

Analysis of Electroencephalogram Resting State in Diffuse Axonal Injury – A Pilot Study

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Abstract

Traumatic brain injury (TBI) is caused by an external mechanical force. The most common causes of TBI are car crash, falls, assaults and thefts and accidents during recreational activity. The acceleration-deceleration mechanism responsible for DAI, often damage the lateral and ventral regions of the frontal and temporal lobes. Deficits in attention and memory, difficulty in learning new information, solving problems and planning are common sequelae. Impulsivity and lack of self-control are also common after a TBI. Our goal was to correlate functional changes obtained in the electroencephalogram (EEG) with the cognitive decline in diffuse axonal injury (DAI) and understand the pathophysiology of this injury. Ten patients with diffuse axonal lesion and 5 control subjects were recruited by the Group of Cognitive Rehabilitation after TBI in the Division of Neurology, Clinics Hospital, Faculty of Medicine, University of São Paulo. A high-resolution EEG with 128 channels were performed at the Psychiatry Institute (LIM-23), University of São Paulo. The cortical sources of EEG rhythms were estimated by analysis of low resolution brain electromagnetic tomography (LORETA), which revealed that patients with DAI had reduced alpha activity and increased theta activity, indicating a slowing of brain activity. In contrast, control subjects showed a predominance of alpha activity and hippocampal activation during the exam, which was not observed in the group with DAI. The DAI patients showed reduced brain activity and little or no hippocampal activation, which is consistent with memory complaints presented by these patients.

Keywords: Electroencephalogram; Traumatic brain injury; Diffuse axonal injury; Low resolution brain electromagnetic tomography

Abbreviations

TBI: Traumatic Brain Injury; DAI: Diffuse Axonal Injury; EEG: Electroencephalogram; CAP Pesq-Ethics in Research Committee of the Clinics Hospital of School of Medicine; University of São Paulo; LIM-23: Laboratory of Medical Investigation on Psychiatry Institute; LORETA: Low Resolution Brain Electromagnetic Tomography

Introduction

Traumatic brain injury (TBI) is a non-degenerative and non-congenital insult to the brain from an external mechanical force. It is associated with a diminished or altered state of consciousness and it can lead to permanent or temporary impairment of cognitive, physical, and psychosocial functions [1]. The most common causes of TBI are car crash, falls, assaults and thefts and accidents during recreational activity [2].

The acceleration-deceleration mechanism, responsible for diffuse axonal injury [DAI], causes the fast rotational forces that are attributed to shear strain, damaging the axons [3]. This mechanism often damages the lateral and ventral regions of the frontal and temporal lobes. Deficits in attention and memory, difficulty in learning new information, solving problems and planning are common sequelae.

Impulsivity and lack of self-control are also common after a TBI [2]. The EEG immediately after TBI initially shows epileptiform activity [4], followed by suppressed cortical activity - which may last from seconds to about a minute [5]. Many patients return to normal within an hour, while others continue presenting focal or generalized slowing - which can last from weeks to a few months [6].

Although many neuroimaging techniques can show changes in the brain parenchyma following a DAI, there is a lot to learn about the relationship between the structural changes in the brain observed in a neuropathological examination and the objective evidence of dysfunctional electrophysiological changes measured with an electroencephalogram [EEG] [7]. EEG has been employed clinically as a measure of brain function [8]. Our interest is to know if the electrophysiological changes are persistence's years after a TBI.

Objective

Correlate functional changes obtained in the electroencephalogram (EEG) with the cognitive decline in diffuse axonal injury (DAI) and understand the pathophysiology of this injury.

Methods

Ethics statement

This study was approved by the Ethics in Research Committee (CAPPesq) of the Clinics Hospital of School of Medicine, University of São Paulo. All recruited participants provided written consent.

Participants

10 patients with diffuse axonal lesion and 5 control subjects were recruited by the Group of Cognitive Rehabilitation after TBI in the Division of Neurology, Clinics Hospital, Faculty of Medicine, University of São Paulo.

We diagnosed patients with DAI based on the following criteria [9]:

- 1) A loss of consciousness from the time of injury that persisted beyond 6 h;
- 2) No apparent hemorrhagic contusion on computed tomography (CT);
- 3) The presence of white matter injury on MRI.

