitude and latency in male junior ice hockey players. 43 players were baseline tested and 12 sustained a concussion over the duration of study. Results demonstrated an increase in P300 amplitude in the athletes following concussion when compared with their own pre injury baseline at the acute phase. P300 amplitude remained significantly elevated at RTP timepoint when compared to their own baseline. Furthermore, P300 latency was shown to decrease significantly in the acute phase, which trended back to normal in the RTP group (32).

Acute & Sub-Acute concussion

There were eight studies that evaluated the P300 changes in the acute and sub-acute phases following injury. The timepoints of initial assessment ranged from within 24 hours to 14

days, with follow up assessments as far out as 8 months post injury (7,9,11,26,28,31–33). Studies reported variable findings, ranging from both amplitude and latency changes to no differences in P300 amplitude or latency.

Pratap-Chand et al. found significantly reduced P300 amplitude and increased P300 latency within the first four days of injury in twenty concussed patients compared to age and sex matched controls in a prospective cohort. These participants were followed up with over one to eight months and both amplitude and latency normalized over time (31). In a similar study design, Nandrajog et al. also reported significantly longer P300 latency, which normalized at two to three months following injury in seventeen concussed patients compared to controls. Significant reduction in P300 amplitude was detected at the frontal electrode (Fz) only (26).

Following motor vehicle accidents, Ford and Khalil's cross-sectional study found significant P300 amplitude attenuation across the central, frontal, and temporal electrodes in 54 concussed patients, with the maximum effect seen at Fz. There was no significant latency differences reported (9).

Two prospective studies evaluating RTA, appeared to evaluate the same data set of 38 participants, half of which suffered a concussion, were compared to healthy controls at the acute phase (7 days or less), and follow up (2-6 months). Reza et al.'s 2019 study demonstrated a significant amplitude increase over only the T6 electrode site in the acute post-injury (11). Reza et al.'s 2025 study used sLORETA (standard low resolution electromagnetic tomography) source localization in the RTA population across the same timepoints. In contrast to other studies, they measured dipole intensity of the P300 rather than raw P300 amplitude, which is a measure of the strength of the estimated neural source from the sLORETA localization. They reported significantly increased P300 dipole intensity in both timepoints when compared to controls,

rather than P300 amplitude. P300 latency was not reported (28).

Cavanagh et al. investigated a neurosurgery and emergency medicine patient population in the sub-acute phase (14 days or less) and at a two-month follow-up compared to initial sub-acute assessment. Both subcomponents of the P300 were evaluated (P3a and P3b). They reported no significant group difference between concussion patients and controls for both P300 amplitude and latency. However, the P3b amplitude in the sub-acute phase significantly predicted symptom scores at follow up (7). Similar results regarding non-significant group level P300 amplitude and latency differences were found in von Bierbrauer and Weissenborn's prospective study across all timepoints (24 hours, 1 week, 3 week, 8 week). Despite no significant changes at the group level, prolonged P300 latency were found in roughly 20% of a symptomatic subgroup at 24 hours, which normalized by about 3 weeks (32). In contrast, Sivák et al. found no significant P300 amplitude or latency differences between 31 concussed patients versus controls in both the acute phase and follow-up 3-7 months later in their cross-sectional study (33).

Chronic concussion and Persistent Post-Concussion Symptoms (PPCS)

There were six studies performed evaluating the P300 responses in the chronic concussion population. The timepoints ranged from three months to over three years following injury. More variation was seen in the results of this chronic subset.

A cross-sectional investigation of chronically symptomatic patients by Solbakk et al. reported significantly reduced P300 amplitude at one year or more post injury in response to auditory stimuli compared to healthy controls. No significant differences in P300 latency were found (36). Similarly, Chebotariova et al. reported a 60% reduction in P300 amplitude in chronic

PPCS patients who suffered a mine blast injury half a year to three years prior in a cross sectional study. Differing from Solbakk et al., however, they reported an increase in P300 latency by approximately 45-50ms compared to healthy controls (8). Furthermore, Oakley et al.'s retrospective study reported significantly lower P300 amplitude in a PPCS cohort compared to controls, with a significant increase in amplitude that coincided with symptom resolution. There was no significant latency differences reported between the PPCS cohort and healthy controls (37).

