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Impaired auditory processing and neural representation of speech in noise among symptomatic post-concussion adults

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Abstract

Background: The purpose of the study was to examine auditory event-related potential (AERP) evidence of changes in earlier and later stages of auditory processing in individuals with long-term post-concussion problems compared to healthy controls, with a secondary aim of comparing AERPs by functional auditory behavioral outcomes.

Methods: P1-N1-P2 complex and P300 components recorded to speech in quiet and background noise conditions were completed in individuals with on-going post-concussion symptoms following mTBI and healthy controls. AERPs were also examined between sub-groups with normal or impaired auditory processing by behavioral tests.

Results: Group differences were present for later stages of auditory processing (P300). Earlier components did not significantly differ by group overall, but were more affected by noise in the mTBI group. P2 amplitude in noise differed between mTBI sub-groups with normal or impaired auditory processing.

Conclusion: AERPs revealed differences between healthy controls and those with chronic post-concussion symptoms following mTBI at later stage of auditory processing (P300). Neural processing at the earlier stage (P1-N1-P2) was more affected by noise in the mTBI group. Preliminary evidence suggested that it may be only the proportion of individuals with functional evidence of central auditory dysfunction with changes in AERPs at earlier stages of processing.

Keywords

mild traumatic brain injury; cortical auditory evoked potential; auditory processing; speech in noise

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INTRODUCTION

Evidence has accumulated that a significant minority of individuals experience persistent long-term deficits following mild traumatic brain injury (mTBI) or concussion [1-4]. Among the chronic problems, studies have documented auditory dysfunction following both non-blast and blast-related mTBI. Despite the fact that few of these individuals have hearing outside the normal range on a clinical audiogram, a significant percentage report difficulty understanding speech in background noise as well as difficulty on other complex auditory tasks, such as understanding rapid speech or detecting small gaps in noise [5-13].

Difficulties with these tasks may result from damage to structures in the central auditory pathway which may be especially vulnerable to mTBI, including auditory brainstem nuclei, structures in the left temporal lobe, pathways between the thalamus and primary auditory cortex, the planum temporale, and the corpus callosum [14-16]. Identifying whether there is electrophysiological evidence of changes in how speech information is represented along the central auditory pathway and whether behavioral deficits correlate with these neural representations is of interest to better understand the long-term auditory consequences of mTBI.

Auditory event-related potentials (AERPs) provide important tools to investigate auditory processing abilities following mTBI, as they are time-locked measures of electrical activity representing distinct phases of processing of sound, particularly speech. From the most peripheral levels in the cochlea, auditory nerve and brainstem, fine spectro-temporal information critical to speech understanding is precisely encoded in these obligatory neural responses. We previously reported electrophysiological evidence using the auditory brainstem response (ABR) to speech stimuli that subcortical processing of auditory information was affected by mTBI [17], in agreement with other studies in both children and adults [18; 19]. These results suggest that neural encoding of acoustic information and not just top-down cognitive processing of auditory information, can be impaired in some individuals with concussion. Beyond the subcortical level, the P1-N1-P2 waveform complex, called the cortical auditory evoked potential (CAEP) represents arrival of sensory information at the level of the auditory cortex and the beginning stages of central auditory processing.[see review 20]. While the N1 and P2 are sensitive to early attention [21], the passive P1-N1-P2 complex is generally considered an obligatory, exogenous evoked potential and has been shown to reflect sensory or acoustic-level processing of onsets and acoustic feature changes within a speech signal [20; 22-29]. Prior to the level of active discrimination, the auditory P1-N1-P2 can provide insight into deficits in central auditory processing by indicating whether important spectral and temporal features of sounds, particularly speech, have been properly encoded up to the level of the cortex. For instance, using various speech stimuli and background noise conditions, studies have shown that CAEPs can reflect aspects of difficulty understanding speech in a background of noise [30-37].

P300 event-related potentials reflect a later or higher order stage of auditory processing and can be elicited by auditory discrimination tasks in an oddball paradigm. In an active oddball paradigm, auditory P300 responses are related to the ability to perceive, attend to, and discriminate between auditory stimuli. Both central auditory dysfunction and general

cognitive impairments, therefore, may influence P300 outcomes. In both visual and auditory domains and across a range of head injury severities, the most frequent significant ERP findings in various fields of research have been reductions in the P300 component amplitude [38; 39]. Reduced P300 amplitude in those with mTBI could reflect fewer cognitive resources available to evaluate acoustic differences in stimuli, or a change in attentional allocation, while latency may reflect changes in the speed of processing of auditory information [38]. While few studies have specifically recorded passive obligatory CAEPs to examine post-concussion processing, many studies have used the cognitive auditory P300 ERP, which typically requires active participation of the listener, to examine questions about cognitive effects of TBI. Gallun et al. [5] used a typical auditory tone oddball paradigm to record the P300, but analyzed both the earlier peaks (N1 and P2) generally considered to be part of the CAEP exogenous response as well as the P300 endogenous component in blast-exposed veterans, including those diagnosed with mTBI. They found significant differences in P300 amplitude between the blast-exposed and control groups, as well as a significant difference in N1 amplitude, though only for the left ear in the deviant stimulus condition. While not all mTBI studies have shown significant effects on P300 [40-42], a number have demonstrated significant changes in P300 latency and/or amplitudes [43-51].

