

Original article:

Mild cognitive impairment in mild brain injury (MBI) patients: An event related potential (ERP) and neuropsychology study.

Faruque Reza¹, Tahamina Begum²

Abstract:

Objectives: To evaluate auditory cognitive function in mild brain injury (MBI) patients, which is important to determine for rehabilitation and improve quality of their life. **Methods:** Participants (n=19/group) were divided into group 1 (G1-control), group 2 (G2/1st test-MBI/within 7 days of road traffic accident-RTA) and group 3 (G3/2nd test-MBI/2-6 months after RTA). Event related potentials (ERPs) were conducted using a 128-sensor net; participants counted silently rare target tone stimuli and ignored standard tones. Several neuropsychology tests like Verbal fluency test (PAS), Wisconsin Card Sorting Test (WCST), Rey Auditory Verbal and Learning Test (RAVLTIM, RAVLTDR and RAVLTTS) and Beck Depression Inventory (BDI) were subsequently administered. **Results:** Sensory (P50, N100) and cognitive (P300) ERP components were analysed from ERP waveforms. There were no significant group differences in amplitudes or latencies for all components across sites except P300 component amplitudes at T6 location. P50, N100 and P300 ERP components exhibited non-significantly increased amplitudes in G2 and G3 compared with G1 at all sites; non-significantly shorter latencies were identified at various sites. At several locations, G3 evoked non-significantly increased amplitudes and longer latencies with shorter latencies to other sites compared with G2 in all components. The MBI (G3) group exhibited significantly increased WCST, RAVLTIM and RAVLTDR scores compared with G1. **Conclusion:** These findings indicate MBI patients may have mild auditory, cognitive and executive dysfunctions with good auditory memory. MBI was associated with mild depression.

Keywords: Event related potential, Cognition, Neuropsychology tests, Executive function, Auditory stimuli, Mild brain injury.

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Introduction

Traumatic brain injury (TBI) is a leading cause of death and long term disability in young individuals¹⁻² with cognitive, emotional and social dysfunctions³⁻⁴; 20% of cases comprise moderate to severe brain injury, and 80% of cases comprise mild brain injury (MBI)^[2]. It is well documented that moderate to severe brain injury patients have cognitive dysfunction; however, controversy remains regarding cognitive impairment in MBI patients. Cognitive impairment comprises issues with learning, memory, attention, concentration, speed of processing, and complex information processing⁵. MBI involves slower information processing with impaired attention⁶.

Furthermore, there are emotional disturbances with personality changes in MBI patients⁷. Impaired working memory and executive function⁸, and impaired learning and memory⁵ have been reported in MBI patients, which result in poor quality of life. Executive functions are explained as an ability that is critical to guide someone's thought and everyday's actions of life. It is difficult to define the executive functions⁹, and also difficult to determine. Moreover verbal fluency and set-shifting are the important parts of executive function¹⁰.

Cognitive dysfunction should be examined following brain injury¹¹ using brain wave recording with event related potential (ERP) and

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neuropsychological assessments to improve the quality of life of MBI patients. Electrophysiological measures, such as ERP, comprise a promising, non-invasive and inexpensive method to investigate the functional integrity of neuronal activity in the brain. ERPs combined with neuropsychological tests represent a unique diagnostic approach to identify cognitive dysfunction in MBI patients¹². ERP has several components, including auditory perception components (P50, N100) and a cognitive component (P300); thus, auditory stimulation and other components are available during visual and somatosensory stimulations for individual analysis. Auditory attention measurement is a primary step in the assessment of executive function^[13]. Auditory P50 and N100 early components are referred to as 'sensory' components because they reflect physical parameters¹⁴, and the late P300 component reflects the cognitive component sensitive to the patient's mental state^{14, 15}. The P50 ERP component (first positive pick) is related to auditory maturation, which reflects auditory attention¹⁶. The N100 component (negative pick) is preceded by P50^{17, 18} and primarily comprises the pre-attentive component; it is involved in perception¹⁹, arousal²⁰ and selective attention^[21]. The P300 ERP component is the next major positive peak following the N100 and results from auditory stimulation²². The use of the P300 component is an important component in the diagnosis of cognitive dysfunction. Its latency and amplitude are thought to quantify the speed of information processing. There are documents that the socio-economic status also may affect the cognitive functions. Higher education can lead to the higher cognitive function, reflected in the P300, N170 and N200 ERP components^{23, 24}. Finally, higher education can ensure higher level of occupation and elevated income^{25, 26}. However, the results of ERP and neuropsychological assessments vary in MBI patients.