Vander Werff and Rieger's cross-sectional study reported significantly altered P300 responses in a chronically symptomatic concussion cohort compared to healthy controls, but with variation in the direction of association. Participants were assessed three to eighteen months post injury. There was no significant difference in latency between patients compared to healthy controls. They evaluated the auditory P300 response to speech in both quiet and noisy environments. However, no significant difference between noise condition and group level responses was seen in amplitude or latency (38). Gaetz and Weinberg measured the P300 response in a cross sectional study using the auditory N2-P3 complex, which reflects the amplitude difference between the N2 trough and P3 peak of the auditory ERP. They reported no significant amplitude or latency differences (39).

In a cross sectional evaluation of post traumatic headaches at three to six months post injury, Alberti et al. found significantly prolonged P300 latency in 25 concussed patients compared healthy controls with no significant differences in amplitude (12).

Across the 19 included studies, the most consistent finding was the alteration of the P300 amplitude. There were 10 studies that showed P300 differences in concussed individuals compared to healthy controls or pre injury baseline. In eight studies, the amplitude was reported to

significantly decrease in concussed individuals compared to healthy controls or pre injury baseline. In two studies, the P300 amplitude was shown to increase in concussed individuals compared to healthy controls or pre injury baseline. One study reported significantly altered P300 amplitude changes compared to controls, with the direction of change varying across individuals in the patient group. Four studies reported no significant differences in P300 amplitude or latency between concussed individuals and healthy controls. Regarding P300 latency, 9 studies reported no significant differences between concussed individuals and healthy controls or pre injury baseline. Five studies showed significantly longer P300 latency, two showed a significant decrease, and two it was not reported/limited in its data.

DISCUSSION

This narrative review examined the evidence of P300 event-related potential (ERP) changes following concussion across 19 studies from 1988 to 2025. The findings reveal that P300 differences are variable and context dependent. While there were P300 differences across concussion populations and timepoints, heterogeneity in methods and findings limit the current clinical utility of the P300 ERP component. Diagnostic utility of the P300 may depend on the population and availability of pre injury baseline data. While P300 amplitude and latency may differ in concussion populations compared to controls at various timepoints, the magnitude and direction of the P300 response was not uniform. The evidence is most convincing for the P300 as a complement to the existing tools we have clinically, rather than a standalone diagnostic marker that replaces current standard of care.

Overall Pattern of P300 Findings

The most consistent finding across studies was a reduction in P300 amplitude, reported significantly in 10 of the 19 studies reviewed. Although the magnitude and direction of differences varied, reduced P300 amplitude was the most consistent, reported across sport-related concussion (SRC), acute, and chronic concussion cohorts in 10/19 studies (2,8,20,26,31,34–37). This finding was most consistently reported in the SRC studies, where significant amplitude reduction was seen relative to preinjury baseline or healthy controls in 4/5 studies (2,20,34,35). Generally, a reduction in P300 amplitude following concussion reflects impaired attentional resource allocation, and reduced information processing capacity. This suggests a reduction in neural recruitment in response to a stimulus.

In contrast, two studies in the RTA population demonstrated an increase in P300 amplitude, or more specifically in the study by Reza et al. 2025, a higher intensity of the P300 source dipole in patients in the acute and chronic phases after concussion. Both studies suggest this is due to a compensatory neural recruitment mechanism following concussion (11,28).

While a less consistent marker, P300 latency was reported to be significantly longer in both acute and chronic symptomatic patient populations (5/19 studies), which was indicative of slower cognitive processing (8,12,26,31). The largest P300 differences were reported in blast injured military cohorts, with latency prolongation of 45-50ms alongside amplitude reductions of 60% (8). Another unique finding was in a post-traumatic headache cohort, reporting a mean isolated latency prolongation of approximately 30ms with no change in amplitude (12). In 11 studies, no differences in P300 latency were reported. The differential findings between P300 latency and amplitude suggest that neural processing speed was maintained, but neural recruitment was impaired following concussion. Null findings at the group level, however, do

not discount meaningful neurophysiological disruption at the individual level. Cavanagh et al. found no significant difference in P300 amplitude or latency between concussion patients and controls, yet within the concussion group, P3b amplitude significantly predicted symptom recovery. Furthermore, von Bierbrauer and Weissenborn found similarly normal group-level values for both amplitude and latency, but found latency prolongation in 20% of patients, exclusively those with suspected organic brain syndrome. This suggests that the P300 may have limited utility as a diagnostic evaluation but may identify those at risk of delayed recovery or those with comorbidities.

