Visual electrodiagnostic findings in mild traumatic brain injury

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Patients with traumatic brain injury (TBI) frequently exhibit varied forms of visual system dysfunction including: binocular, oculomotor, accommodative, refractive error shift, visual field loss, and visual perceptual deficits. A 5-year collaborative study between optometry and ophthalmology was initiated to follow documented mild TBI patients utilizing diagnostic methods to assess the quantity and quality of visual system deficits and recovery. A group of patients with mild TBI receiving optometric rehabilitation were compared with a group of age-matched, gender-matched, and headsize-matched TBI patients not receiving such treatment. Eighteen patients diagnosed with mild TBI underwent a treatment regimen of optometric rehabilitation (group I); 32 patients diagnosed with mild TBI did not receive optometric rehabilitation (group II). Pattern visually evoked cortical potential (VECP) testing and electroretinography (ERG) evaluation were utilized initially, repeated 6–12 months later and then 12–18 months after baseline. All TBI patients' VECP and ERG results were compared to age-matched, headsize-matched controls. Once the ERG had been used to exclude retinal involvement, identification of visual pathway dysfunction was possible with the VECP. Full-field ERG results in all groups were not remarkable and not sensitive for patients with mild TBI. Initial testing results revealed that 72% of those TBI patients in group I demonstrated VECP waveform abnormalities and 81% of those patients in group II showed waveform dysfunction. In the testing performed 12–18 months later, 38% of group I TBI patients, after receiving a treatment regimen of optometric rehabilitation, showed VECP waveform abnormalities; 78% of group II TBI patients demonstrated waveform abnormalities. VECP evaluation in patients with mild TBI can provide a useful and reliable tool for objective assessment of visual system deficit and recovery. Significant differences in visual system recovery were shown when comparing group I and group II.

Introduction

Traumatic brain injury (TBI) patients can demonstrate many types of visual dysfunction including accommodative, binocular and/or oculomotor dysfunction, refractive error shift, visual perceptual deficits, and visual field loss [1–18]. Much of the research pertaining to TBI has been concerned with those patients with moderate to severe involvement. More recently, additional attention has focused on the rehabilitative needs of those with mild TBI [19,20]. TBI injuries can be associated with blunt and/or penetrating trauma, acceleration/deceleration, suffocation, hypoxia, toxic substance exposure, and cerebral vascular accidents (CVA). Whiplash or cervical strain can also cause visual system dysfunction [21–23]. The current definition of mild TBI was developed by the Mild Traumatic Brain Injury

Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitative Medicine [24]. A patient with mild TBI is one who has had a traumatically induced physiological disruption of brain function. It is manifested by at least one of the following:

(1) Any period of loss of consciousness.
(2) Any loss of memory for events immediately before or after the accident.
(3) Any alteration in mental state at the time of the accident (e.g. feeling dazed, disoriented or confused).
(4) Focal neurological deficit(s) that may or may not be transient; but where the severity of the injury does not exceed the following:
 (a) loss of consciousness of approximately 30 minutes or less;
 (b) after 30 minutes, an initial Glasgow Coma Scale of 13–15;
 (c) post-traumatic amnesia not greater than 24 hours.

The patient may be hospitalized for a relatively short period of time, or often not at all. Functional deficit can occur even in the absence of documentation or loss of consciousness. Mild TBI can also encompass a diffuse injury which can disrupt the overall speed, efficiency, and integration of mental and central nervous system function. These deficits can affect the speed and capacity to process information, attend or learn; alter decision-making abilities; affect emotional stability; and compromise sensory motor integration. The patient's subjective symptomatology can include headaches, nausea, dizziness, visual complaints, confusion, agitation, and fatigue. Many affected patients have difficulties returning to prior levels of performance when the areas involve speed, attention, memory and integrative processing [19].

The anatomy and physiology of the skull, the vascular network of the brain, as well as the physics of TBI, contribute to the effects of insult to the visual system [1]. Since a significant amount of patients' day-to-day functioning requires effective visual processing and systemic integration, it is essential that the visual system be thoroughly evaluated, and deficiencies appropriately addressed in this population.

Visual system assessment in this population is not usually performed during the acute stage of medical intervention following TBI. Frequently, patients with mild TBI are not evaluated thoroughly with regard to the visual system. In many cases, where visual system examination takes place, the globes and posterior segment findings are unremarkable. In some patients, however, visual system compromise is manifested by direct or indirect optic nerve trauma, damage to the anterior visual pathways and/or subcortical or cortical damage which can result in visual processing disorders.