Patients who participated in this study were examined in the chronic stage, during cognitive neurorehabilitation in our hospital. Three researchers selected the samples-2 neurosurgeons and 1 neurologist after evaluation by Neuroimage Service. The sample was not randomized. Most of our patients came into our group 2, 3 years after TBI with cognitive complaints (memory problems were one of the most common complaints). For that reason we decided to investigate if the electrophysiology of the hippocampus was affected in these patients.

Data acquisition

A high-resolution EEG with 128 channels were performed at the Laboratory of Medical Investigation on Psychiatry Institute (LIM-23), University of São Paulo. Placement of scalp electrodes (referential montage) followed the international 10–10 system [10].

These recordings were performed with digital equipment (Brain Vision). Sampling frequency was 10000 Hz. The impedance of all electrodes was maintained below 10 kΩ. Our recordings were performed at rest state. Subjects sat in a reclined chair for approximately 25 min. During this period the subjects kept their eyes closed most of the time (20 minutes). When drowsiness was noticed, they were asked to open their eyes.

An EEG technician and neurophysiologist were present during the entire recording session to observe the behavioral state of the patient and to monitor on-line for signal quality.

Analysis

The data from baseline EEG were processed off-line using the software of Brain Vision (Analyzer). The EEG was band pass filtered for 0,5-30 Hz. Samples were selected by visual inspection, by two independent neurophysiologists, in order to get epochs of 40 seconds (several 10- to 15-s periods edited to produce a single 40-s file) that were free of eye blinking, drowsiness, muscle movements, or equipment-related artifacts. The frequency domain analysis was performed using the Fast Fourier Transform and the cortical sources

of EEG rhythms were estimated by analysis of Low Resolution Brain Electromagnetic Tomography (LORETA).

Results

Demographic measures for the groups are reported in Table 1. Age ranged from 20 to 60 years (mean age: 38,6 years old) in the diffuse axonal injury group. Age ranged from 24 to 55 years old (mean age: 33,2 years old) in the control group.

| DAI | | | Control | | |
|------|--------|-------------|---------|--------|-------------|
| Case | Gender | Age (years) | Case | Gender | Age (years) |
| 1 | Male | 53 | 1 | Female | 55 |
| 2 | Male | 39 | 2 | Male | 31 |
| 3 | Female | 20 | 3 | Male | 24 |
| 4 | Male | 60 | 4 | Male | 28 |
| 5 | Male | 60 | 5 | Female | 28 |
| 6 | Female | 20 | | | |
| 7 | Female | 20 | | | |
| 8 | Female | 48 | | | |
| 9 | Male | 32 | | | |
| 10 | Female | 34 | | | |

Table 1: Demographic characteristics of the groups

To illustrate the findings two sections of EEG traces were chosen - one of a patient with DAI (Figure 1a) and one from a control subject (Figure 2a).

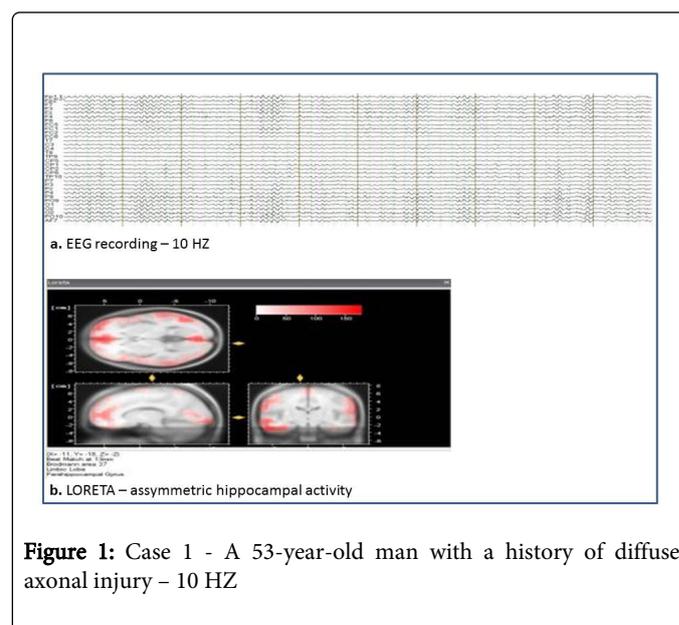


Figure 1: Case 1 - A 53-year-old man with a history of diffuse axonal injury – 10 HZ