Sources of Heterogeneity

One of the biggest challenges when synthesizing the data from these 19 studies is the variability in the definition and classification of concussion patients. While consensus definitions for the diagnosis of SRC exist (5), diagnostic criteria were not made explicit in any of the five SRC studies. Two studies administered the Sport Concussion Assessment Tool (SCAT), consistent with current standard of practice (20,35). Civilian studies predominately used Glasgow Coma Scale based definitions of 13-15, with a loss of consciousness less than 30 minutes (7,26,31,33,36). Studies in military populations diagnosed their mine blast injuries (MBI) by a special military medical commission, using VA/DoD guidelines (8). New diagnostic criteria for concussion was published in the Archives of Physical Medicine and Rehabilitation (ACRM) in 2023. This involves criterion assessing mechanism of injury: clinical signs, acute symptoms, clinical exam/lab findings, neuroimaging (lack of positive findings), and finally symptoms not being better accounted for by confounding factors (40). Included studies used injury definitions which overlap with portions of the ACRM definition, although the lack of

uniform definitions and reporting standards limited cross study comparison and may partially explain heterogeneous findings. It is expected that studies which included patients with more severe TBI would report greater P300 disruption. However, several studies with less strict injury definitions reported null P300 findings (7,32,33), while some with stricter criteria found significant differences (2,20,34). This suggests that injury definition alone may produce more heterogenous samples, which could account for some of the P300 differences between studies.

Study design differences may also explain heterogenous findings. Longitudinal designs with preinjury baselines, used by several SRC studies, provide a higher level of evidence than matched cross-sectional studies. Cohort studies are able to account for individual variation in P300 outcomes which cannot be fully accounted for in cross-sectional designs (19,31,32). This point is critical as P300 amplitude varies between individuals at baseline (24), so a single post injury measurement compared to a population control cannot reliably detect injury related change in a given patient, highlighting the clinical utility of preinjury baseline data. A relatable concern is the degree to which control groups were matched. While 6 studies had both age and sex matched controls explicitly noted, this was not consistently reported across all studies. Studies also varied in reporting how their P300 parameters were measured. Electrode placement contributed to spatial inconsistency, as significant change was seen to the frontal (Fz) electrode in 2 studies, and one study only reported values at the central (Cz) and parietal (Pz), with the rest not being reported on (2,9,12,26).

A unique approach combining auditory ERP with brain MRI by Sivák et al. (2008) reported the failure of standard P300 to detect differences in patients with traumatic brain lesions, indicating that the P300 ERP may not be as clinically valuable in more severe TBI populations. This is particularly interesting as it suggests that ERP and MRI are measuring

different aspects of brain function post-concussion. Instead of morphological damage, the P300 reflects the integrity of neural networks and processing function which may be disrupted in the absence of any detectable structural change.

Heterogeneity was also seen in the ERP extraction method itself. Fickling et al. used a proprietary “brain vital signs” extraction method with normative scoring, rather reporting tradition amplitude and latency measures, which may provide an explanation for their divergent findings from other SRC. The direction of any bias is unclear without direct comparison of the two approaches in the same population. A unique example of methodology shaping results is seen between the two studies by Reza et al. The 2019 study used standard EEG scalp measurements which found limited P300 differences (significantly increased amplitude at a single electrode). In contrast, the 2025 study used the same population using a technique called sLORETA source localization which demonstrated significantly increased P300 dipole intensities at the acute and follow up timepoints. This technique estimates the strength of underlying neural generation rather than the raw voltage of the P300 on the scalp. This suggests that conventional amplitude measurement may underestimate neurophysiological disruption that source localization is able to detect (11,28). Furthermore, three studies used a more complex paradigm, demonstrating P300 differences where simpler designs had not (36,38,39). Instead of the standard two-tone oddball paradigm (standard and deviant tones), Solbakk et al. used a three-tone test that added an additional novel stimuli which demanded more attentional filtering (36). Vander Werff and Rieger measured the auditory P300 using speech stimuli in both noisy and quiet conditions (38). Finally, Gaetz and Weinberg utilized a multimodal battery of both auditory and visual ERP paradigms, suggesting the auditory component alone was considerably less sensitive (39). This suggests that neural processing differences following injury may be better