MBI patients are rarely managed in the hospital. Most patients are discharged with symptomatic treatment without further follow up because they appear well. MBI patients have mild residual cognitive impairments that may become obvious only in challenging and demanding situations. Moreover, a significant number of patients with MBI remain unemployed at 2 years post-injury^[27]. A thorough physical and neuropsychological examination is necessary to identify cognitive dysfunction in MBI patients to improve their quality of life. Therefore, in this study, we aimed to determine the amplitudes and latencies of ERP components evoked by auditory

stimulation via an auditory oddball paradigm and to use various neuropsychological tests to assess cognitive dysfunctions of MBI patients to ensure a correct diagnosis, which may ultimately facilitate further treatment/rehabilitation and improve their quality of life.

Methodology

Ethical Approval: The study was approved by the Human Ethics committee of Universiti Sains Malaysia (USM) (USM/KK/PPP/JEPeM (232.3[9])).

Study procedure

This is a prospective study. This study was done between January 2012 and December 2013. Age and education matched subjects were recruited and organised into three groups (Table 1). Group 1 (G1) (n=19) comprised control healthy subjects; group 2 (G2/1st test-MBI) (n=19) comprised participants with MBI who underwent an ERP test within 7 days of a road traffic accident (RTA); and group 3 (G3/2nd test-MBI) (n=19) included the same patients who were tested with both ERP and neuropsychology tests 2-6 months after a RTA. Sample size was calculated by one statistician using power and sample size (PS) software^[23, 24]. G1 was recruited by online advertisements, and G2 was selected by neurosurgeons in the Neurosurgery Department in the Hospital Universiti Sains Malaysia (HUSM) based on the recruitment criteria of MBI. All participants from all groups were Malaysian and signed a written informed consent form prior to enrolment. We excluded the persons from both groups who have secondary gain issues, history of intellectual disability and learning disability. Neuropsychology tests were performed by an expert neuropsychologist, and all procedures were conducted in the MEG/ERP laboratory at HUSM within two years (2012-2013). The demographic data of all groups are shown in Table 1.

Table 1: Demographic data of the control (G1) and mild brain injury (MBI) patient groups.

Characteristics		MBI group (n = 19)	Control group (n = 19)
Age(years)	Mean±SD	28.98± 9.32	31.11±8.28
Gender	Male,	16, 3	11, 8
	Female		
Education	Mean±SD	11.32± 1.4	11.05±2.04
Dominancy	Left,	1, 18	3, 16
	Right		

ERP study

A128-electrodesensor net was used for data acquisition

in the ERP experiment. Auditory stimulation was presented using E-prime v 2.0 software (Psychology Software Tools, Inc., Sharpsburg, Pennsylvania, USA) [23, 24]. All participants were seated in a dimly lit and sound treated room with head phones. For the auditory stimulation, we used an ‘auditory oddball’ paradigm. In this paradigm, the subjects were instructed to silently count rarely presented low frequency (20%) and high pitched (2000 Hz) sounds. The delivery of the tone duration was 100 ms with a rise/fall time of 10 ms. The band pass filter was 0.1-50 Hz, and the stimulus rate was 0.5 Hz. The electrode impedances were 10-50 K Ω . The amplitudes and latencies of the P50, N100 and P300 ERP components were measured for both the control and MBI groups.

Neuropsychology study

The Verbal fluency test (PAS), Wisconsin Card Sorting Test (WCST) [28], was done to assess the parts of executive functions. PAS was a verbally-based test that was done in English and all participants could follow the instruction as the alphabets of their language were English. Rey Auditory Verbal and Learning Test: immediate or interference recall (RAVLT IM), delayed recall (RAVLT DR) and total score (RAVLT TS) [29] were done to assess auditory attention and memory tests and Beck Depression Inventory (BDI) [30] that was a self report questionnaire was administered for ‘depression test’. The neuropsychology assessment in the G1 and G3 groups were done. There was no assessment for G2 because this group was in the acute stage after a RTA. Therefore we could not do any particular baseline for MBI group. We compared the scores of all neuropsychological testing among control and MBI groups where scores of control group were determined as baseline.

