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Assessing Traumatic Brain Injuries Using EEG Power Spectral Analysis and Instantaneous Phase

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Abstract— **Although mild traumatic brain injury (mTBI) occurs commonly, little is known about how multiple mTBI incidents accumulate over time to produce serious morbidity or how the extent of injury can be quantified. This work presents a rat model that uses deceleration-induced brain trauma and an implantable EEG system for recording injury-induced changes in brain activity. Specifically, we present an analysis method to assess and quantify mTBI by combining information derived from EEG power spectral analysis and EEG phase shifts. We found that in different frequency bands, both EEG power spectra and the instantaneous phases of the two EEG channels before the impact were different from those measured after the impact. This study shows that EEG analysis can be used as a tool to identify and assess brain related injuries.**

I. Introduction

Traumatic Brain Injury (TBI) is a term used to describe the damage induced into the brain by traumatic events such as motor vehicle accidents, falls and other trauma. TBI events are classified as mild traumatic brain injury (mTBI) when loss of consciousness, confusion and disorientation are limited to short periods of time. Despite being classified as mild, mTBI may cause a vast series of symptoms and disabilities that can adversely affect an individual's quality of life.

mTBI is challenging to diagnose, even advanced medical imaging techniques, such as MRI and CT can fail to assess the magnitude of the accumulated brain damage. Our long-term goal is to establish a rat model for studying the cumulative effects of multiple mild traumatic brain injuries. We are also interested in studying how EEG can be used to quantify the severity of accumulated TBIs in this model. EEG has been shown to be highly sensitive (96%) in identifying Traumatic Brain Injury (TBI) and post-concussion syndrome [1]. This paper presents preliminary methodology and data from our model. Specifically, we demonstrate how combining information derived from EEG power spectral analysis and phase shifts can be used to assess the presence of brain injury. In order to accurately identify damage induced into the brain by head trauma, we recorded two-channel EEG signals from 350-g adult male Sprague Dawley rats using a surgically implanted EEG acquisition and telemetry system (Data Sciences International, St. Paul, MN). We then subjected these rats to controlled traumatic brain injuries using a high-speed impact system [2], subjecting each rat to a deceleration of 80g on two separate occasions. EEG measurements were made both before and after the experimental impact to allow for a comparison of the baseline activity of a specific rat to data recorded from the same rat after the traumatic impact. We first computed EEG power spectra, we then divided every EEG channel into six clinical frequency bands and measured changes in the signal analytic phase shifts (also known as instantaneous phase) [4,5] in each frequency band, before and after the traumatic impact. The instantaneous phase has been proven, together with some standard EEG processing, to be a valuable tool to measure and investigate synchronous components in EEG activity [5].

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II. Material and Methods

A. Wireless EEG System Implant

Each Sprague Dawley rat was implanted with a wireless EEG acquisition device, TL11M2-F40-EET, (Data Sciences International (DSI), St.Paul, MN). These devices allowed us to record two EEG channels via wireless communication. The EET system is composed of two major components: two pairs of biopotential leads, which are connected to a device body, containing the battery and the electronics module that is able to measure, digitize and wirelessly transmit biopotential voltage fluctuations. The body of the device was implanted subcutaneously along the dorsal flank between the forelimb and hind limb through a dorsal incision. The lead pairs from the transmitter were led subcutaneously to the skull and the bare ends placed in contact with the dura mater through holes in the skull and kept in place by specific screws, as shown in Figure 1. Electrical insulation between the electrode-screw contacts and the surrounding tissue was guaranteed by a coating of dental acrylic cement.

All surgical procedures were performed under aseptic conditions and surgical anesthesia was attained by injecting a solution of ketamine and xylazine. All procedures were carried out in accordance with the National Institutes of Health Guide for the Care and Use of Experimental Animals and were approved by the Temple University Institutional Animal Care and Use Committee.

B. High Deceleration Impact System

To recreate a controlled traumatic brain injury, we used a custom-made linear impact system (Actuator: PT-USA, SpeedLine WH120). This system consisted of two parallel 6 m long tracks, of which one track was active and the other was passive. The former was driven by a servomotor and it was used to accelerate a freely sliding cart mounted on the passive track.

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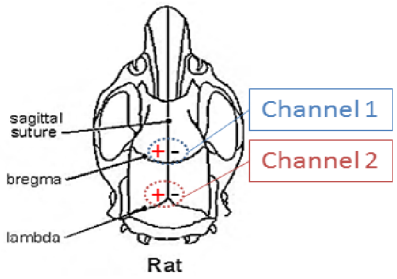

Figure 1: Electrode Placement

The instrumented rat was anesthetized and placed in a silicone enclosure that was then mounted on the sliding cart of the passive track. The cart was accelerated and allowed to collide with a shock absorber placed at the end of the track. The collision system was designed to subject the rat's brain to linear decelerations of 80g along the dorso-ventral axis. The traumatic impact was repeated twice per each animal.

C. Data Acquisition

We acquired two-channel differential EEGs for each experimental subject during different recording sessions. Our main intent was to include in our EEG acquisitions various rat behavioral states. In particular, given the known difference in EEG activity between sleep and wakefulness states [3], we acquired distinct sessions both with the rat asleep (anesthetized) and awake.