detected by tasks requiring more complex neural integration. Simple neural function may be preserved despite underlying pathophysiology, whereas more complex neural demands may reveal deficits. These observations support the need for broader adoption of standardized ERP guidelines such as those proposed by Duncan et al. (27).

Recovery Trajectory and Persistence of P300 Abnormalities

A key finding across studies is that the phase of recovery in which the P300 was assessed influenced findings. The most pronounced abnormalities were seen in the acute phase, though the definition of acute phase varied from 24 hours to 14 days post injury, reflecting further heterogeneity. Normalization was reported as recovery progressed in some studies (11,20,26,30,31,35). Additionally, P300 abnormalities persisted beyond symptomatic recovery and clinical clearance in some studies (2,20,30). This implies incomplete neural recovery despite symptom resolution following a concussion.

Given evidence of P300 abnormalities outlasting symptom resolution, the underlying neurophysiological processes of attentional resource and cognitive processing likely recover at different rates than subjective symptoms. Clayton et al's study reported 38% of athletes with persistent P300 amplitude deficits at RTP timepoints despite meeting standard clearance criteria, even though other neuropsychological scores and reaction time had returned to each athlete's own preinjury baseline values (20). Gosselin et al. found that asymptomatic athletes had reductions to their P300 amplitude comparable to their symptomatic counterparts, meaning there is detectable neural dysfunction independent of symptom reporting (2). Together, these findings challenge the notion that symptom resolution coincides with concussion recovery. The absence of symptoms may reflect a threshold where dysfunction is no longer perceived consciously, but

true preinjury neural function has yet to be reached. Furthermore, Fickling et al's brain vital signs study also suggested that cumulative impacts that do not meet the criteria of a diagnosed concussion may be associated with P300 changes in the absence of concussion. Further research by this author replicated this connection between "brain vital sign" changes reported after sub-concussive head impacts experienced over a sports season without sustaining a concussion, which raises important questions about the cumulative neurophysiological burden of contact sport (41,42). Thus, assessment of P300 following SRC may be confounded by exposure to repetitive head impacts. Further research is required to determine the influence of repetitive head impacts on ERP outcomes. Moreover, it is presently unclear if ERP outcomes can discriminate between SRC and repetitive head impact exposures.

The results of this review have implications for current clinical practice in interpreting medical clearance. If P300 abnormalities persist despite symptom resolution, then current protocols which are primarily driven by subjective symptom reporting may be clearing patients before full brain recovery, potentially exposing them to reinjury. This is concerning in the context of second impact syndrome, where subsequent head trauma before complete recovery can be associated with catastrophic cerebral consequences (13). Therefore, P300 assessment may be particularly useful for RTP decision making and recovery monitoring.

A compelling case can be made for athletes in high risk for concussion sports to receive P300 monitoring, particularly given the feasibility of serial baseline testing and portable EEG technology seen in studies by Clayton et al., and Fickling et al (20,30). In contrast, preinjury assessments are less feasible in civilian and military populations. Without preinjury baseline data and substantial investments into a normative database for these populations, the ability to utilise P300 assessment for concussion detection is challenging.

In chronic populations, P300 amplitude remains suppressed alongside symptom patterns and recovers with symptom improvement. One study demonstrated P300 amplitude recovered in parallel with symptom resolution after multidisciplinary rehab in the PPCS cohort, while another showed persistent P300 amplitude attenuation at a year or more after initial injury (36,37). This further supports the meaningful role of the P300 as a longitudinal monitoring tool to current monitoring procedures.