Ophthalmology, neurology, and rehabilitative medicine can provide diagnostically valuable, accurate, and reproducible data regarding visual system dysfunction in this population. Optometrists have historically treated functional and visual perceptual problems in children and adults [2,12,25,26]. Therefore, it is appropriate for optometric services to be included in the rehabilitation of the TBI patient with visual system dysfunction [1–3].

In 1989 a collaborative 5-year study between optometry and ophthalmology was initiated. Primary objectives were to follow documented mild TBI patients utilizing: Visual electrodiagnostic methods to assess the rate, quantity, and quality of visual system deficits and recovery. Patients with mild TBI receiving optometric rehabilitation (group I) were compared with age-matched, gender-matched, and

Table 1. *Amplitudes and implicit times of photopic and scotopic B-waves in normal controls*

Subject classification	PBA†	SBA†	PBI†	SBI†
Male	152 ± 31‡	328 ± 42	30·6 ± 1·4	76·2 ± 11·3
Female	157 ± 38	344 ± 58	31·7 ± 1·4	71·3 ± 8·2
Age groups (by decades)				
0–19	164 ± 42	352 ± 57	30·7 ± 1·3	72·4 ± 5·2
20–39	153 ± 31	329 ± 60	31·6 ± 1·4	73·5 ± 8·2
40–59	146 ± 26	321 ± 63	32·2 ± 1·3	80·2 ± 6·5
60–79	150 ± 24	318 ± 47	32·9 ± 1·3	85·9 ± 11·0

† Data from the Colorado Eye Center Physiology Laboratory are means and standard deviations of male and female patients and patients by age group. PBA=photopic B-wave amplitude: SBA=scotopic B-wave amplitude; PBI=photopic B-wave implicit time; SBI=scotopic B-wave implicit time. All amplitude values are in microvolts and implicit times in milliseconds.
‡ Values are means ±1 SD (1·06–1·32).

subtended a visual angle of 0–14 degrees with a check size of 56 minutes. The reversal rates was 1·88 r.p.s. Impedances were below 5 kΩ. The VECP was recorded utilizing a single-channel montage (FpZ–Cz–Oz) and then from both hemispheres using standard bipolar linkage O2–C4 and O1–C3. This technique allowed lateralization of the potential over the visual cortex contralateral to the field illuminated [31–34]. The resulting signal was recorded and computer-averaged. The activity at the left and right occipitus was separately recorded. Utilizing this method it was possible to identify optic nerve and visual pathway speed of conduction and conduction amplitude response.

Normative VECP latency data for 124 laboratory normal controls are shown in Table 2. The criteria for VECP abnormality for single channel montage to pattern-reversal stimulation for this study were as follows:

(1) A P100 latency delay of greater than 15% over three trials; and/or

Table 2. *Normal values for VECP latency to flash and pattern-reversal stimulation in normal controls (Colorado Eye Center/Ophthalmology Physiology Laboratory)*†

Decades (years)	Pattern-reversal P100 (56-minute check), latency (ms)	Flash P100 Component, latency (ms)
10–19	102·53 ± 10·4‡	108·3 ± 9·34
20–29	110·53 ± 6·36	120·73 ± 10·58
30–39	113·73 ± 4·62	121·50 ± 8·3
40–49	111·48 ± 5·38	125·4 ± 11·5
50–59	114·38 ± 6·44	123·69 ± 14·31
60–69	118·17 ± 10·31	127·49 ± 11·53
70–79	117·56 ± 8·92	134·31 ± 12·41

† Normal values for VECP latency throughout the adult life span to pattern-reversal stimulation and to flash stimulation. It should be noted that the smaller standard deviations seen for pattern-reversal stimulation are apparent only over a limited age group between 20 and 60 years of age.
‡ Values are means ±1 SD (1·84–2·16).

headsize-matched mild TBI patients not receiving optometric rehabilitation (group II). The optometric rehabilitation for mild TBI patients in group I included the following: prescription of lenses (refractive and non-compensatory), prisms (compensatory and yoked), partial occlusion, and vision therapy [27,28].

We measured the extent of visual system abnormalities in visual processing utilizing visual electrodiagnostic methods in this population to ascertain whether they were reproducible, reliable and clinically significant indicators of dysfunction. In addition, we evaluated whether optometric rehabilitation impacted this population.