In the current study, we used both the CAEP and the P300 AERPs to examine earlier and later central auditory processing of speech information in a group of adults who were reporting long-term post-concussion problems after a single mTBI. Further, to specifically correlate with common reports and evidence of difficulty understanding speech in noise following mTBI, we recorded both types of neural responses to speech stimuli in a background of noise that consisted of other people talking. We compared the AERP outcomes to a group of age- and sex-matched controls with no history of head injury. These data were collected as part of a larger study, and results were analyzed in context with performance outcomes on a battery of behavioral auditory processing tests. We predicted that the group with long-term problems following mTBI would demonstrate attenuated or delayed AERP components compared to healthy un-injured controls, at least under more difficult listening conditions, i.e. in background noise. Finally, as a secondary exploratory aim, we examined whether individuals with measurable performance deficits on auditory behavioral tests following mTBI would have larger reductions in both sensory (P1, N1, P2) and cognitive (P3) components than those with mTBI who performed within normal ranges on such assessments of auditory processing abilities.

METHODS

Participants

Thirty-two individuals diagnosed with mTBI (22 female; mean age 42.3 ± 13.1 years, range 19–61) and 32 healthy individuals (22 female; mean age 41.2 ± 12.7 years, range 18–60) without any history of concussion requiring medical intervention participated in the study. Individuals in the mTBI group were recruited prospectively from the patient population of a local outpatient brain injury rehabilitation and concussion management program. Potential participants referred for study inclusion were between 3–18 months post-injury and continued to report multiple on-going post-concussion symptoms to the extent that they were

participating in rehabilitation. This time range was targeted because symptoms prior to three months may be transient and post-concussion syndrome is often defined as symptoms persisting beyond this time [52; 53]. A maximum of 18 months was chosen because symptoms beyond this time would be more difficult to ascribe to a specific injury event. The majority of participants were tested within 9 months of injury (mean 6.8 ± 4.0 months, median 5.1 months). Reported on-going symptoms were not required to be auditory in nature, and included physical symptoms such as headache, dizziness, light and sound sensitivities as well as poor concentration, irritability, fatigue, anxiety, and depression, rates of which are reported in Vander Werff and Rieger [13]. All were diagnosed with mTBI consistent with criteria from the American Congress of Rehabilitation Medicine [54]. All participants were civilians with no history of blast injury. The majority of injuries were due to motor vehicle accident (38%), falls (22%), sports and recreation (9%), assault (9%), and other blows to the head (22%).

Participants were excluded if they had evidence of more than minimal hearing threshold loss, defined as average hearing thresholds (1.0, 2.0 and 4.0 kHz) greater than 49 dB hearing level (HL) [55], evidence of middle ear pathology, or air-bone audiometric differences. Pre-injury history of neurological disorder, previous head injury requiring medical care, diagnosed learning disability or attention disorder, or history of severe psychiatric disorder were also exclusionary factors. Control participants had no history of head injury but met the same additional inclusion/exclusion criteria and were recruited to achieve age and sex matching across groups. Of screened potential participants, 6 did not meet inclusion criteria from the control group and 5 did not meet inclusion criteria for the mTBI group. The Institutional Review Boards of Syracuse University and Upstate Medical University approved all procedures and participants were monetarily compensated for their time.

Procedures

All participants completed audiometric evaluation and case history interview, including symptom questionnaires of auditory and post-concussion symptom rating. A battery of behavioral tests of central auditory processing abilities and neuropsychological tests of cognitive performance were completed on all participants in both the mTBI and control groups. Detailed methods and results of these evaluations are presented in a companion paper [13], but a summary of raw scores and group outcomes for all cognitive and behavioral tests is shown in Table 1. Cognitive function was assessed using a neuropsychological test battery which included the California Verbal Learning; Test [CVLT-II; 56]; the Digit Span (DS) subtest of the Wechsler Adult Intelligence Scale [WAIS-IV; 57]; the Trail Making Test [TMT; 58]; the Symbol Digits Modality Test [SDMT; 59]; and the Design Fluency Test (DFT), Verbal Fluency Test (VFT) and Color-Word Interference Test (CWIT) subtests of the Delis-Kaplan Executive Function System [D-KEFS; 60]. The Medical Symptom Validity Test (MSVT) was also administered as a test of effort [61, 62]. Four participants in the mTBI group failed the criteria for sufficient test effort on the MSVT. Because insufficient effort would affect behavioral test performance on the auditory tests and likely the P300, these four were excluded from the analyses in the current study and the sample size for the mTBI group was 28, compared to the control group of 32. While raw scores on most of the individual neuropsychological tests were slightly and significantly

poorer for the mTBI group on most of the individual cognitive tests, the rate of abnormal performance compared to standard cut-offs was not significantly different between groups.