Population and Mechanism of Injury

Differences seen in P300 findings may be attributed to the underlying mechanism of injury. SRC showed the most consistent findings, likely reflecting the homogeneity of injury type, younger age, and availability of pre-injury baseline data compared to non-sport acute and subacute studies. A greater variation in MOI existed in the non-sport populations, from mine blast injury, motor vehicle or road traffic accidents, falls, assault, to general concussion population not specified (7–9,11,12,26,28,31–33,36–39). Distinct and significant persistent P300 amplitude abnormalities in blast injury populations likely reflects the unique pathophysiology of blast concussion. Blast injury has an additional pressure wave component not accounted for in other concussion injury mechanisms. There was also concurrent acoustic/barotrauma in 92% of their patients, which complicates direct comparison to other populations (8). Auditory system damage may independently contribute to auditory P300 abnormalities, given the auditory stimulus which is used to elicit the P300 response. In contrast, Vander Werff and Rieger found P300 abnormalities independent of noise condition or auditory processing deficits. This may be suggestive that in non-blast populations, the P300 deficit may reflect broader cognitive attentional disruption which is robust to auditory interference. This supports the feasibility of

point of care testing in a sporting environment, where a silent lab environment may not be available to complete assessments.

Diagnostic and Prognostic Utility

The diagnostic and prognostic utility of the P300 in concussion is context dependent and nuanced. P300 abnormalities (mostly amplitude reduction) are consistent with concussion at the group level. However, it is difficult to translate this difference to a standard diagnostic threshold, given individual variability in the response. Clayton et al. reported an receiver operating characteristic area under the curve (AUC) of 0.92 for P300 amplitude change from baseline in classifying concussion relative to controls. This was mildly improved to an AUC of 0.95 with the addition of reaction time measures. While promising, this is only applicable in contexts where preinjury data can be collected.

The most important prognostic evidence comes from Cavanagh et al., whose finding of the sub-acute P3b amplitude predicted symptom recovery trajectory within a two-month span adjusting for demographic predictors. This suggests the P300 could be used as a tool for identifying risk for prolonged recovery rather than confirming concussion presence (7). This has direct implications on triaging, monitoring, and referral decisions for clinical practice. In comparison, many studies found no group level differences despite clinical symptomology, which may reflect the limits of the paradigm, population heterogeneity, and the possible obscurity of individual variation not seen at the group level (32,33,39). These null findings reinforce the argument that the P300 is not a binary diagnostic test, but a continuous signal whose clinical meaning depends on how, when, and in whom we are measuring.

Unique EEG spectral and coherence measures were found in Mortazavi et al. (2023) in

the PPCS subgroup. This suggests that integrating the P300 as part of a multimodal neurological assessment might improve clinical utility. Reinforcing this idea, Nandrajog et al. (2017) found P300 latency abnormality in the acute population even when neuropsychological tests were normal. Complementary integration of ERPs with behavioural assessments and symptom reporting may be optimal for detecting concussion, monitoring treatment efficacy, and predicting recovery. Taken together, the evidence reported in this review suggests the P300 may be more valuable as a complementary measure rather than a replacement for current clinical evaluation standard of practice.

Clinical and Practical Implications

The findings of this review offer current and future implications for clinicians and physician assistants involved in concussion assessment and management. Current practice relies heavily on subjective tools and symptom checklists, the SCAT5, and neuropsychological testing (5). These evaluations are susceptible to underreporting, and potentially act independently from neurophysiological disruption. The P300 offers a non-invasive, objective, and increasingly portable complement to current tools, capable of detecting functional brain changes in patients whose CT, MRI, and EEG are unremarkable (9,12,39). Persistent abnormalities in the P300 ERP beyond clinical clearance challenges current return to activity protocols in all populations (2,20,35). Moreover, the P300 ERP may aid in cases where subjectivity complicates medicolegal disputes. In studies which evaluated post traumatic headache patients, many were involved in insurance cases, where the ability to quantify their impairment in the absence of imaging carries practical importance (12). Beyond diagnosis, Oakley et al. demonstrated P300 amplitude normalization tracked alongside clinical improvement during multidisciplinary rehabilitation,

supporting its use as a monitoring tool for primary care/outpatient practice (37). While Gaetz and Weinberg demonstrated that a multimodal EP/ERP battery of auditory and visual components was able to detect functional disruption in 17 of the 20 PPCS patients, they found the P300 was the least sensitive measure (39). This should caution over reliance on a single ERP paradigm in clinical evaluation. Furthermore, practical barriers remain such as cost, training, variability in ERP techniques and stimuli, and the absence of validated normative databases. Future technology is emerging, enabling portable and simplified P300 extraction which may make clinical integration of ERP measures following concussion practical.

