The Role of Brainstem Auditory Evoked Potential in Patients with Moderate Closed Head Injury

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Summary:

Background: The high prevalence of head injury among civilian populations and the provision of the adequate hospitals services have become matters of worldwide concern. Brain-stem auditory evoked potentials (BAEPs) have been shown to be of highly resistant to systemic factors and toxic or metabolic derangements, making them particularly useful in differentiating reversible brain-stem dysfunction from that due to structural disruption.

Objectives: The purpose of this study was to demonstrate the changes in brainstem evoked response (BAER) in patient with moderate closed head injury and assessing again these changes in BAER six months later as follow up indices.

Material and methods: This is a prospective study conducted in the unit of Neurophysiology /Hospital of Neurosurgery/Baghdad from November 2010 to February 2012, using Esoata 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 (GCS) (9-12 score) undergo BAER and only 86 patients will be followed up by BAER and both were compared to control group of 55 healthy subject.

Results: Central wave latencies of wave III, IV and V of BAER of right and left sides at early measurements were found to be significantly prolonged in comparison to that of healthy subject, Moreover, 6 months later measurements of right and left sides were significantly prolonged as compared to healthy subject at an early measurement, which indicates significant improvement. However 6 months later measurements have significant reduced toward normal when compared to that at early measurements and with control group.

Conclusion: This study revealed that head injury had serious effect on the brain functions reflected by changes in brainstem auditory evoked response which needs long time to return to normal levels.

Key word: head injury, Brainstem auditory evoked Response.

Introduction:

Traumatic Brain injury (TBI): is defined as damage to the brain resulting from an external mechanical force, such as rapid acceleration or deceleration, impact, blast waves, or penetrating a projectile object (1). In head injury, brain functions temporarily or permanently impaired and structural damage may or may not be detectable with the current technology (2).

Diffuse axonal injury is a common finding after TBI, 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 (3). The annual incidence of TBI in the United States has been estimated to be 180-250 cases per 100,000 population and 229 per 100,000 population in England (4). About (75-80%) have mild head injuries; the remaining injuries are divided equally between moderate and severe categories (5). Mechanism attributed to TBI involved acceleration, deceleration and rotation, which cause microscopic damage to neurons, cerebral axons and circulation (6). 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 (7). The Brainstem Auditory Evoked Response (BAER): Is an auditory evoked potential extracted from ongoing electrical activity in the brain and recorded via electrodes placed on the scalp. The resulting recording is a series of vertex positive waves of which I through V are evaluated. These waves, labeled with roman numerals in Jewett and Williston convention, occur in the first 10 milliseconds after onset of an auditory stimulus. The BAER is considered exogenous response because it is dependent upon external factors (8 and 9). BAERs are very resistant to alteration by anything.
other than structural pathology in the brainstem auditory tracts (10). The purpose of this study was to demonstrate the changes in brainstem evoked response (BAER) in patient with moderate closed head injury with assessing again these changes in BAER six months later as follow up indices.

Subjects and Methods:
Two groups of subjects were included in this study: the patients and control groups. This study was performed at Department 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 and 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, 27 female and 28 male with an age ranging from (8-44) years old with a mean (26.1±9.49) years. 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 a previous brain disease or head injury. 3. No history of drug taking. The Brainstem Auditory Evoked Response (BAER) study was performed within the first 3 days of injury, moreover, 6 months later we followed up (86) out of (93) patients (41 female and 45 male) by BAER. The BAER was performed by using Esoata Italian machine, the amplifier of the machine is attached with a thin durable, fiber-optic tube to the machine. The amplifier bandwidth is set to 1-30 Hertz, amplifier sensitivity is 100 milli volts and base time is 20 milliseconds. The system is equipped with electronic apparatus of automatic rejection of artifacts. The subject was lying supine comfortably on the coach , the scalp was cleaned and sterilized with rectified spirit, cerebral responses were registered over Cz (at vertex ) by 2 needle electrodes and references was situated at the ipsilateral mastoid ( M1 and M2), and grounding needle electrode was placed at Fpz, the impedance below 5KΩ, then stimulation were introduced to the subjects by repetitive auditory rarefaction click stimuli delivered monaurally by the headphone; stimulus frequency was 12 HZ, intensity 95 decible (dB), the contralateral ear was masked with white noise at 65dB, sweep time was 20 milliseconds(ms) and band pass filter was 100-1500Hz. Two channel montage is used: channel 1 is ipsilateral ear to vertex, and channel 2 is contra lateral ear to vertex. Because of relative vertex positivity, the waveforms are recorded as upward deflections. 5000 averaged signal for each side were recorded.

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:
In order to exclude the influence of age and gender as a variable that might affect the present electrophysiological study, the measured parameters of healthy subjects and head injured patients were classified according to the age and gender. Table (1) showed that, the age and gender difference has no effect therefore; the parameters were pooled together and were considered as one group accordingly.

Table1: Demographic data of patients with head injury & control.

<table>
<thead>
<tr>
<th></th>
<th>Patients n=93</th>
<th>Controls n=55</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean±SD (Range)</td>
<td>26.9±11.95(5-51)</td>
<td>26.1±9.49(8-44)</td>
<td>0.67</td>
</tr>
<tr>
<td>No %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>49</td>
<td>52.7</td>
<td>28</td>
</tr>
<tr>
<td>Female</td>
<td>44</td>
<td>47.3</td>
<td>27</td>
</tr>
</tbody>
</table>

P value > 0.05 was considered as statistically non significant.