The auditory test battery included: 1) monaural word recognition in quiet (WRQ), 2) binaural word recognition in a constant background of noise at a +10 signal-to noise ratio (WRB+10), 3) time-compressed speech recognition (TC, 65% compression), 4) speech reception in noise threshold using the Words in Noise test (WIN, 63; 64), 5) gap detection using the Gaps in Noise test (GIN, 65), and 6) dichotic listening using the Staggered Spondaic Word test (66). The first 3 tests used NU-6 lists [67]. The background noise for the constant +10 signal-to-noise ratio (SNR) condition was 20-talker babble pre-recorded on the second track of the same CD as the NU-6 words (Auditec Inc., St. Louis, MO, USA) and was the same babble used for the background noise condition for the ERP testing. All auditory behavioral tests were completed with the participant seated in comfortable chair in a sound-treated audiometric booth (ambient noise levels < 15 dB SPL at octave frequencies from .125 to 8.0 kHz). Tests were performed and scored according to the standard instructions for each published test, and were conducted at comfortable listening levels of either 50 dB sensation level or 60 dB HL. For our secondary question, those in the mTBI group who performed normally on the auditory behavioral test battery as a whole were considered to have Normal Auditory Processing (NAP), defined as no more than one abnormal score on the battery of seven tests; $n = 19$). Those in the mTBI group with performance outside the normal range on two or more auditory behavioral tests were considered to have evidence of abnormal auditory processing (mTBI Impaired Auditory Processing IAP; $n = 9$). There were no members of the control group who met the criteria for IAP, therefore the control group was not sub-divided in the analyses. It is interesting to note from Table 1 that rates of abnormal performance on auditory behavioral tests exceeded rates of abnormal performance on the cognitive test battery.

Electrophysiological recordings of CAEP and P300 were conducted in the same audiometric test booth with participants seated in a comfortable chair. A PC-based system was used for stimulus presentation and acquisition (Compumedics Neuroscan, Charlotte, NC, USA). EEG activity was recorded using a subset of electrodes on a 64-channel Compumedics Neuroscan Quik-Cap including frontal (F3, Fz, F4), central (C3, Cz, C4), parietal (P3, Pz, P4) and temporal (T7, T8) sites using the international 10–20 system [68]. Linked mastoids served as reference. Eye movement was monitored with electrodes located above and below the left eye. EEG was recorded from 0.1 to 100 Hz, and amplified with a gain of 10 and sampling rate 1 kHz. Artifact rejection ($\pm 100 \mu\text{V}$) and reduction of vertical blink artifacts were completed prior to averaging of trials and off-line filtering from 1–30-Hz. Data were epoched from 100 ms pre-stimulus to 1000 ms post-stimulus.

Stimuli were naturally produced speech syllables of “ba” and “pa” from the Nonsense Syllable Test (NST, Auditec Inc., St. Louis, MO) recordings. Syllable durations were edited to 300 ms by truncating at vowel zero crossing and exponential offset ramping. Stimuli were delivered binaurally using the Neuroscan Stim2 system at 75 dB SPL via ER-3A insert earphones. For the CAEP, stimuli were presented with 1216 ms inter-stimulus interval (ISI) with equal probability for “ba” and “pa” (50%) with approximately 200 presentations of each so that responses could be recorded to both stimuli in one session. Participants were

seated upright and asked to remain alert and still. They were instructed to ignore the stimuli and were allowed to read during the recording. For the P300, the same stimuli were presented with “ba” serving as the frequent (80%) and “pa” as the infrequent (20%) in an oddball paradigm. Stimuli were presented with 1516 ms ISI and approximately 100 presentations minimum of the infrequent stimulus. Participants were asked to press a button for the infrequent stimulus. A stable visual target was provided for focus and minimize ocular movement during the task. All participants were easily able to discriminate between the target and rare stimuli in practice.

For both CAEP and P300, responses were recorded under two conditions, quiet and background noise. The noise condition consisted of presentation of continuous speech noise for the entire duration of the recording of that specific condition. Speech noise was a recorded multi-talker babble consisting of 20 separate simultaneous talkers. Speech stimuli and noise were presented to both ears through the same headphones via a sound mixer with the noise at 65 dB SPL (+10 dB SNR). Total recording time after electrode application and preparation was approximately 45 minutes. The order of conditions was randomized and short breaks were offered between conditions.