Methods and materials

Patients who were entered in this study met the following criteria:

(1) A validated and confirmed history of mild TBI diagnosed by neurology or neuropsychology.
(2) Visual acuity 20/70 or better by Snellen acuity in each eye with best correction. No uni-ocular patients.
(3) No other unrelated ocular pathology.
(4) No other unrelated systemic pathology.
(5) Visual symptomatology including, but not limited to diplopia, visual fluctuation, asthenopia, spatial distortion, and photophobia.

The patients were divided into two groups:

Group I consisted of 18 patients: 10 males (mean age 31·8 years) and eight females (mean age 33·2 years) with documented mild TBI. These patients received a comprehensive eye/vision evaluation by optometrists who were certified as Fellows of the College of Vision Development (FCOVD). Additional sensory motor testing (in depth accommodative and binocular) was given to patients in group I in order to diagnose and prescribe the appropriate treatment regimen for optometric rehabilitation [17]. The patients were then referred for visual electrodiagnostic evaluation by a licensed and certified electrophysiologist. The patients then participated in a treatment regimen of optometric rehabilitation. Optometric rehabilitation has previously been found to improve specific acquired vision dysfunction as determined by standardized diagnostic criteria [1].

Group II encompassed 32 age-matched, gender-matched, and headsize-matched mild TBI patients: 20 males (mean age 30·2 years) and 18 females (mean age 33·8 years) who were referred by ophthalmology, neurology and family medicine for visual electrodiagnostic evaluation. These patients recieved a comprehensive eye/vision evaluation by an ophthalmologist. Patients in group II did not receive additional sensory motor testing or optometric rehabilitation.

Each participating optometrist or ophthalmologist included items from the following list in the examination procedure:

(1) History: in-depth history relating to TBI, neurological status, description of other professional/health-care, general health, medications, symptomatology, pre- and post-trauma and visual history.

(2) Ocular health assessment: pupillary function, internal and external ocular health evaluation including a dilated fundus examination, tonometry.
(3) Visual acuity: uncorrected and corrected visual acuity at distance and near.
(4) Refractive status: retinoscopy, subjective refinement.
(5) Binocularity: cover test at distance and near in primary gaze, with all fields of gaze assessed when deviation was non-comitant; near point of convergence; phorias, horizontal and vertical fusion ranges at distance and near in phoropter; Worth four-dot at distance and near; Randot stereopsis.
(6) Accommodation: near point of accommodation, positive relative accommodation, and negative relative accommodation.
(7) Colour vision: Ishihara plates.
(8) Visual fields: Goldmann full-threshold static and kinetic perimetry (three isopters).
(9) Contrast sensitivity: Vistech 6500 system at distance.

A variety of visual electrodiagnostic methods to assess visual function were utilized and included: full-field electroretinography (ERG), which represents the overall score of retinal function and the visually evoked cortical potential (VECP), which shows neural representation of retinal function with a macular preference and the integrity of the visual pathway from the retina to, and including, the visual cortex. Waveform reproducibility, multiple-trial assessment of visual processing stability and conduction quality (amplitude) were assessed in all groups. The mean elapsed time from TBI to initial electrodiagnostic evaluation for TBI patients in group I was 1·86 years for males and 1·54 years for females. Mean elapsed time from TBI to initial electrodiagnostic evaluation for TBI patients in group II was 1·28 years for males and 1·42 years for females. Full-field ERGs were recorded by methods which adhered to the international standards set by the Standards Committee of the International Society for Clinical Electrophysiology of Vision (ISCEV) [29]. There are no such standards agreed upon for the recording of the VECP [30].

Patients were tested utilizing full-field electroretinography. Ganzfeld dome stimulation was used, as well as a Burian–Allen corneal contact lens electrode. ERG testing was performed after VECP testing because VECP evaluation was performed without dilation. Patients were then dilated with 1% mydriacyl for ERG testing. The montage included a ground skin electrode at Fpz and reference electrodes at each ipsilateral ear. Impedances were below 5 KΩ.

Both amplitude and implicit times were measured for the cone response, rod response, maximal B-wave amplitudes and flicker B-wave time to peak. A-wave amplitude was measured from the baseline to A-wave trough; B-wave amplitude from A-wave trough to B-wave peak; and the B-wave time-to-peak from flash onset to the peak of the wave. All ERG findings for TBI patients were compared to 68 age-matched patients out of 165 ERG laboratory normals. Normative full-field electroretinography B-wave values for controls are shown in Table 1.