Data analysis.

The mean differences between the target and standard stimuli for the amplitudes and latencies of the P50, N100 and P300 ERP components were collected using Net-Station software 5.2 (Electrical Geodesics, Inc., Eugene, OR, USA) [23, 24]. We subsequently determined the significance level among the groups across electrode positions using Statistical Package for Social Sciences (SPSS) version 22.0 [23, 24]. One way analysis of variance (ANOVA) was used for this purpose. The significance level was set at $p < 0.05$. The neuropsychology test scores for the control and 2nd test-MBI were compared using non-parametric, independent t-tests with SPSS-22 software.

Results

Figure 1 shows the grand average waveform of the ERP components at six different electrode sites (T3, T4, T5, T6, Cz and Pz). Because our paradigm comprised auditory stimulation, we selected temporal electrodes (T3, T4, T5, and T6) and midline electrodes (Cz and Pz), which reflect auditory perception and attention, respectively. We used the mean differences between the target and standard stimuli for the values of the amplitudes and latencies of all ERP components among the groups (Tables 2, 3, and 4). The amplitudes and latencies of the P50 ERP components of the three groups at the various electrode sites are shown in Table 2. There were no significant differences in the amplitudes or latencies of the P50 component (Table 2) or the N100 component (Table 3) among the groups across the various areas.

At all sites, the amplitudes of the P50 ERP component were non-significantly increased in both G2 and G3 compared with G1. G2 exhibited non-significantly increased amplitudes of the P50 component at four electrode sites (T4, T5, Cz, and Pz), and the other two electrodes (T3 and T6) exhibited non-significantly increased amplitudes in G3. This trend was not identified in the P50 latency. In this case, non-significantly shorter latencies of the P50 component were identified in various electrode positions across the groups: G1 (T3, T4, and T5), G2 (Cz) and G3 (T6 and Pz) (Table 2). Non-significantly longer latencies of P50 were identified in G3 at T3, T4, and T5 compared with the other two groups. In a comparison of G2 and G3, the tendency of the longer latency remained present in G3 (Table 2). The latency of the N100 component was different. However, in general, non-significantly increased amplitudes of the N100 component were identified in G2 and G3 compared with G1 at all sites (Table 3). G2 evoked non-significantly increased amplitudes of N100 at the most electrode sites (T3, T6, Cz, and Pz) compared with G3. Non-significantly lower amplitudes of the N100 component were identified in G3 (T6 and Pz) compared with the other two groups; this finding was not demonstrated in the P50 component. Regarding the N100 component latency, non-significantly shorter latencies were evoked at various sites across the groups: G1 (T6), G2 (T3, Cz, and Pz) and G3 (T4 and T5) (Table 3). Non-significantly shorter latencies were identified at equal electrode sites in G2 (T3, Cz, and Pz) and G3 (T4, T5, and T6) (Table 3).