Averaged recordings were analyzed offline. Identified and analyzed waves included P1, N1, and P2 for the CAEP and P3 for the P300. The P1-N1-P2 complex was analyzed at the Cz electrode, while P3 data were analyzed at the Pz electrode, where responses were maximal. Peaks were identified visually by at least two observers using defined latency time windows based on previously published data and using the global field power (GFP) waveform and were based on agreement of two judges. Peak amplitudes were calculated baseline to peak as the maximum voltage in the time window and peak latencies were determined for this point relative to stimulus onset. The sensitivity index d' prime (d') was calculated for the ability to discriminate infrequent from frequent stimuli using the formula $d' = Z_{\text{HIT}} - Z_{\text{FA}}$ where HIT represents the proportions of hits when the signal is present (hit rate) and FA represents the proportion of false alarms when a signal is absent, the false alarm rate.

Amplitude and latency for each peak, as well as reaction time and d' for the P300, were each separately analyzed using two-way mixed model analysis of variance (ANOVA) to test for group (mTBI vs. control) condition (quiet vs. noise) effects. Exact p-values for each of the ten separate ANOVAS are provided, corrections for multiple comparisons were not applied. The outcome of a battery of central auditory behavioral tests was used to group the participants with mTBI into subgroups with and without auditory performance deficits. As an exploratory analysis, differences in peak amplitudes, latencies, and reaction times between these sub-groups, and between each mTBI subgroup and the control group were assessed using independent t-tests. Where applicable, results were adjusted if Levene's test indicated that the assumption of equal variance between groups was violated.

RESULTS

The results for the main question of whether there were overall group differences in earlier or later auditory evoked potential components between the mTBI group as a whole (mTBI All) and the control group are displayed in grand mean waveforms in Figures 1 and 2.

Means of individually identified amplitudes and latencies for both groups in quiet and noise conditions are shown in the bar graphs in Figures 3 and 4. The secondary question of whether the auditory evoked potentials might differ between those in the mTBI group with either normal auditory processing (NAP) or impaired auditory processing (IAP) based on the auditory behavioral test outcomes is also addressed in the bar graphs in Figures 3 and 4, as well as in the grand mean waveforms in Figures 5 and 6.

Figure 1 shows grand mean waveforms for the earlier passive CAEP for the controls and mTBI group by electrode location for both the quiet and noise conditions, illustrating that the P1-N1-P2 complex was largest at the Cz electrode and decreased slightly in amplitude for the frontal, parietal and hemispheric locations. Presence of 20-talker babble in the noise condition resulted in delayed peak latencies, particularly observed for N1 and P2, with reductions in P2 peak amplitude in the grand mean waveforms. Similar trends between quiet vs. noise conditions are observed for the control (grey) and mTBI (black) waveforms, though some visual group differences are noted, such as a smaller N1 amplitude for the mTBI group in both quiet and noise conditions. Figure 2 shows the same display format for the P300 deviant (infrequent) waveform grand means by electrode location for each group and condition. These grand mean waveforms suggest smaller amplitudes in both quiet and noise conditions for the mTBI group compared to controls, with a larger shift in P3 latency between quiet and noise conditions for the mTBI cohort.

Peak amplitudes and latencies from individual waveforms for each participant in each condition were averaged and analyzed by group. Bar graphs in Figure 3 display mean and standard deviation of peak amplitudes and latencies for the P1, N1, and P2 peaks of the CAEP for the control group and mTBI group as a whole (mTBI All). The bars for the NAP and IAP sub-groups are discussed below in relationship to Figures 5 and 6. As shown by the mixed model ANOVA results in Table 2, the main effect of condition was significant for the P1, N1 and P2 peaks with smaller amplitudes and longer latencies in noise compared to quiet, with the exception of P1 amplitude. The main effect of group between controls and all mTBI participants did not reach significance for any of the P1, N1, or P2 amplitudes or latencies, which was contrary to our hypothesis. There was, however, a significant interaction effect between group and condition for P2 amplitude ($p = 0.045$). That is, while the presence of background noise reduced P2 amplitude for both groups, it resulted in a larger reduction in amplitude for the mTBI group compared to controls. Similarly, there were significant group by condition interactions for P1, N1 and P2 latencies ($p = 0.017$, 0.028 , and 0.021 respectively), indicating that noise caused a larger latency delay for the mTBI group than for controls for these primarily sensory components.

In the same format of the previous figure, Figure 4 shows mean P3 amplitudes and latencies for the mTBI and control groups in quiet and noise, as well as the mean measured behavioral reaction time for each group. ANOVA results (Table 2) revealed that the main effect of condition was significant for all three variables. In addition, there was a significant overall main effect of group for the P3 amplitude ($p = 0.033$) as well as reaction time ($p = 0.007$) and d' ($p < 0.0005$). That is, the mTBI group as a whole had significantly reduced P3 amplitudes, longer reaction times, and poorer discrimination between standard and deviant targets overall compared to the control group.