All patients were also investigated using pattern-reversal stimulation from a television monitor. Stimulations were performed binocularly and to each eye independently with best correction. All TBI subject waveform results were compared to 21 gender-matched, headsize-matched, and age-matched normal controls who were a subgroup of a total of 124 VECP laboratory normals. Three trials of 100 repetitions were recorded with a 10-minute rest period between trials. The stimulus

(2) Interwave amplitude decrease of more than 50% over three trials;
   and/or
(3) non-recordable waveform response over three trials;
   and/or
(4) a difference in recording of more than 15% between left and right side responses;
   or
(5) Waveform responses found to be within normal limits

TBI patients from groups I and II were further divided into subgroups satisfying the VECP abnormality criteria described above:

Group A: criteria (1) and/or (2) met.
Group B: criterion (3) met.
Group C: criteria (1) and/or (2) and/or (4) met.
Group D: criterion (5) met.

Some patients in TBI groups I and II met criteria in both subgroups A and C. Patients meeting criteria in subgroups B and D did not fall into any other subgroup.

Results

Full-field ERG results for all patients in groups I and II were recorded during baseline testing and subsquent evaluations. Full-field ERG findings were compared and correlated separately for patients in groups I and II. Baseline and subsequent mean (±1 SD) photopic B-wave amplitudes for TBI patients in groups I and II are shown in Tables 3 and 4.

Full-field ERG findings for photopic B-wave amplitude; scotopic B-wave amplitude; photopic B-wave implicit time; and scotopic B-wave implicit time for all patients in groups I and II were found to be within normal limits for baseline and subsequent evaluations. There were no statistically significant differences in findings for TBI patients in groups I or II.

This study correlated ERG and VECP test specificity, accuracy (predictive value) and sensitivity in patients with TBI in groups I and II. Table 5 shows

Table 3.   Full-field ERG B-wave findings for TBI patients receiving optometric rehabilitation (group I)

Patient gender and test interval	PBA†	SBA†	PBI†	SBI†
Male (10 males: mean age 31·8 years)				
Baseline	143 ± 22†	317 ± 37	30·2 ± 1·2	72·4 ± 10·8
6–12 months	144 ± 22	325 ± 32	30·8 ± 1·3	74·1 ± 10·1
12–18 months	142 ± 24	321 ± 33	30·3 ± 1·2	74·3 ± 9·3
Females (eight females: mean age 33·2 years)				
Baseline	152 ± 34	356 ± 44	31·5 ± 1·3	70·8 ± 8·5
6–12 months	154 ± 39	346 ± 52	31·1 ± 1·2	71·2 ± 9·2
12–18 months	151 ± 40	352 ± 49	31·7 ± 1·2	70·2 ± 9·5

† Data from the Colorado Eye Center Physiology Laboratory are means and standard deviations of male and female patients. PBA=photopic B-wave amplitude; SBA=scotopic B-wave amplitude; PBI=photopic B-wave implicit time; SBI=scotopic B-wave implicit time. All amplitude values are in microvolts and implicit times in milliseconds.

Table 4. *Full-field ERG B-wave findings for TBI patients not receiving optometric rehabilitation (group II)*

Patient gender and test interval	PBA†	SBA†	PBI†	SBI†
Male (20 males: mean age 30·2 years)				
Baseline	141 ± 20†	336 ± 42	30·1 ± 1·2	73·6 ± 10·3
6–12 months	142 ± 22	341 ± 37	30·8 ± 1·2	76·2 ± 11·2
12–18 months	140 ± 26	329 ± 41	30·1 ± 1·3	72·3 ± 10·1
Females (12 females: mean age (33·8 years)				
Baseline	157 ± 31	361 ± 48	31·2 ± 1·3	70·2 ± 9·2
6–12 months	166 ± 26	372 ± 41	31·7 ± 1·2	72·1 ± 10·3
12–18 months	169 ± 33	360 ± 36	31·9 ± 1·2	72·7 ± 11·2

† Data from the Colorado Eye Center Physiology Laboratory are means and standard deviations of male and female patients. PBA=photopic B-wave amplitude; SBA=scotopic B-wave amplitude; PBI=photopic B-wave implicit time; SBI=scotopic B-wave implicit time. All amplitude values are in microvolts and implicit times in milliseconds.