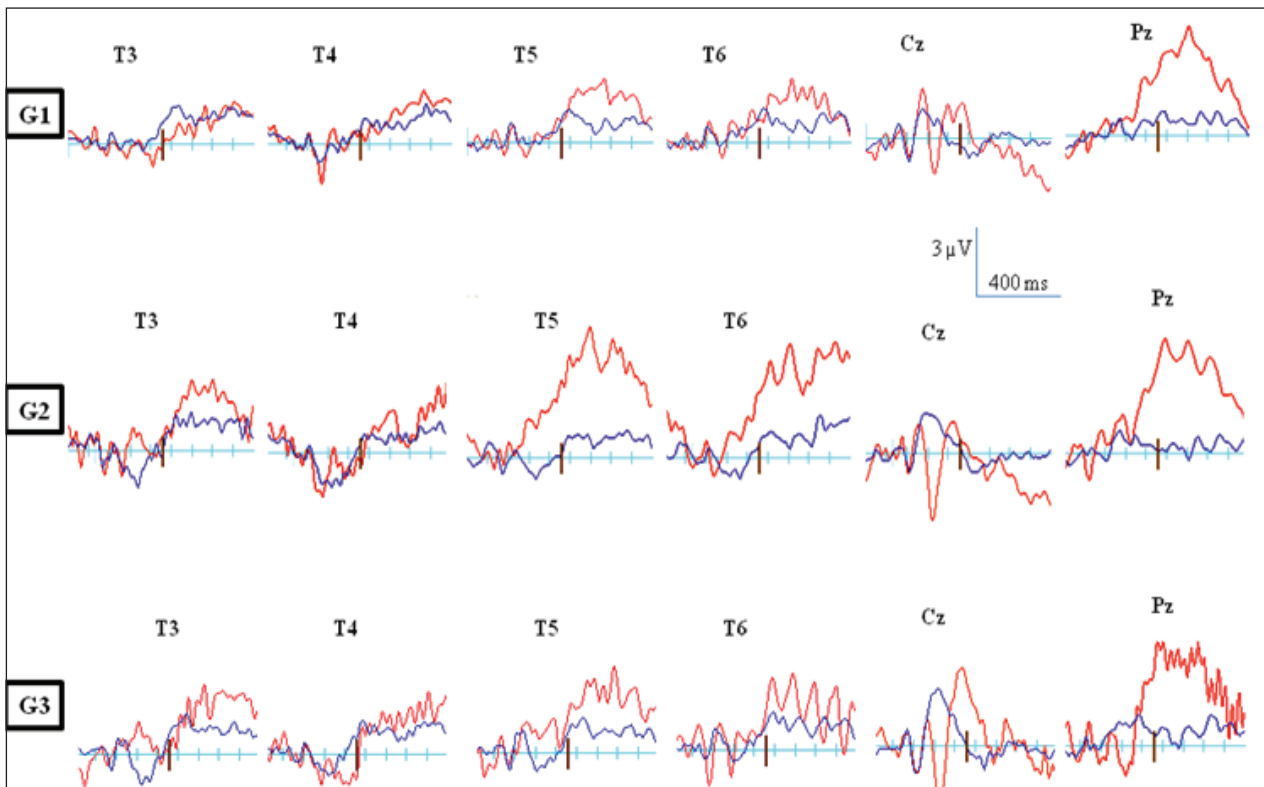


Figure 1: Grand average waveform of ERP components at T3, T4, T5, T6, Cz and Pz in G1 (control group), G2 (1st test-MBI group; ERP performed in mild brain injury patients within 7 days of a road traffic accident: RTA) and G3 (2nd test-MBI group; ERP performed at 2-6 months after a RTA in the same MBI patients). Red color indicates target stimuli, and blue color indicates standard stimuli.

Table 2: Amplitudes and latencies of the P50 ERP component at T3, T4, T5, T6, Cz and Pz locations in G1, G2 and G3 groups. Values represent mean differences between the target and standard stimuli.

P50 ERP Amplitude in μV (mean \pm SD)

Electrode Sites	Control (G1)	1 st test-MBI (G2)	2 nd test-MBI (G3)	df	F-value	P-value
T3	1.40 \pm 0.64	1.61 \pm 1.72	1.64 \pm 1.17	2,54	0.286	NS
T4	1.13 \pm 0.62	1.67 \pm 1.29	1.36 \pm 1.03	2,54	1.63	NS
T5	1.67 \pm 0.99	1.93 \pm 2.76	1.68 \pm 1.34	2,54	0.136	NS
T6	1.44 \pm 0.78	1.62 \pm 1.38	1.98 \pm 1.97	2,54	0.832	NS
Cz	1.18 \pm 0.70	1.60 \pm 1.60	1.34 \pm 0.96	2,54	0.806	NS
Pz	1.38 \pm 0.91	2.01 \pm 3.17	1.92 \pm 1.55	2,54	0.653	NS

P50 ERP Latency in ms (mean \pm SD)