Limitations

Several limitations of this narrative review must be acknowledged. As with all narrative reviews, the findings are subject to interpretation bias. There were 19 studies included, and the variability of concussion definition, study design, ERP methodology, population traits, and timepoints assessed limits the confidence of comparison across studies. Small sample sizes in included studies reduce statistical power, increasing the risk of type II error. SRC findings yielded mostly consistent findings that may not generalize across non-sporting populations with different mechanisms of injury. The lack of prospective cohort studies investigating neurophysiological changes from pre- to post-injury limits the strength of evidence presented in this review. Evidence from blast injury populations is likely confounded by concurrent auditory system damage. Finally, while the scope of this review was restricted to the auditory P300, other ERP components elicited with different types of stimuli may also relate to concussion diagnosis and recovery.

Future Directions

There are a few priority areas of research that emerge from this narrative review. Firstly, standardization of ERP acquisition and analysis is an important need, consistent with the guidelines proposed by Duncan et al (27). Furthermore, longitudinal studies in civilian concussion cohorts will allow for within person reference data and bridge a critical methodological gap. A multimodal approach exploring the complementary ERP components is warranted. Further investigation of source localization and EEG spectral and coherence analysis which may complement P300 amplitude and latency assessment is warranted.

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Finally, this review reflects a genuine passion I have for improving the lives of those navigating traumatic brain injuries without the objective tools these patients deserve. I look forward to the future of portable EEG technology in clinical practice, and am grateful to have played a small part in bringing better care to this patient population alongside fellow clinicians.

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APPENDIX A

Medline Search Strategy with yielding results:

1	Brain Concussion/	13246
2	Post-Concussion Syndrome/	1899
3	Craniocerebral Trauma/	24290
4	Brain Injuries/ or Brain injuries, Traumatic/	74139
5	Brain Injury, Chronic/	802
6	(concussion* or concussed or concussive or subconcuss* or sub-concuss*).tw,kf.	15387
7	(post-concuss* or postconcuss*).tw,kf.	4670
8	(mild adj3 (traumatic brain injur* or tbi*)).tw,kf.	10325
9	mtbi*.tw,kf.	4985
10	(head adj2 (injur* or impact* or trauma*)).tw,kf.	41855
11	(neurotrauma* or "neuro-trauma*").tw,kf.	3213
12	(traumatic brain injur* or tbi*).tw,kf.	68935
13	(craniocerebral adj2 (trauma* or injur*)).tw,kf.	3577
14	evoked potentials/ or event-related potentials, p300/ or evoked potentials, auditory/	78088
15	(N100 or n1a).tw,kf.	4816
16	(N200 or n200 or n2a or n2b or n2c).tw,kf.	5614
17	(P200 or p2a or p2b).tw,kf.	2067
18	(P300 or p3a or p3b).tw,kf.	15883
19	(ERN or Error-related negativity or Error related negativity).tw,kf.	2043
20	N400.tw,kf.	3705
21	(PMN or Phonological Mismatch Negativity).tw,kf.	15849
22	(MMN or mismatch negativity).tw,kf.	4828
23	(LPP or late positive potential).tw,kf.	3987
24	1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9 or 10 or 11 or 12 or 13	156992
25	14 or 15 or 16 or 17 or 18 or 19 or 20 or 21 or 22 or 23	120197
26	24 and 25	993

Embase Search Strategy:

1	brain concussion/ or concussion/	19999
2	postconcussion syndrome/	3994
3	craniocerebral trauma/	58552
4	traumatic brain injury/	81348
5	(concussion* or concussed or concussive or subconcuss* or sub-concuss*).tw,kw.	21572
6	(mild adj3 (traumatic brain injur* or tbi*)).tw,kw.	14760
7	mtbi*.tw,kw.	8068
8	(head adj2 (injur* or impact* or trauma*)).tw,kw.	53096
9	(traumatic brain injur* or tbi*).tw,kw.	107047
10	(neurotrauma* or neuro-trauma*).tw,kw.	4826
11	(craniocerebral adj2 (trauma* or injur*)).tw,kw.	3583

12 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9 or 10 or 11	203457
13 evoked response/	43047
14 (N100 or n1a).tw,kw.	10931
15 (N200 or n2a or n2b or n2c).tw,kw.	9274
16 (P200 or p2a or p2b).tw,kw.	2811
17 (P300 or p3a or p3b).tw,kw.	20618
18 (ERN or Error-related negativity or Error related negativity).tw,kw.	2970
19 N400.tw,kw.	4944

APPENDIX B

Author (Year)	Study Design	Population	N (Concussed/Control)	Timepoints	P300 Amplitude	P300 Latency	Main Takeaway
Sport-Related Concussion (SRC)							
Gosselin et al. (2006)	Cross-sectional	SRC – Symptomatic & Asymptomatic Athletes	20 / 10	~5 weeks post-injury	↓ ($p < 0.05$)	↑ central electrode (Cz) only ($p < 0.05$)	Amplitude reduced in both symptomatic and asymptomatic athletes. Challenges symptom-based return to activity criteria.
Ruiter et al. (2020)	Cross-sectional	SRC – Adolescent Athletes	26 / 28	~20 days post-injury	↓ ($p < 0.01$)	No significant difference	Significant amplitude reduction in concussed adolescents suggests detectable neurophysiological deficits post-concussion.
Clayton et al. (2020)	Longitudinal Cohort	SRC – NCAA & High School Athletes	45 / 319	Baseline, Acute, RTP	↓ acute & RTP ($p < 0.05$)	No significant difference	P300 abnormalities persisted in 38% of athletes at RTP despite clinical clearance and normalized neuropsychological scores.
Mortazavi et al. (2023)	Longitudinal Cohort	SRC – Adolescent Athletes	33 acute / 63 PPCS / 120 controls	Baseline, Acute, RTP, PPCS	↓ acute, RTP & PPCS ($p < 0.05$)	↓ PPCS only*	Persistent amplitude reduction across recovery. Distinct electrophysiological profile in PPCS. *Latency finding limited by low signal amplitude.
Fickling et al. (2019)	Longitudinal Cohort	SRC – Junior Ice Hockey Players	12 / 43 baseline	Baseline, Acute, RTP	↑ acute & RTP vs. own baseline ($p < 0.05$)	↓ acutely; normalized at RTP	Amplitude increase interpreted as compensatory neural recruitment. Portable rinkside ERP assessment demonstrated feasible in under 10 minutes.
Acute & Subacute Concussion							
Pratap-Chand et al. (1988)	Prospective Cohort	Acute Concussion – Mixed Mechanism	20 / 20 (age & sex matched)	≤4 days; 1–8 months follow-up	↓ acutely ($p = 0.003$); normalized at follow-up	↑ acutely ($p = 0.002$); normalized at follow-up	Both amplitude and latency normalized with recovery. Supports P300 as marker of acute cognitive disruption.
Ford & Khalil (1996)	Cross-sectional	Subacute/Chronic Concussion – Primarily MVA	54 / 27	30 hours – 1 year	↓ max at Fz ($t = -4.63$)	No significant difference	Significant P300 attenuation with unremarkable neuroimaging. Supports P300 sensitivity over structural imaging.
von Bierbrauer & Weissenborn (1998)	Prospective Cohort	Acute Concussion – Mixed Mechanism	15 / 145	24hr, 1w, 3w, 8w	No significant group difference	No group difference; prolonged in ~20% symptomatic subgroup at 24hr; normalized by 3w	Normal group-level values mask individual-level abnormalities. Highlights limits of group comparison designs.
Reza & Begum (2019)	Prospective Cohort	Acute Concussion – Road Traffic Accident	19 / 19	≤7 days; 2–6 months	↑ at T6 electrode only ($p = 0.02$); otherwise NS	No significant difference	Subtle, spatially limited amplitude increase. Interpreted as compensatory neural recruitment following concussion.
Cavanagh et al. (2019)	Prospective Cohort	Subacute Concussion – ED/Neurosurgery	38 / 24 (age & sex matched)	≤14 days; 2-month follow-up	No significant group difference	No significant difference	No group-level P300 differences, but P3b amplitude in sub-acute phase predicted symptom recovery at 2 months. Supports prognostic over diagnostic utility.
Nandrajog et al. (2017)	Prospective Cohort	Acute Concussion – Mixed Mechanism	17 / 19	≤7 days; 2–3 months	↓ at Fz only for standard tones ($p = 0.045$)	↑ acutely ($p = 0.046$); normalized at follow-up	P300 latency prolonged acutely despite normal neuropsychological testing. Reinforces complementary role of ERP alongside behavioral assessment.
Sivák et al. (2008)	Cross-sectional	Acute Concussion – Mixed Mechanism	31 / 31	2–4 days; 3–7 months follow-up	No significant difference	No significant difference	Standard auditory P300 failed to detect changes even in patients with MRI-confirmed traumatic lesions. ERP and MRI measure distinct aspects of brain function.
Reza et al. (2025)	Prospective Cohort	Acute Concussion – Road Traffic Accident	19 / 19	≤7 days; 2–6 months	↑ P300 dipole intensity ($p < 0.001$) via sLORETA	Not reported	sLORETA source localization detects increased dipole intensity not captured by conventional amplitude. Methodology shapes results even in same population.

