Original Article

Evaluation of Moderate Closed Head Injury by Visual Evoked Response

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Summary:

Background: Traumatic brain injury (TBI) is a serious public health problem. Even injuries classified as mild which is the most common, can result in persistent neurobehavioural impairment. Diffuse axonal injury is a common finding after TBI, and is presumed to contribute to outcomes, but may not always be apparent using standard neuroimaging. All severities of TBI can result in a degree of axonal damage, while irreversible myelin damage was only apparent for moderate to severe TBI.

Objectives: To evaluate the effects of moderate closed head injury on visual evoked response (VER) and to assess the changes in VER 6 months later as follow up indices.

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Subjects and methods: This prospective study was conducted in the Department of Neurophysiology /Hospital of Neurosurgery/Baghdad from November 2010 to February 2012, using Esoata Italian machine. Ninety three patients with moderate closed head injury (49 female and 44 male), with an age range from (5 -51) years, with a mean age of (26±11.95) years, were chosen according to Glasgow coma scale (9-12 score) undergo VER and only 86patients will follow up by VER and both were compared to control group of 55 healthy subjects.

Results: P100 wave latency of visual evoked response (VER) was found to be significantly prolonged (P100= 145.41±9.67 msec.), in patients with moderate head injury at an early measurement after injury as compared to that of the healthy (P100= 97.66±4.58 msec.), whereas, the amplitude of P100 at an early measurements is reduced significantly (2.71± 0.77 µv) when compared to healthy subjects (7.32± 1.62 µv). By 6 months post injury the latency of P100was significantly prolonged (P100=137.19±8.45msec.) when compared to that of control group, while it is significantly reduced when compared to the patients with head injury at an early measurements. However, at 6months post injury the P100 amplitude was significantly increased (amplitude =3.26±0.62µv) when compared to the patients with head injury at an early measurement whereas, significantly reduced when compared to the control group.

Conclusion: this study revealed that head injury had serious effect on the brain functions reflected by changes in VER which needs long period of time to return to normal levels.

Keyword: Traumatic brain injury, visual evoked response.

Introduction:

Rapid deformation of the brain matter is the likely cause of concussion, as well as more severe traumatic brain injury, however rapid deformation of brain tissue is thought to cause "mechanically diffuse depolarization" of cortical neuron (1). In severe trauma, diffuse axonal injury (DAI) occurs throughout the white matter of the brain, as axons are stretched beyond a physiological injury threshold (2). TBI is usually, though not always, the result of a blow to the head that is hard enough to damage normal brain functioning. The mechanisms whereby lesions are produced involve acceleration, deceleration and rotation, which cause microscopic damage to neurons, cerebral axons and circulation (3). Acceleration-deceleration refers to the sudden change in direction of the brain, produced by coup and contrecoup, i.e. the damage that may occur at the site of the impact or on the opposite side to the injury by the movement of the brain striking the bony wall, which may cause cerebral contusions. Lesions may also be produced by rotational forces that stretch or distort brain matter (4). Diffuse axonal injury (DAI), is one of the most common pathologies of TBI, all severities of TBI can result in a degree of axonal damage, while irreversible myelin damage was only apparent for moderate to severe TBI (5). DAI are lesions typically existing in the white matter of injured brains, these lesions vary in size from about 1–15 mm and are distributed in a characteristic way (6). Visual evoked response (VER) tests the function of the visual pathway from the retina to the occipital cortex. It measures the conduction of the visual pathways from the optic nerve, optic chiasm, and optic radiations to the occipital cortex. The usual waveform is an initial negative peak (N1 or N75), followed by a large positive peak (P1 or P100), and followed by another

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negative peak (N2 or N145) (7). The generator site for VERs is believed to be the peristriate and striate occipital cortex(8). The GCS is a simple 15-point scale that measures the level of consciousness by measuring eye opening, verbal responses and limb movement For practical purposes, a GCS score of 8 or less has become the generally accepted definition of a comatose patient, head injured patients with a GCS of 9 to 12 have been categorized as "moderate " and those with a GCS of 13 to 15 have been designated "mild " (9). The aim of the study is to evaluate the effects of moderate closed head injury on visual evoked response (VER) and to assess the changes in VER, 6 months later as follow up indices.