Electrode Sites	Control (G1)	1 st test-MBI (G2)	2 nd test-MBI (G3)	df	F-value	P-value
T3	43.20 \pm 22.24	47.40 \pm 26.19	53.82 \pm 25.26	2,54	1.109	NS
T4	42.72 \pm 24.97	45.20 \pm 22.14	47.27 \pm 25.20	2,54	0.208	NS
T5	43.68 \pm 21.45	50.00 \pm 23.91	51.64 \pm 24.03	2,54	0.786	NS
T6	49.44 \pm 23.09	51.60 \pm 24.14	43.09 \pm 22.29	2,54	0.785	NS
Cz	53.44 \pm 25.24	47.00 \pm 23.96	48.91 \pm 26.01	2,54	0.398	NS
Pz	43.04 \pm 24.17	51.00 \pm 25.06	40.91 \pm 22.01	2,54	1.044	NS

Table 3: Amplitudes and latencies of the N100 ERP component at T3, T4, T5, T6, Cz and Pz locations in G1, G2 and G3 groups. Values represent mean differences between the target and standard stimuli.

N100 ERP Amplitude in μV (mean \pm SD)

Electrode Sites	Control (G1)	1st test-MBI (G2)	2nd test-MBI (G3)	df	F-value	P-value
T3	1.09 \pm 0.86	2.06 \pm 2.15	1.84 \pm 1.23	2,54	2.76	NS
T4	1.21 \pm 1.10	1.40 \pm 1.12	1.52 \pm 1.19	2,54	0.44	NS
T5	1.19 \pm 1.35	1.88 \pm 3.62	1.98 \pm 1.91	2,54	0.76	NS
T6	1.64 \pm 1.57	1.95 \pm 1.72	1.47 \pm 1.87	2,54	0.43	NS
Cz	1.35 \pm 0.77	1.76 \pm 2.27	1.60 \pm 1.31	2,54	0.41	NS
Pz	1.64 \pm 1.26	2.20 \pm 3.48	1.45 \pm 1.68	2,54	0.62	NS

N100 ERP Latency in ms (mean \pm SD)

Electrode Sites	Control (G1)	1st test-MBI (G2)	2nd test-MBI (G3)	df	F-value	P-value
T3	111.84 \pm 24.45	108.60 \pm 29.46	116.91 \pm 25.66	2,54	0.532	NS
T4	120.64 \pm 25.94	126.20 \pm 21.58	111.64 \pm 26.10	2,54	1.864	NS
T5	113.76 \pm 25.43	113.20 \pm 27.93	112.00 \pm 21.02	2,54	0.03	NS
T6	113.76 \pm 26.05	125.00 \pm 22.32	114.91 \pm 24.69	2,54	1.346	NS
Cz	117.76 \pm 24.27	112.00 \pm 27.38	116.18 \pm 23.21	2,54	0.309	NS
Pz	120.48 \pm 26.34	113.20 \pm 26.57	115.27 \pm 21.62	2,54	0.519	NS

Five of six areas (T3, T4, T5, Cz, and Pz) exhibited non-significantly increased P300 amplitudes in both G2 and G3 compared with G1, in which G2 exhibited increased P300 amplitudes compared with G3. A significantly increased amplitude of the P300 component was identified in G2 at the T6 area compared with G1, in which the group effect was high [$F(2,54) = 3.9, P = 0.02$] (Table 4). At the T6 area, the amplitude of P300 in G3 was increased compared with G1; however, the difference was not significant. Equivalent sites exhibited non-significantly increased

amplitudes in G2 (T3, T5, and T6) and G3 (T4, Cz and Pz) (Table 4). Regarding the P300 latency, non-significantly shorter latencies were identified at various electrode sites in G1 (T5, T6, and Cz), G2 (T3, T4, and Pz) and G3 (no electrodes) (Table 4). In a comparison of G2 and G3, an equal number of areas exhibited non-significantly shorter (G2: T3, T4, and T5; G3: T6, Cz, and Pz) and longer (G2: T6, Cz, and Pz; G3: T3, T4, and T5) latencies of the P300 ERP component (Table 4).

Table 4: Amplitudes and latencies of the P300 ERP component at T3, T4, T5, T6, Cz and Pz locations in G1, G2 and G3. Values represent mean differences between the target and standard stimuli.