Grand mean waveforms for the sub-groups of mTBI participants with NAP and IAP are shown compared to the control and overall mTBI groups means in Figure 5 for the CAEP (at Cz) for the quiet and noise conditions. While the grand mean waveform for the mTBI group as a whole had similar morphology to that of the controls, when divided by functional auditory performance a slightly different pattern for the IAP subgroup can be observed. While the amplitude of P2 appears to be reduced in quiet in the mTBI IAP sub-group compared to either controls or those in the mTBI with NAP, these differences did not reach statistical significance ($p > 0.05$). However, P2 amplitudes in noise were significantly smaller for the IAP compared to the NAP mTBI sub-groups ($t = 3.148$, $df = 26$, $p = 0.004$). T-tests also showed that N1 latencies were significantly longer in the mTBI NAP sub-group compared to controls in the quiet ($t = 2.101$, $df = 45$, $p = 0.041$) and noise ($t = 2.087$, $df = 48$, $p = 0.042$) conditions. None of the differences between the controls and the mTBI IAP sub-group reached statistical significance.

P300 ERP waveforms at electrode Pz for the control and overall mTBI groups as well as the NAP and IAP sub-groups are shown in Figure 6. Waveforms are shown for both the standard (thin lines) and deviant stimuli. Unlike the CAEP, general morphology differences are observed in the P3 response between the controls and the mTBI group as a whole, with less observable difference in waveforms between the sub-groups of mTBI participants. The presence of noise reduced the amplitude and increased P3 latency in all groups, but more so for the mTBI participants as a whole than for controls. Preliminary statistical analyses showed that P3 amplitude was significantly reduced for the NAP sub-group compared to controls in the quiet condition ($t = 2.161$, $df = 48.419$, $p = 0.036$), but no other P3 amplitude or latency comparisons between groups were significant by *t*-test for either quiet or noise. In terms of the behavioral outcomes of the P300 paradigm, control participants had significantly faster reaction time than the NAP sub-group in noise ($t = 2.759$, $df = 49$, $p = 0.007$) and the IAP sub-group in either quiet or noise ($t = 2.579$, $df = 39$, $p = 0.014$; $t = 2.297$, $df = 39$, $p = 0.027$). However, reaction time was not significantly different between mTBI sub-groups for either quiet or noise ($t = 0.767$, $df = 26$, $p = 0.450$; $t = 0.271$, $df = 26$, $p = 0.788$ respectively). Similarly, control participants had significantly better d' scores compared to the mTBI NAP and IAP groups in quiet ($t = 2.189$, $df = 49$, $p = 0.033$; $t = 2.552$, $df = 39$, $p = 0.016$ respectively) and noise ($t = 3.425$, $df = 49$, $p = 0.001$; $t = 4.225$, $df = 39$, $p < 0.0005$ respectively). D' scores did not differ significantly between the NAP and IAP groups in either quiet ($t = 0.210$, $df = 26$, $p = 0.836$) or noise ($t = 0.410$, $df = 26$, $p = 0.685$).

In summary, there was some evidence that individuals with mTBI and long-term post-concussion symptoms had changes in neural representation of speech at both earlier and later stages of auditory processing as evidenced by the morphology of CAEP and P300 waveforms. At the earlier primary sensory cortical level, the CAEP showed few significant overall group differences, but there was a significant group by noise condition interaction, indicating that those with mTBI were more adversely affected by background noise at least as represented by P2 amplitude. Grand mean waveforms give some indication that there was a relationship between auditory behavioral test outcome and waveform morphology, although only the P2 amplitude in noise was statistically significantly different between the two mTBI sub-groups. On the other hand, the cognitive level auditory P300 amplitude was

affected by both group and condition, but with no statistically significant sub-group difference by auditory performance in P3 amplitude or latency. Behavioral measures of RT and d' varied significantly between controls and either mTBI sub-group, but not between the two sub-groups with normal and impaired auditory processing.

DISCUSSION

Because of accumulating evidence that mTBI can affect central auditory pathway and auditory processing abilities, especially speech understanding in noise, we hypothesized that there would be group differences in auditory event related potentials recorded to speech stimuli at least in the more challenging background noise condition. We also hoped to determine whether such group differences would be evident at both earlier and later stages of auditory processing by recording the passive onset CAEP and the active oddball P300. If differences were only observed for the P300, it's possible that they may not be specific to auditory processing but rather reflect more general top-down cognitive changes. We further predicted that changes in these waveforms would be more evident in those individuals with mTBI who had measurable functional performance deficits on auditory behavioral tests than those with mTBI who performed within normal ranges on these assessments.

Overall, our hypotheses were only partially supported. At the earlier, primarily sensory level, although some differences in the grand mean waveforms were observed, there were no significant overall group effects for any of the peak amplitudes or latencies. There were, however, significant group-by-condition interactions for P2 amplitude and all three peak latencies. The presence of background noise, therefore, was more detrimental for the neural representation of speech at this sensory cortical level for those with mTBI than for the control group. Few previous studies have included CAEP comparisons between mTBI and healthy control groups, although Gallun et al. [5] reported a significant reduction in N1 amplitude analyzed as part of their P300 paradigm in one ear of participants with blast injury compared to un-injured controls. Their result was also only in quiet, and the current results suggest that group differences may be more apparent only under more difficult listening tasks, i.e. in noise. This finding agrees with the behavioral literature which has generally shown differences between mTBI and control groups for speech understanding in noise, but not in quiet [5; 8; 10; 11; 13].