Chronic Concussion & Persistent Post-Concussion Symptoms (PPCS)							
Solbakk et al. (2000)	Cross-sectional	Chronic Concussion – Symptomatic	20 / 20	≥1 year post-injury	↓ ($F(1,29) = 6.78$, $p < 0.014$)	No significant difference	Persistent amplitude attenuation ≥1 year post-injury in chronically symptomatic patients. Supports longitudinal monitoring role of P300.
Gaetz & Weinberg (2000)	Cross-sectional	Chronic Concussion – Symptomatic (PPCS)	20 / 43	Mean ~38 months post-injury	No significant group amplitude reported; abnormal in few individuals	Non-significant prolongation in 2/10 individuals	Auditory P300 among least sensitive measures in multimodal battery. Visual P300 and CNV considerably more sensitive. Caution against single-paradigm reliance.
Alberti et al. (2001)	Cross-sectional	Post-Traumatic Headache – Chronic Concussion	25 / 18 (age matched)	3–6 months post-injury	No significant difference	↑ ~30ms ($p < 0.001$); abnormal in 52%	Isolated latency prolongation with normal MRI in 52% of patients. P300 more sensitive than structural neuroimaging in this population.
Vander Werff & Rieger (2019)	Cross-sectional	Chronic Concussion – Symptomatic	32 / 32	3–18 months post-injury	↓ vs. controls ($p < 0.05$); direction variable across individuals	No significant difference	P300 abnormalities independent of auditory processing deficits, reflecting broader cognitive-attentional disruption robust to noise condition.
Chebotariova et al. (2020)	Cross-sectional	Chronic PPCS – Mine Blast Injury	115 / 30	0.5–3 years post-injury	↓ ~60% vs. controls	↑ ~45–50ms vs. controls	Most severe P300 abnormalities in review. 92% had concurrent acoustic/barotrauma — complicates auditory P300 interpretation in blast populations.
Oakley et al. (2025)	Retrospective Cohort	Chronic PPCS	64 / 70	Chronic; follow-up through symptom resolution (~20 months)	↓ vs. controls ($p < 0.001$); ↑ with rehabilitation ($p < 0.05$)	No significant difference	P300 amplitude normalization tracked alongside symptom resolution during rehabilitation. Supports P300 as treatment monitoring tool.

Summary of included studies that examined the auditory P300 event-related potential component following concussion. Studies are grouped by population and mechanism of injury: sport-related concussion (SRC), acute and subacute concussion, and chronic concussion and persistent post-concussion symptoms (PPCS). Amplitude and latency findings reflect comparisons to pre-injury baseline or healthy control values unless otherwise noted.