P300 ERP Amplitude in μV (mean \pm SD)

Electrode Sites	Control (G1)	1st test-MBI (G2)	2nd test-MBI (G3)	df	F-value	P-value
T3	2.42 \pm 2.69	5.26 \pm 8.17	4.09 \pm 2.15	2,54	0.878	NS
T4	2.92 \pm 3.09	4.62 \pm 7.25	4.66 \pm 4.74	2,54	2.882	NS
T5	4.01 \pm 2.79	7.57 \pm 8.37	5.09 \pm 2.59	2,54	2.923	NS
T6	3.58 \pm 2.78 (*: G1-G2)	8.05 \pm 8.20	5.36 \pm 4.12	2,54	3.9	0.02
Cz	3.51 \pm 3.12	3.64 \pm 2.47	5.05 \pm 3.62	2,54	1.676	NS
Pz	5.49 \pm 3.20	6.28 \pm 5.92	7.28 \pm 5.47	2,54	0.774	NS

P300 ERP Latency in ms (mean ± SD)

Electrode Sites	Control (G1)	1st test-MBI (G2)	2nd test-MBI (G3)	df	F-value	P-value
T3	586.56 ± 139.36	540.80 ± 151.11	593.45 ± 137.39	2,54	0.845	NS
T4	555.36 ± 138.85	533.60 ± 198.89	555.09 ± 153.42	2,54	0.123	NS
T5	546.08 ± 127.98	556.40 ± 165.61	569.64 ± 153.09	2,54	0.262	NS
T6	514.88 ± 134.03	553.40 ± 171.92	536.91 ± 136.83	2,54	0.389	NS
Cz	394.88 ± 119.91	497.00 ± 186.08	457.09 ± 138.39	2,54	2.734	NS
Pz	541.60 ± 127.48	494.20 ± 139.83	477.82 ± 129.84	2,54	1.489	NS

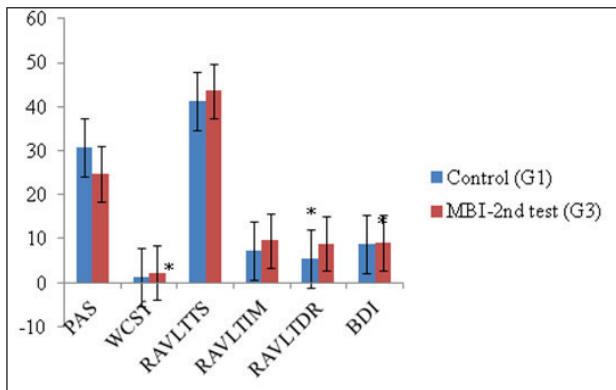


Figure 2: Bar graph of the neuropsychology test results in G1 (control) and G3 (2nd test-MBI) patients. I – symbol indicates error bars with standard error.

Table 5: Results of the neuropsychological tests in the control (G1) and MBI (G3) groups.

Tests	Control (G1)	MBI (G3)	P value
PAS	30.84 ± 12.63	24.79 ± 11.47	NS
WCST	1.32 ± 1.25	2.26 ± 1.15	0.02
RAVLTTS	41.21 ± 12.70	43.58 ± 13.75	NS
RAVLTIM	7.37 ± 1.95	9.58 ± 3.42	0.03
RAVLTDR	5.47 ± 3.55	8.89 ± 4.42	0.02
BDI	8.84 ± 8.46	9.05 ± 10.76	NS

Figure 2 showed a bar graph that compares the neuropsychology tests between the control (G1) and 2nd test-MBI (G3) groups. G2 (within 7 days of RTA) was not administered neuropsychological assessments because the MBI patients were assumed to be uncomfortable because of symptoms, such as nausea, dizziness, and body ache, at the time. The test scores are shown in Table 5. To assess executive function, the PAS and WCST were performed. In G3, the average (P=0.10) PAS score was non-significantly decreased (24.79±11.47) compared with G1 (30.84±12.63); however, the average

WCST score was significantly increased (P=0.02) in G3 (2.26±1.15) compared with G1 (1.32±1.25) (Table 5). Regarding the auditory memory tests (RAVLT), G3 had significantly increased scores on the RAVLTIM (9.58±3.42) (P=0.03) and RAVLTDR (8.89±4.42) (P=0.02) compared with G1 (7.37±1.95 and 5.47±3.55, respectively); the increased score on the RAVLTTS in G3 (43.58±13.75) compared with G1 (41.21±12.70) was not significantly different (P=0.67). Furthermore, G3 had a non-significantly (P=0.84) increased score on the depression test (BDI) (Table 5).