Subjects and Methods:

This prospective study involved two groups of subjects were included in this study: the patients and control groups. Both groups of subjects were informed about the aim of the study and their consents for the test were taken. This study was performed at unit of Neurophysiology/Hospital of Neurosurgery/Baghdad from November 2010 to February 2012. Regarding Patients Group; Medical history with complete neurological examination were carried out for every patient by a specialist neurosurgeon that included the GCS score, cranial nerve with complete motor and sensory testing. All subjects underwent Computerized Tomography (CT) scanning of the head. Patients having any history, signs or symptoms of stroke or any other neurological illnesses other than head injury were excluded from the study. Patient with closed head injury of GCS of 9-12 and with negative CT scanning of the head were involved in this study. The head injured group comprised of 93 patients with moderate closed head injury (49 female and 44 male), with an age range from 5 -51 years, with a mean age of (26±11.95) years. In addition to age and gender matched 55 healthy volunteers served as control group. The control group comprised of 55 healthy volunteers (relatives, friends and staff in hospital) (27 female and 28 male) with an age ranging from (8-44) years old. They were regarded as normal when meeting the following criteria: 1. no past or present history of systemic illness (diabetes mellitus, psychiatric illness such as depression, anxiety, nor neurological disease, e. g. multiple sclerosis) 2. Absence of any of previous brain disease or head injury. Visual evoked response (VER) study was performed within the first 3 days of injury, moreover, 6 months later we follow up (86) out of 93 patients(41 female and 45 male) by VER. The VER was performed by using Esoata- Italian machine. The patient was seated on chair comfortably, in an upright position, in a dim light room to promote alertness; checkerboard of pattern- reversal stimulation type comprised of high contrast squares of black and white that periodically exchange places and was used at temporal frequency of 2 Hertz (Hz), binocular stimulation was done and the subjects asked to watch with both eyes in the center of the screen. The amplitude (wave size) was calculated in microvolt (μV) from the base line to the maximum (positive or negative) of the wave, the peak time was the time in milliseconds between the start of stimulation, and the point of the maximum peak at the midline occipital electrode normally contains a prominent positive component which occurs approximately 100 ms after the pattern reversal (P100). The patient was seated at about 100 centimeters (cm) from the stimulating screen, sweep time was250 millisecond (ms), band pass filter 1-30Hz, sensitivity was 20microvolt. After cleaning and sterilizing the scalp of the subject with rectified spirit, four platinum needle electrodes were inserted subcutaneously in the following sites: Oz: at the occipital protuberance. Cz: at vertex. Fz: 2 cm proximal from Cz. Fpz: 3 cm from nasion. Then the impedance was checked and kept below 5Kilo-ohm (KΩ) then the averaging processing of the stimulus was started and it will be stopped automatically after 500 averaging.

Statistics: all statistical analysis was performed using SPSS version 16, (descriptive statistics) presented as numbers and percentage, in addition to t-test. $P<0.05$ was considered as a leveled significance.

Results:

From data of the present research, there is no significant difference between the patients and control group regarding age and gender (table1).

Table 1: Demographic data of patients with head injury and control.

		Patients n=93		Controls n=55		P value
Age (years)	Mean±SD (Range)	26.9±11.95 (5-51)		26.1±9.49 (8-44)		0.67
		No	%	No	%	
Sex	Male	49	52.7	28	50.9	0.83
	Female	44	47.3	27	49.1	0.83

P value > 0.05 was considered as statistically non significant

Table 2: Characteristic of data of the studied groups:

Parameters	Early measurement n=93	6 months later measurement n=86	Control n=55	P value Early measurement v. control	P value Early measurement v. 6 months later measurement	P value 6 months measure-ment v. control
VER P100 latency(msec.)	145.41±9.67	137.19±8.45	97.66±4.5	<0.0001	<0.0001	<0.0001
VER –P100 amplitude (µV)	2.17±0.77	3.26±0.62	7.32±1.62	<0.0001	<0.0001	<0.0001

The present study shows a significant prolongation (P<0.0001) of P100 latency of the patient at an early measurement (145.41±9.67msec.) as compared to that at 6 months later (137.19±8.45msec.) as well as to that of control group (97.66±4.58msec.). while, there is a significant reduction(p<0.0001) in amplitude of P100 of the patient at an early measurement (2.17±0.77 µV) as compared to that at 6 months later measurement (3.26±0.62µV),and that of control group(7.32±1.62 µV). Moreover, P100 waves latencies of VER was shown to have a significant negative correlations with GCS, as the GCS increase in degree, the degree prolongation of P100 latency were reduced(table2).

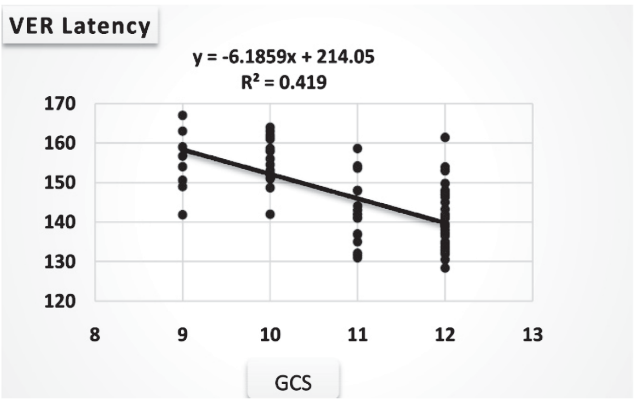

Figure 1: Correlation between P100 wave latency of the patient with head injury at an early measurement with GCS. r = - 0.647, significant correlation at 0.01. P<0.0001, significant level at 0.05.

Discussion:

The visual system is made up of the most complex neuronal circuit of all sensory systems. The prolongation of P100 wave latency of the head injury patient at an early measurement and at 6 months later measurement and this findings are in agreement with the study of Lachapelle, Alain and Michelle, 2005, they found that the prolongation of P100 latency of VER of the patients with moderate head injury at time of admission, also they found that the abnormality of P100 persist up to 31 months post-TBI. They conclude that as the latency of P100 is taken as a measure of retino-striate conduction time so that increase in P100 due to increased in retino-striate conduction time caused by processes such as demyelination that can be detected by measuring the latency of this cortical response (10, 11). However the study of Padula, Argyris and Ray, 2004, revealed that VERs are frequently delayed after traumatic brain injury presumably as a result of diffuse axonal injury, and the magnitude of the delay is correlated with other measure of injury severity, our results are in parallel with that(Table2)(12). Moreover, Lachapelle, Quim and Bach et al., 2004, in their study were explained the improvement in P100 latency after 6 months measurements by the fact that some function recovered or compensated so the affected VER measures decrease with the time post-TBI.and our findings are in agree with that (Table 2)(13). In this study the reduction in the amplitude of P100 of VER in patient with moderate head injury at an early measurement and 6 months later measurement as compared to control group and this in agree with the study of Lachapelle, Alain and Michelle, 2005, they were found that the amplitude appear to be diminished in TBI, moreover they were followed up their head injured patients at 3 months post- TBI and they found significant improvement in patients with moderate head injury, however, about 88%of their patients still have abnormality in amplitude of P100 at 31 months post –TBI (10). In addition, the study of Padula, Argyris and Ray, was revealed that the amplitude of VER is a function of cortical binocular integration and that is influenced by dysfunction of the ambient visual process. And they found that the occipital cortex does not solely represent the visual system, but there are at least two separate visual processes: focal process concerned primarily with the detail discrimination

and the ambient process which relate to spatial orientation, movement detection and balance. The ambient system comprised up to 20% of the nerves emanating from both eyes and these are directed to link up with midbrain and even brainstem prior to occipital cortex. Disturbance of the ambient visual process would interfere with the amplitude of the VER only when under binocular conditions. An increased in the binocular VER amplitude should occur if the ambient visual process resumes its role in stabilizing the peripheral field and enhancing the foveation process and this is in complete agreement with our findings of 6 months later measurement (Table2)(11).