In this study we focus on the changes of central waves (III, IV and V) latency of BAER as a result of moderate closed head injury. Regarding right side of BAER a significant difference (P<0.0001) was found between wave III latency of the patients with head injury at an early measurement (5.05±0.50msec.) when compared to the patients with head injury after 6 months later measurement (4.73±0.48msec.). Moreover, there was high significant difference when compared wave III latency measurement of control group (3.62±0.14msec.) to that at an early measurement and after 6 months later measurement. On comparing the wave IV latency of patient at an early measurement (6.00±0.55) and at 6 months later measurement (5.82±0.48msec.) with that of control (4.71±0.22msec.) a significant difference (P<0.0001) was found. Regarding wave V latency, there
There is a significant difference (P<0.0001) on comparing early measurement to that of the control group and 6 months later measurement. (table 2).

Regarding central wave latency of left side BAER we found that wave III of the patient at an early measurement (5.04±0.5msec.) was significantly prolonged as compared to the 6 months later measurement and that of control group (4.73±0.48msec. and 3.65±0.13msec.) respectively, in addition when compared the wave IV latency at an early measurement (6.02±0.51msec.), there was a significant prolongation (P=0.001and 0.0001) as compared to 6 months later measurement and to that of control group accordingly.

It was found that there was a significant prolongation (P<0.0004) of wave V latency at an early measurement (6.95±0.39msec.) when compared to that at 6 months later measurement (5.46±0.12msec.).(table 2).

### Table 2: Characteristics of studied group.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Head injury early measurement n=93</th>
<th>Head injury 6 months later measurement n=86</th>
<th>Control n=55</th>
<th>P value Early vs control</th>
<th>P value Early vs 6 months</th>
<th>P value 6 months vs control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rt.BAER-III latency(msec.)</td>
<td>5.05±0.5</td>
<td>4.73±0.48</td>
<td>3.65±0.14</td>
<td>&lt;0.0001</td>
<td>&lt;0.0001</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>RT. BAER IV (msec.)</td>
<td>6.04±0.55</td>
<td>5.82±0.48</td>
<td>4.71±0.22</td>
<td>&lt;0.0001</td>
<td>0.821</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Rt. BAERV. Latency (msec.)</td>
<td>6.98±0.39</td>
<td>6.65±0.38</td>
<td>5.47±0.12</td>
<td>&lt;0.0001</td>
<td>&lt;0.0001</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Lt. BAER-III latency(msec.)</td>
<td>5.04±0.5</td>
<td>4.73±0.48</td>
<td>3.65±0.13</td>
<td>&lt;0.0001</td>
<td>&lt;0.0001</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Lt. BAER IV latency (msec.)</td>
<td>6.02±0.51</td>
<td>5.81±0.37</td>
<td>4.71±0.19</td>
<td>&lt;0.0001</td>
<td>0.001</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Lt. BAER V Latency (msec.)</td>
<td>6.95±0.39</td>
<td>6.74±0.38</td>
<td>5.46±0.12</td>
<td>&lt;0.0001</td>
<td>0.0004</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

Data presented as a mean ± SD.
P< 0.05 statistically considered significant.

**Discussion:**

In this study it was found that patient with moderate head injury at an early measurement showed highly significant prolongation of central wave latencies (III, IV and V) of right and left sides of BAER in comparison to that of healthy subject which indicates structural damage because Brain-stem auditory evoked potentials (BAEPs) have been shown to be highly resistant to systemic factors and toxic or metabolic derangements, making them particularly useful in differentiating reversible brain-stem dysfunction from that due to structural disruption (Duric, Milenkovic and Jolic et al., 2004). Moreover when comparing the central wave latencies (III, IV and V) with that at 6 months later measurements for both sides we found highly significant reduction in waves latencies at 6 months later measurement indicating improvement, however it was found that at 6 months later measurement the central wave latencies still show significant prolongation when compared to healthy subject reflecting that although there was improvement but still showed abnormality. The prolongation of central wave latencies (III, IV and V) of BAER of right and left sides are due to impaired conductivity of the auditory pathway at the brainstem level (11). Moreover, the abnormality of wave V latency indicates supratentorial brain injury (12, 13), while the abnormality in latency indicates structural alteration of brain after TBI (14). All previous findings are similar with our findings. Other researcher found that abnormality on testing of central auditory function continues up to several years post-TBI (15). BAER abnormalities are reported even in mild traumatic brain injury that can be result in auditory dysfunction at the brainstem level as evidenced by delayed latencies(16), however this study include a moderate head injury that the abnormality is more evident. Moreover, Thatcher and Cantor and McAlaster they were evaluated head injured patients at time of admission by BAER and they were found out prolongation of waves I, III and V latencies of and they were follow up their patients 12 months after injury and they had been demonstrated that waves I, III and V remained abnormal (15), and this consisting with our results.

**Conclusion:**

Patients with moderate closed head injury at an early measurement showed prolonged values of central wave latencies III, IV and V of BAER of right and left side as compared to that of healthy subjects, while patients 6 months after injury measurements still have prolonged values of central wave latencies (III, IV and V) of BAER of
right and left side as compared to that of healthy subjects. But have lower values of that as compared to patients at early measurements. i.e moderate traumatic brain injury causes structural changes as indicated by the prolongation central wave latencies of the right and left sides of BAER, and these changes lasts even 6 months after that.

Authors Contributor:
Dr. Maha Kamil: Preperaion, Performing and doing the test of the research.
Prof. Dr. Najeeb Hassan Mohammed: This is to certify that this paper is preparad and performed under his supervision. However it is derived from PhD thesis.
DR. Anwar Noori Hafth: Support and assist Dr. Maha in performing the test of this research. In addition to provide specific instruments, apparatus and patients to accomplish this research.

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