It may be reasonable that only individuals with actual functional deficits in auditory processing would have evidence of differences in the neural representations of speech. The mTBI group in this sample had rates of abnormal performance on the auditory behavioral tests that exceeded their rates of cognitive performance deficits, and the overall rate was significantly different from the control group. However, not all individuals in the mTBI showed had functional auditory processing deficits by these measures, nor were they selected specifically for auditory symptoms. It is possible that overall differences between mTBI and control groups in auditory evoked potential outcomes may be obscured because only part of the group experienced post-concussion auditory dysfunction. As a secondary analyses, therefore, we divided the mTBI group into sub-groups based on auditory behavioral performance including speech-in-noise performance and other complex listening tasks. While these results can only be considered preliminary due to the small and unequal

n-sizes, grand mean CAEP waveforms appeared reduced in amplitude and with slightly different morphology for those who had impaired auditory processing performance. Few of the exploratory statistical test results were significant, likely due to the small n-size when divided into sub-groups, but P2 amplitudes in noise were significantly reduced for those who met the criteria for impaired auditory processing performance compared to the mTBI cohort with normal auditory processing performance. This finding may suggest that not everyone has auditory performance deficits following mTBI, but of those who do, there may be changes in the neural representation of speech at an earlier auditory processing stage.

While there were not overall group differences for the earlier components, there were significant differences at the level of the active, cognitive processing P300 component. P3 amplitude, reaction time, and discrimination ability (d' scores) all differed significantly between healthy controls and the mTBI group as a whole. Noise was clearly detrimental for both groups, and there were no significant interaction effects between group and noise condition for P3 amplitude, latency or RT. There was a significant interaction, however, for the d' score, meaning that individuals in the mTBI group had a larger reduction in detection accuracy in the noise condition than did controls. The preliminary P300 sub-group analyses did not reveal any significant difference between those with normal or abnormal auditory performance for P3 amplitude or latency. RT and d' scores were significantly different between healthy controls and either mTBI sub-group, but not differentiate the two mTBI sub-groups. These P300 results are in general agreement with a number of studies in the literature that have demonstrated significant changes in auditory P300 latency and/or amplitudes following mTBI [43-51], particularly when studies have used more complex stimuli and paradigms (beyond simple frequency changes) [43-46; 69], as was used in the current study. Gallun et al. [5] also found significant differences in P300 amplitude between blast-exposed and control groups, as well as significant differences in behavioral auditory test performance, although they did not analyze their results by sub-group or use background noise.

Overall, the results of the current study suggest that central auditory evoked potential components can be affected in those who report chronic post-concussion symptoms following mTBI, particularly when recorded to speech stimuli in a background of noise. However, results did not clearly establish overall group differences for all components. The later P300 component revealed overall group effects for at least P3 amplitude and the behavioral RT and d' scores. That is, at this level of cognitive auditory processing involving discrimination, attention, and memory, the mTBI group as a whole showed a change in waveform morphology and behavioral discrimination and reaction time, whether or not there were specific measured functional auditory deficits on the auditory test battery. This group difference was not specific to the noise condition, and there were no significant group x condition interactions for P3 amplitude or latency. Noise was detrimental to the waveforms representations of speech for both the mTBI and control groups, perhaps to the extent that group differences could not be detected. While performance on a cognitive test battery did not differ between groups in terms of the rate of abnormal scores, there were significant differences in raw scores on most of the measures [13]. It is possible this overall cognitive difference could account for the P300 group results.

At the earlier primarily sensory level, there were no overall main effects of group on the passively-evoked CAEP. There were, however, group-by-condition interactions showing that noise reduced P2 amplitude and increased all latencies more for the mTBI group than it did for controls. This finding and the difference by sub-groups in P2 amplitude particularly are interesting, indicating that a more complex paradigm (speech in noise) was needed to see the group effect. In addition, averaging together responses from participants in the mTBI group as a whole, including both those with and without auditory processing deficits may have obscured group differences in neural processing between those with mTBI and controls. The small n-size when the mTBI group is divided into sub-groups is clearly a limitation, however, this will be interesting to follow up in future studies. It should also be noted that inclusion criteria for hearing thresholds allowed for some peripheral hearing differences across subjects and between groups. These differences are likely minimal, as there were no significant differences between groups in pure-tone audiometric thresholds at any frequency. There were, however, significantly poorer cochlear distortion-product otoacoustic emission signal-to-noise ratios for the mTBI group in the left ear at two frequencies (1500 and 8000 Hz). The influence of small differences in sensation levels of the stimuli for either the auditory evoked potential tests or the auditory behavioral tests, which are all completed well above threshold ensuring audibility, is likely small but can't be completely ruled out.