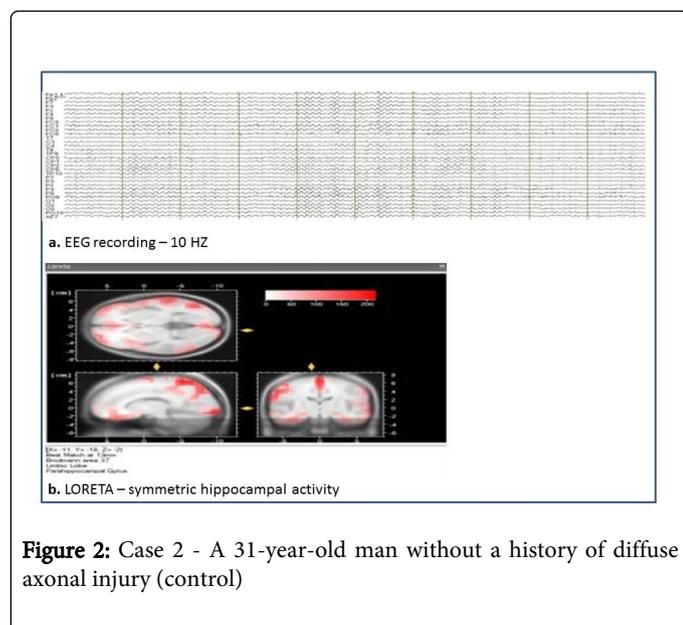
Three patients in the DAI group had symmetric activity in LORETA and the other seven patients had an asymmetric activity

(Table 2). In contrast, all the subjects in the control group had symmetric activity in LORETA (Table 2).

| DAI | | Control | |
|------|----------------------------------|---------|---------------------|
| Case | EEG | Case | EEG |
| 1 | 10 Hz – AHA R | 1 | Low amplitude – SHA |
| 2 | Low amplitude –AHA L | 2 | 10 Hz – SHA |
| 3 | 9 Hz – AHA R | 3 | Low amplitude – SHA |
| 4 | 8-9 Hz – AHA L | 4 | 9 Hz - SHA |
| 5 | Low amplitude –AHA R | 5 | 9 Hz - SHA |
| 6 | Low amplitude –AHA L | | |
| 7 | 9-10 Hz – SHA | | |
| 8 | 9 Hz – AHA L | | |
| 9 | 7-8 Hz – SHA | | |
| 10 | Generalized Identification – SHA | | |

Table 2: EEG of the groups. AHA – asymmetric hippocampal’s activity; SHA – symmetric hippocampal’s activity; R - right hippocampus shows less activity; L – left hippocampus shows less activity

Both traces showed symmetry and revealed a posterior 10 Hz activity. Analysis by LORETA of the chosen patient with DAI showed an asymmetry in the hippocampal activity - the right hippocampus has less activity than the left (Figure 1b). In the analysis of the chosen control subject, the LORETA showed that both hippocampi have a symmetric activity (Figure 2b).



Discussion

The electrical activity is a relatively sensitive index of pathophysiological brain’s response to immediate and secondary TBI. Thus, is closely related to the outcome of the patient’s situation [11].

Quantitative EEG is a valuable method for evaluating brain function in healthy and also in diseased states [12]. But its use as a technique for mapping injured brain areas has not been sufficiently explored [13].

The ratio theta / alpha increases after a mild TBI and tends to return to normal within weeks to months [14]. The quantitative EEG also shows an immediate reduction in the mean frequency of alpha and increase in theta slow activity. These changes usually take weeks to months to resolve. The improvement is associated with reduction of symptoms [6]. We asked ourselves if these changes could also been seen in the chronic stage after a TBI.

LORETA analysis revealed that patients with DAI had reduced alpha activity and increased theta activity, indicating a slowing of brain activity. In contrast, control subjects showed a predominance of alpha activity and hippocampal activation during the exam, which was not observed in the group with DAI.

In conclusion, the DAI patients showed reduced brain activity and little or no hippocampal activation, which is consistent with memory complaints presented by these patients.

Future studies should include a larger sample group and a more detailed analysis of the data.

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