Table 5. *Formulae for test sensitivity, accuracy and specificity*

A = TBI patients with abnormal VECP or ERG results
B = Controls with abnormal VECP or ERG results
C = TBI patients with normal VECP or ERG results
D = Controls with normal VECP or ERG results

Sensitivity: A/A+C
Accuracy (Predictive Value): A+D/A+B+C+D
Specificity: D/B+D

Table 6. *Combined ERG results for TBI groups I and II for test sensitivity, accuracy and specificity at baseline*

Combined amplitude latency values	Numbers of patients		Statistical analysis
	TBI	Control	
Abnormal ERG	0	0	Sensitivity (%): 0
Normal ERG	50	68	Accuracy (%): 42
			Specificity (%): 100

p < 0·005

those statistical formulae utilized to aid in determination of the diagnostic value, accuracy and efficacy of the VECP and full-field ERG in this population.

Combined ERG results for test sensitivity, accuracy and specificity for TBI groups I and II are listed in Table 6, and show that the ERG's sensitivity to TBI pathology was very poor, as was test accuracy. Test specificity for this population, however, was found to be 100% (*p* < 0·005).

VECP abnormalities were compared and correlated separately for TBI patients in groups I and II. Table 7 illustrates VECP findings by subgroup for criteria established for group I TBI patients. Table 8 illustrates VECP findings by subgroup for criteria established for group II TBI patients. A comparison of VECP waveform abnormalities is summarized in Table 9.

Table 7. Single-channel VECP findings for TBI patients receiving optometric rehabilitation (group I)

Subgrouping by VECP abnormality criteria met	Baseline	6–12 months	12–18 months
Subgroup A (P100 latency delay >15% and/or amplitude decrease >50% over three trials)	12 of 18 (66%)	9 of 18 (50%)	6 of 18 (33%)
Subgroup B (non-recordable waveform response)	0 of 18 (0%)	0 of 18 (0%)	0 of 18 (0%)
Subgroup C (P100 latency delay >15% and/or amplitude decrease >50% and/or difference of >15% between right and left sides over three trials):	13 of 18 (72%)	10 of 18 (55·5%)	7 of 18 (38%)
Subgroup D (waveform recording within normal limits)	5 of 18 (28%)	8 of 18 (44%)	11 of 18 (61%)

Table 8. Single-channel VECP findings for TBI patients not receiving optometric rehabilitation (group II)

Subgrouping by VECP abnormality criteria met	Baseline	6–12 months	12–18 months
Subgroup A (P100 latency delay >15% and/or amplitude decrease >50% over three trials)	23 of 32 (72%)	25 of 32 (78%)	21 of 32 (66%)
Subgroup B (non-recordable waveform response)	0 of 32 (0%)	0 of 32 (0%)	0 of 32 (0%)
Subgroup C (P100 latency delay >15% and/or amplitude decrease >50% and/or difference of >15% between right and left sides over three trials):	26 of 32 (81%)	28 of 32 (87·5%)	25 of 32 (78%)
Subgroup D (waveform recording within normal limits)	6 of 32 (19%)	4 of 32 (13%)	7 of 32 (22%)

Table 9. Single-channel VECP abnormal findings for groups I and II (percentages)

VECP testing interval and subgroup†	TBI group I (receiving optometric rehabilitation)	TBI group II (no treatment)
Subgroup A		
Baseline	66	72
6–12 months	50	78
12–18 months	33	66
Subgroup C		
Baseline	72	81
6–12 months	55·5	87·5
12–18 months	38	78

† Subgroup B not listed because no TBI patient in either group satisfied this criterion.

TBI patients in group I with subgroup A abnormalities demonstrated a decrease in VECP waveform dysfunction, with 66% showing abnormal findings at baseline to only 33% with waveform abnormalities after 18 months. There was also a reduction in subgroup C abnormalities from 72% to 38%. TBI patients in group II showed a reduction in subgroup A VECP abnormalities from 72% initially to 66%

after 18 months and in subgroup C abnormalities from 81% baseline to 78% after 18 months.

Group I TBI patients also demonstrated an increase in numbers of normal VECP waveform results: 28% of group I TBI patients showed normal VECP waveform function at baseline. At 12–18 months, 61% of these patients demonstrated normal VECP findings. Group II TBI patients, who were not receiving optometric rehabilitation, demonstrated a smaller increase in numbers of patients with normal VECP function over time. Nineteen per cent of patients in group II were found to have normal waveform function at baseline, with only 22% showing normal VECP findings after 12–18 months.