There is not a clear division between sensory and cognitive processing, of course, and while the CAEP is generally considered obligatory/exogenous, it has been suggested that the P2 in particular may be influenced by top-down factors such as attention and learning [70; 71]. Given that the P2 was the most reduced by noise and showed the interaction between group and noise condition, as well as the sub-group difference, this is likely an important point. It may be that in the noise condition, though recorded passively, attention and other top-down factors are responsible for any group differences rather than auditory processing abilities alone.

Limitations of the current study include the small sample size, particularly when considering the sub-groups of normal and impaired auditory processing. Statistical analyses were limited and were not corrected for multiple comparisons. However, given the heterogeneous nature of mTBI in general, these results and others in the literature finding significant auditory effects provide evidence that at least in a significant proportion of those who experience concussion have auditory-related dysfunction. Further study and multi-disciplinary clinical attention to auditory problems in this population are warranted.

CONCLUSIONS

Auditory evoked potentials representing earlier and later stages of central auditory processing, the CAEP and P300, revealed some differences in neural representation of speech in noise between healthy controls and individuals with mTBI who remained symptomatic 3–18 months post-injury. CAEP results may be more dependent on the effect of noise and by the functional auditory performance of the individuals in the group. At a higher level, P300 results were seen for the mTBI group as a whole, but were not further separated by noise conditions or auditory processing behavioral test performance. These findings suggest that both earlier and later auditory processing may be affected by mTBI in

the long-term, but perhaps only in a proportion of individuals with chronic symptoms who have functional auditory deficits.

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Figure 1:

Grand average CAEP response waveforms for the control (grey) and mTBI (black) groups by electrode location for the quiet (solid) and noise (dotted) conditions. Approximate locations of the P1, N1, and P2 components are labeled for the Cz waveforms in quiet. The scale shown at bottom left is the same for all panels.

Figure 2:

Grand average P300 response waveforms to the deviant (infrequent) stimulus for the control and mTBI groups by electrode location for the quiet and noise conditions. Approximate location of P3 in the quiet condition shown for the Pz location. See Figure 3 for additional detail. Set up and legend are the same as for Figure 1.

Figure 3:

Mean and standard deviation of amplitudes and latencies for the CAEP peaks for the control and mTBI groups overall in quiet (filled) and noise (open) conditions are shown in the first two sets of bars in each panel. Note that N1 amplitudes are plotted as negative voltages upward for consistency in direction of the bars. The last two sets of bars in each panel show results for subgroups of mTBI participants with normal auditory processing (NAP) and impaired auditory processing (IAP).

Figure 4:
Mean and standard deviation of amplitudes, latencies and reaction time for the P300 AERP for each group/subgroup in quiet (filled) and noise (open) in the same format as in Figure 3.

Figure 5:

Grand average CAEP response waveforms for the /ba/ stimulus for electrode Cz in the quiet (left panel) and noise (right panel) conditions. Thick lines (grey) indicate control group averages while medium thickness lines indicate the entire mTBI group. The subgroup of mTBI participants with normal auditory processing (NAP) are shown by the dotted (black) lines. Thin lines (red) represent the subgroup with impaired auditory processing (IAP). Approximate locations of P1-N1-P2 peaks are labelled for the control group in each panel.

Figure 6:

Grand average P300 ERP response waveforms for electrode Pz in the quiet (left panel) and noise (right panel) conditions. Lines for the groups for the deviant (infrequent) stimulus are the same as described in Figure 3. Thin lines in the same colors represent the standard (frequent) response waveforms for each group. Approximate locations of P3 peaks are labelled for the control group in each panel.

Table 1.

Mean and standard deviation of raw scores on cognitive/neuropsychological and behavioral auditory tests and sub-tests for the control and mTBI groups and the number and percentage of scores in each group that were outside of criteria for normal performance. Full behavioral data appear in Vander Werff and Rieger [13].