Discussion

The mean differences in the amplitudes and latencies of the P50, N100 and P300 ERP components were measured in the control (G1), 1st test-MBI (within 7 days of a RTA) (G2) and 2nd test-MBI (2-6 months after a RTA) (G3) groups to assess auditory perception and cognitive function in MBI patients. Non-significantly increased amplitudes at the P50, N100 and P300 (and significant at T6) ERP components were identified at all electrodes in the MBI groups (G2 and G3) compared with the control group (G1). Non-significantly shorter latencies were identified at various electrode sites in all components. In a comparison of G2 and G3, G3 evoked non-significantly increased amplitudes and shorter latencies at several (2-3) sites compared with G2 in all components (Tables 2, 3, and 4). The neuropsychological tests indicated non-significantly decreased scores on the PAS, significantly increased scores on the WCST, RAVLTIR and RAVLTDR, and non-significantly increased scores on the RAVLTTS in G3 compared with G1. G3 also exhibited slightly more depressive symptoms compared with G1; however, the difference was not statistically significant (Table 5).

The earliest ERP component at P50 has rarely been investigated using an auditory stimulation paradigm. Previous research has demonstrated that the auditory

P50 does not typically change with various factors, such as age³¹ or attention³². However, recent, extensive research has changed this idea. Attention is a component of cognitive function that may change the amplitude and latency of the P50 ERP component. Increased amplitude of P50 compared with the control indicated mild cognitive impairment^{33, 34}. Increased amplitude of P50 is present in normal aging; however, if this amplitude is increased compared with normal aging, it indicates cognitive dysfunction³⁵. Therefore, large P50 amplitude indicates a high risk for the development of Alzheimer's disease or dementia, and mild cognitive impairment is common in this patient group. However, the P50 amplitude tends to be smaller in patients with Alzheimer's disease or dementia.³⁵ An increased amplitude and prolonged latency of P50 have been identified in mild cognitive impairment patients³⁶. Our results strongly support the findings of Golob (2000, 2002, 2007)^{33, 35, 36} and Frodl (2002)³⁴ because we identified increased amplitudes of the P50 component at all electrodes and a longer latency of P50 at some electrodes in both G2 and G3 compared with G1 (Table 2). Although a shorter latency of P50 was also identified in our research, the increased amplitudes and longer latency of P50 strongly indicate that MBI patients had mild dysfunction in auditory perception because P50 measures sensory perception. In the acute stage after a RTA (within 7 days) (G2), the MBI patients had more dysfunction as evidenced by the highest amplitudes and longest latency among the three groups. At 2-6 months after a RTA, the same group (G3) recovered their dysfunction as compared to G2 as evidenced by lower amplitudes and shorter latencies in some electrodes compared with the acute stage but not based of neuropsychological data.

The N100 ERP component has also been investigated during auditory tasks. Increased N100 auditory amplitude has been identified in patients with mild cognitive impairments^{36, 37}. A small decrease in the N100 amplitude has also been reported in patients with Alzheimer's disease who have mild cognitive dysfunction³⁸, as well as patients with social phobia³⁹. Similar to the P50 ERP component, N100 is also considered a perception component. We identified non-significantly increased amplitudes of the N100 component in both G2 and G3 at all sites compared with G1, in which G2 evoked increased amplitudes of the N100 components at maximum sites compared with G3. However, longer latencies were identified at equivalent sites in G2 and G3 (Table 3). Considering the previously discussed research and based on the

findings of the N100 component, MBI is associated with mild dysfunction in auditory perception, which is recovered in G3 over time after the acute stage.