Finally VER abnormalities reflects the cerebral dysfunction in certain localizing pathway, thus VER was considered as an effective means for potentials localization and prognosis use in traumatic brain injury (14 and 15).

Authors Contributors:

Dr. Maha Kamil: Preperation, Performing and doing the test of the research.

Prof. Dr. Najeeb Hassan Mohammed: This is to certify that this paper is prepard and performed under his supervision. Howeverit is derived from PhD thesis.

DR. Anwar Noori Hafdh: Support and assist Dr. Maha in doing the test of this research. In addition to provide specific instruments, apparatus and patients to accomplish this research.

References:

1. *Show NA: The neurophysiology of concussion. Progress in Neurobiology. 2002; 67:281-344.*

2. *Graham DI; Adams JH; Nicoll JAR; Maxwell WL and Grennarelli TA: The nature, distribution and causes of traumatic brain injury. Brain J. Pathology. 1995;5:397-406 .*

3. *Harting JA; Strong AJ and Fabricius M :Spread in depolarizations and late secondary insults after traumatic brain injury. 2007;. J. Neurotrauma 26:1857-.64.*

4.*Marmarous A; Lu J and Butcher I :Database of traumatic brain injury :design and description. Journal Neurotrauma.2007; 24:239-50.*

5. *Kraus M.F; Susmaras T; Caughlin B.P;walker C.J; Sweeney J.A. and Little DM: White matter integrity and cognition in chronic traumatic brain injury : A diffusion tensor imaging study. Brain .2007;130(10): 2508-2519.*

6. *Wasserman J and Koenigsberg RA: Diffuse axonal injury. Brain injury Journal.2007; 21 (3): 309-318*

7. *Kesler A; Vokhapova V; Korczyn AD and Drory VE: Visual evoked potentials in idiopathic intracranial hypertension. Clin Neurol Neurosurg. 2009; 111(5): 433-6 (IVSL).*

8. *Andrew S Blum; Leslie Huszar and Francisco Talavera : Clinical Utility of Evoked potentials. 2010;http:// emedicine medscape.com/article/1137451 overview.*

9. Raj K; Narayan and Suzanne Kempisty: Closed head injury in Principle of neurosurgery. Newyork. McGraw-Hill Book Company.2011; 19.301-30

10. Lachapelle J. ; Ptito A. and Michelle Mckerral : Visual information processing in adults with traumatic brain injury.2005; 2 (51): 3010-3023.

11. *Robert LFolmer, Curtis Billings, Anna Diedesch Rouse, Frederick J Gallun, Henry L Lew: Electrophysiologicl assessment of cognition and sensory processing in TBI: Application for diagnosis, prognosis and rehabilitation. International Journal of Psychophysiology.2007; 82(1):4-15(IVSL).*

12.*Padula E V; Argyris S and Ray S: Visual evoked potentials (VEP) evaluating treatment for post –trauma vision syndrome (PTVS) in patients with traumatic brain injury. Brain Injury.2004; 8(2):125-133.*

13.*Lachapelle J; Quimet Catherine; Bach Michael; Pitito Alain and McKerral Michelle: Texture segregation in traumatic brain injury –a VEP study. Vision Research.2004; 1(44): 2835-2842.*

14.*Gaetz M: The neurophysiology of brain injury Clinical Neurophysiology J. 2004; 115:4-18.*

15.*Xi-Ping Chen, Lu-Yang Tao and Andrew CN Chen: Electroencephalogram and evoked potential parameters examined in Chinese mild head injury patients for forensic medicine. Neurosci Bull.2006; 22(3):165-70.*