		Controls				mTBI			
Cognitive tests		Mean	SD	N*	%	Mean	SD	N*	%
CLVT	Trials 1-5 (T-score)	56.2	8.5	0	0%	51.2	14.2	1	3%
	List B	6.9	1.8	0	0%	5.9	2.3	0	0%
	Short free recall	11.7	2.9	1	3%	10.6	4.0	1	3%
	Long free recall	12.8	3.1	1	3%	10.8	4.3	1	3%
DS	Forward	11.4	1.9	0	0%	9.4	2.6	0	0%
	Backward	8.4	2.3	0	0%	6.9	2.4	0	0%
	Total span	19.8	3.8	0	0%	16.3	4.7	0	0%
TMT	Trail A (s)	17.7	5.2	0	0%	29.2	30.0	1	3%
	Trail B (s)	36.4	13.4	0	0%	68.1	65.3	1	3%
	Total (s)	54.1	16.4	1	3%	97.3	92.5	1	3%
SDMT	Number correct	61.7	9.4	0	0%	46.8	11.6	0	0%
DFT	Filled dots	13.2	3.4	0	0%	9.8	2.8	0	0%
	Empty dots	13.5	2.8	0	0%	10.6	2.3	0	0%
	Switching	9.8	2.6	1	3%	7.6	2.4	0	0%
VFT	Letter	44.3	10.7	0	0%	37.8	12.0	0	0%
	Category	48.1	10.1	0	0%	41.2	9.5	0	0%
	Category Switching	16.1	2.5	0	0%	14.1	3.1	0	0%
	Total Switching Accuracy	15.4	2.6	0	0%	13.6	3.3	0	0%
CWIT	Color (s)	25.9	3.7	0	0%	40.0	28.9	3	9%
	Word (s)	19.7	2.9	0	0%	30.4	21.4	4	13%
	Inhibition (s)	43.3	9.5	0	0%	68.1	34.5	2	6%
	Inhibition/Switching (s)	49.5	7.7	0	0%	73.6	34.9	1	3%
Auditory processing tests		Mean	SD	N*	%	Mean	SD	N*	%
WRQ	Left % correct	97.8	2.3	1	3%	97.5	2.4	1	3%
	Right % correct	98.9	1.5	0	0%	98.6	1.6	0	0%
WRB+10	% correct	87.9	4.3	0	0%	83.1	7.0	4	13%
WIN	Left SNR threshold	4.2	1.4	0	0%	5.8	2.9	6	19%
	Right SNR threshold	4.5	2.0	2	6%	6.1	2.1	5	16%
TC	Left	79.5	7.7	0	0%	68.6	15.1	3	9%
	Right	79.6	9.9	0	0%	71.0	16.8	3	9%
GIN	Left gap threshold	5.6	1.8	3	9%	6.4	1.9	5	16%
	Right gap threshold	5.9	1.5	0	0%	6.8	1.8	3	9%
SSW	Total errors	2.6	2.4	2	6%	7.9	8.4	10	31%

N* and % = number and percentage of participants in each group scoring below normal performance cut-off; WRQ = Word Recognition in Quiet; WRB+10 = Word Recognition Binaural in Babble at a +10 signal-to-noise ratio; WIN = Words in Noise; TC = Time Compressed Speech; GIN = Gaps in Noise; SSW = Staggered Spondaic Word; CVLT = California Verbal Learning; Test – Second Edition; DS = Digit Span Test; TMT = Trail

Making Test; SDMT = Symbol Digits Modalities Test; DFT = Design Fluency Test, VFT = Verbal Fluency Test; CWIT = Color-Word Interference Tests

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Table 2.

Mean and standard deviation of individually picked peak amplitudes and latencies for each component by group and condition with results from the mixed model ANOVAS for the main effects of group (control vs. mTBI) and condition (quiet vs. noise) and the interaction. Significant results ($p < 0.05$) are shown in red/bold.

	Quiet				Noise				Effect of group				Effect of condition				Group × condition interaction			
	Controls		mTBI		Controls		mTBI		F	df	p value	F	df	p value	F	df	p value			
	Mean	SD	Mean	SD	Mean	SD	Mean	SD												
Amplitude (μV)	P1	1.1	0.8	1.0	0.8	1.3	0.6	1.1	0.9	0.65	1,58	0.422	2.56	1,58	0.115	0.56	1,58	0.456		
	N1	-2.2	1.1	-2.1	1.3	-1.9	0.9	-1.6	0.9	0.45	1,58	0.507	7.04	1,58	0.010	0.40	1,58	0.529		
	P2	2.4	1.0	2.0	1.3	0.5	0.7	0.6	0.7	0.56	1,58	0.459	156.82	1,58	0.000	2.70	1,58	0.045		
	P3	4.9	3.0	3.5	2.0	3.2	2.0	2.4	1.3	4.58	1,58	0.033	32.78	1,58	0.000	1.79	1,58	0.186		
Latency (ms)	P1	52.8	17.5	53.6	16.9	74.7	13.3	81.7	23.8	1.21	1,57	0.275	66.17	1,57	0.000	1.00	1,57	0.017		
	N1	105.5	8.3	105.3	17.7	142.2	13.5	151.7	17.3	2.17	1,56	0.146	366.50	1,56	0.000	5.10	1,56	0.028		
	P2	175.3	16.1	185.8	19.3	218.6	18.9	222.3	25.8	2.69	1,56	0.106	169.06	1,56	0.000	1.21	1,56	0.021		
	P3	338.7	51.9	357.6	57.5	403.0	55.4	421.2	61.4	1.97	1,53	0.167	69.36	1,53	0.000	0.00	1,53	0.968		
P300	RT	482.7	95.8	555.9	146.8	531.5	100.4	629.3	144.2	7.78	1,58	0.007	58.16	1,58	0.000	2.35	1,58	0.1311		
behavioral	d'	5.18	0.50	4.71	0.86	4.86	0.59	3.96	1.07	14.21	1,58	0.000	41.716	1,58	0.000	7.026	1,58	0.010		