Cognitive function was assessed by investigating the amplitudes and latencies of the P300 ERP component. Decreased⁴⁰ and increased⁴¹ amplitudes of the P300 ERP component have been identified in MBI patients in the auditory oddball task. Non-significantly increased amplitudes of the P300 component in MBI (G2 and G3) patients were identified at all electrode sites (and significant at T6) (Table 4), which is consistent with Picton et al (1988)⁴¹ and in contrast with the previously described studies. These attenuated amplitudes of the P300 ERP component demonstrated that MBI patients pay more attention to the target stimuli, which indicates that their cognitive function is better than the control group. In investigations of the P300 latency, a longer latency of the P300 ERP component in auditory stimulation has been documented in severe brain injury patients^[40, 42]. This evidence suggests that brain injury patients have a delay in evaluating and categorising auditory target stimuli. Similar to severe brain injury patients, MBI patients have also demonstrated a significantly prolonged latency of the P300 component using an auditory stimulation paradigm^{40, 43}. The findings of our study are consistent with the findings identified in similar research of some but not all electrode sites. Our MBI (G2 and G3 both) groups exhibited a non-significantly longer latency of the P300 component compared with the control (G1) (Table 4). The other minimum electrode positions exhibited a non-significantly shorter latency in G2 and G3 compared with G1. These two controversial findings (both increased and shorter latencies of the P300 component) were documented in our study across various electrode positions (Table 4). An equal number of electrodes exhibited a longer latency in G3 compared with G2; thus, we assume that the MBI (G3) group had mild cognitive deficiencies at 2-6 months post-injury, which was not recovered by timing.

Non-significantly lower scores of phonologic fluency (PAS) and increased scores on the WCST (pers. error rate) have been identified in dementia patients⁴⁴. No difference in the WCST score has been documented in MBI patients compared with a control group⁴⁵. We measured the WCST score according to the error rate. A non-significantly decreased PAS score and a significantly increased error score on the WCST in the MBI group in our study suggest that MBI patients have mild executive dysfunction (Table 5). However,

this finding is limited because the difference in the PAS score was not significant. In addition to executive dysfunction, the MBI patients had good auditory attention and memory in our study because we identified significantly increased scores on the RAVLTIM and RAVLTDR and a non-significantly increased score on the RAVLTTS (Table 5). The lower scores on the three parts of the RAVLT (IM, DR and TS) in moderate to severe brain injury indicate auditory dysfunction⁴⁶. In contrast to the research by Anderson, our study strongly suggests that MBI patients have good auditory memory. Similar to the findings by Arlinghaus (2005), we demonstrated that MBI patients developed mild depression⁴⁷. Studies proved that major depression can reduce auditory attention^{48, 49}. However with consistency of these studies our MBI group has mild depression which was reflected in amplitudes and latencies of the ERP components that were mildly reflected by mild depression.

Conclusion

We investigated auditory perception (attention) and cognitively assessed MBI patients compared with a control group using ERP and various neuropsychology tests within 7 days and 2-6 months after a RTA. Non-significantly increased amplitudes and prolonged latencies of the P50 and N100 ERP components at maximum electrode positions strongly suggested that MBI patients have mild dysfunction, particularly in auditory perception. A significantly increased RAVLT score suggested good auditory memory in the MBI patients. Significantly and non-significantly higher increased amplitudes of the P300 components at all sites and non-significantly shorter latencies of P300 at several sites indicated that MBI patients

have increased cognitive function compared with the control group. Several sites with non-significantly increased latencies of the P300 ERP component suggested mild cognitive impairment with mild executive dysfunction. MBI patients also had mild depression. This mild auditory perception and mild cognitive function impairment may be overcome with proper rehabilitation to recover their mild executive dysfunction and depression and improve their quality of life. Therefore, the combination of ERP and neuropsychological assessments may represent an additional, unique modality to identify cognitive impairment in MBI patients.

Conflict of interest: Nil

Authorship contribution

FR and TB both performed data acquisitions, analysis, interpretations and wrote the manuscript together.

Study limitations

1. Our study population was small (n=19 in each group). However it is difficult to manage mild brain injury patients to come hospital for further investigation, as maximum patients ignore their problem.
2. G2 (acute stage of MBI patients) was not given neuropsychological tests.
3. Another limitation was more males were recruited than females in both groups.

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