Combined VECP results for test sensitivity, accuracy and specificity for TBI groups I and II are shown in Table 10. Results show that VECP test sensitivity was 76% for those patients in subgroup A and increased minimally for those patients in subgroup C when abnormality criteria were widened to include a difference of more than 15% between left and right side responses. VECP test accuracy (predictive value) was 84% for those TBI patients in subgroup A and 85% for those in subgroup C. Specificity of the VECP for this pathology was found to be 100% ($p < 0.005$).

Discussion

Findings for this study clearly show that a majority of patients presenting with mild TBI demonstrate VECP waveform abnormalities. These patients also exhibited subjective visual complaints at baseline. We found no patients with non-recordable waveform function secondary to the inclusion criteria utilized and the extent and severity of type of head injury seen in this study. However, 72% of patients in group I and 81% of patients in group II demonstrated baseline VECP waveform abnormalities which met criteria for inclusion into subgroup C. These included a P100 latency delay of greater than 15% over three trials, interwave amplitude decreases of

Table 10. *Combined VECP results for TBI groups I and II for test sensitivity, accuracy and specificity at baseline*

	Number of patients		
	TBI	Control	Statistical analysis:
Subgroup A			
With P100 latency delay >15% three trials and/or amplitude decrease >50% three trials			
Abnormal VECP	35	0	Sensitivity (0%): 76
Normal VECP	11	21	Accuracy (%): 84
			Specificity (%): 100
Subgroup C			
With P100 latency delay >15% three trials and/or amplitude decrease >50% three trials and/or difference >15% between left and right sides			
Abnormal VECP	39	0	Sensitivity (%): 78
Normal VECP	11	21	Accuracy (%): 85
			Specificity (%): 100

$p < 0.005$

more than 50% over three trials; and a difference in recording of more than 15% between right and left side responses. Three trials were evaluated during VECP testing in this population, because delays in conduction and/or amplitude affectation often occurred during the second trial and not in the first. The third trial was performed to assess waveform reproducibility of latency delays or amplitude decreases noted in the second trial, as well as to measure any further delay in latency function or decrease in interwave amplitude in this population.

After patients in group I received optometric rehabilitation, only 38% met subgroup C VECP abnormality criteria 12–18 months after baseline. This was contrasted with 78% of TBI patients in group II who showed subgroup C VECP abnormalities after 12–18 months. The numbers of TBI patients demonstrating normal waveform function in group I increased from 28% at baseline to 61% after 12–18 months, whereas, in group II, numbers of TBI patients with normal VECP function rose only 3% from 19% at baseline to 22% after 12–18 months. It is important to note that the mean elapsed time from injury to initial visual electro-diagnostic evaluation was 1·86 years for males and 1·54 years for females in group I, and 1·28 years for males and 1·42 years for females in group II. As has been noted in the literature, this was often because other TBI-related injuries were treated initially, and because visual complaints were misunderstood, ignored or not deemed significant enough for a more immediate referral for vision evaluation [35]. It is also important to note the significance of the elapsed time from injury because, in our experience, 'spontaneous recovery' from mild TBI has been found to occur within 6–12 months post-injury. Electroretinographic (ERG) findings for both groups were non-remarkable and ERG findings were not sensitive to visual system abnormalities found in these patients. However, the ERG can can be utilized to rule out globe involvement in these patients.

This study further demonstrates that visual system abnormalities can be objectively documented in patients with documented mild TBI with the use of the VECP, and that this test was pathology-specific, sensitive, diagnostically valuable and reliably documented anterior visual pathway dysfunction in this population. These findings support the efficacy of VECP testing in terms of pathology sensitivity and specificity already noted in the literature [36–42]. In addition, optometric rehabilitation was found to have a positive impact on those patients in group I after TBI.

VECP evaluation in patients with documented mild TBI was found to be an objective and useful aid in the assessment of visual dysfunction in this study. This testing technique is concerned with functional integrity in this population, and provides an excellent correlation with neuroanatomy. Once the ERG had been used to exclude the possibility of retinal dysfunction, we were able to identify visual pathway dysfunction with VECP evaluation. Findings show that many of our patients with subjective visual complaints had a correlate in VECP waveform abnormalities found. Our VECP findings also indicate that TBI patients in group I who participated in optometric rehabilitation showed a significant increase in numbers of patients with normal VECP function. Optometric rehabilitation should be considered an important part of the rehabilitative process in this population.

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