The first set of EEG data was acquired immediately after the surgery, with the rat still under anesthesia, to check whether the wireless device implant had been successful. More data were recorded 24 hours after the surgery when the effects of the anesthetic were no longer present.

Then we allowed ten days for the rat to recover from surgery before performing the experimental impacts. The day of the impact, further recordings were performed with the rat asleep, immediately before and after the traumatic impact. Moreover we acquired data with the rat awake, right before and 24 hours after the impact.

Every recording session consisted of 20 min of EEG acquisition with the rat freely behaving in its cage.

Acquiring signals over multiple days allowed us to gather information on each rat's baseline activity under different behavioral conditions. This helped us include in the our analysis the EEG features that changed with the subject's behavior or physical state as well as those features mainly associated to the effects of the impact. This allowed us to better assess our ability to detect mTBI in a more realistic environment.

D. Signal Processing

The EEG signals were low-pass filtered with cutoff frequency set at 100Hz and wirelessly transmitted to a computer, where they were digitized using a sample frequency of 500 Hz, using a PCI acquisition card and the acquisition software Dataquest A.R.T. both from DSI. The stored files were then imported into Matlab (MathWorks, Natick, MA), which was used to carry out signal processing and analysis.

We divided the recorded EEG channels into the traditional clinical frequency, Delta Band (0.5-3.5Hz), Theta Band (3.5-7Hz), Alpha Band (7-13Hz), Beta Band (13-22Hz), Gamma Band (22-64Hz) and Mu Band (8-12Hz).

The first feature we have used as measure of EEG variation was the signal spectral power, in different frequency bands. The average normalized spectral power in each band and in the entire frequency range of interest was computed for the two EEG channels. Then the data obtained during pre-impact acquisitions were compared to post-impact data to identify changes in spectral power and identify induced brain damage.

The second feature taken into account in our analysis is the signal instantaneous phase, which is the imaginary part of the analytic representation of a real-valued signal. In order to obtain the analytic representation of the two EEG channels we used the Hilbert Transform [6]. More specifically, we computed the instantaneous phase for each channel before and after the crash for each frequency band as follows.

The Hilbert Transform $\hat{x}(t)$ of a signal $x(t)$ is defined in the time domain as:

$$\hat{x}(t) = x(t) * \frac{1}{\pi t} \tag{1}$$

where $*$ denotes the convolution operator.

Alternatively the Hilbert Transform can be defined in the frequency domain using the Fourier Transform as shown in (2):

$$\hat{X}(\omega) = -j\, sgn(\omega) X(\omega) \tag{2}$$

where $sgn(\cdot)$ denotes the signum function.

The effect of the Hilbert Transform is to create a copy of $x(t)$ where each cosine component is shifted to the right by $\frac{\pi}{2}$ radians. Note that, unlike other transforms, the Hilbert Transform converts the time signal into another time signal. This property can be used to build a time signal called analytic signal $x_a(t)$ defined as:

$$x_a(t) = x(t) + j\, \hat{x}(t) \tag{3}$$

The instantaneous phase of $x(t)$ can be derived either as the imaginary part of its analytic representation or alternatively as its Hilbert Transform.

Then we investigated how the relation between the instantaneous phases of the two EEG channels changed before and after the impact. In order to quantify the changes related to the experimental impact, we used correlation, as defined in (4), as a measure of strength of the linear relation between the two channels.

$$corr(X,Y) = \frac{cov(X,Y)}{\sigma_X\, \sigma_Y} = \frac{E[(X-\mu_X)(Y-\mu_Y)]}{\sigma_X \sigma_Y} \tag{4}$$

where E[·] is the expected value, cov(X, Y) is the covariance and σ_X and σ_Y are the standard deviations for X and Y, respectively.

Correlation values close to 1 or -1 indicate that there is a strong linear relationship between the signals. On the contrary, values close to zero indicate the absence of linear relationship.

III. Results

Figure 2 shows the average normalized EEG spectral power in each frequency band, with respect to the two EEG channels, before and after the traumatic impact.

For each experimental subject, the power spectra are normalized by the largest frequency component. The normalized power spectra are then averaged across subjects. The results are displayed in Figure 2, where the light blue and black bars represent respectively data recorded before and after the impact.

It is notable how the total power decreases for both channels after the impact. In particular, Channel 1 displays a 60% decrease in its power content with respect to its original value before the impact, while Channel 2 decreases by 64%. Furthermore these power variations are mainly generated by changes that occur exclusively in the Delta band.

As previously mentioned, in order to quantify differences in the EEG channels, we measure the correlation coefficients between the instantaneous phase of the two channels, before and after the impact. To better emphasize the instantaneous phase changes related to the impact, in Figure 3 we show the differences between the correlation coefficients measured between the two channels before the impact and the correlation coefficients measured after the impact. This allows us to identify those frequency bands in which we find the highest EEG synchrony variation related to the traumatic injury. In Figure 3 we can see that largest changes occur in the Theta and Beta bands.

Figures 4 and 5 show the instantaneous phases of both channels, respectively in the Theta and Beta frequency bands. The correlation coefficients between the instantaneous phases of the two channels are shown to quantify the relation between them before and after the crash.

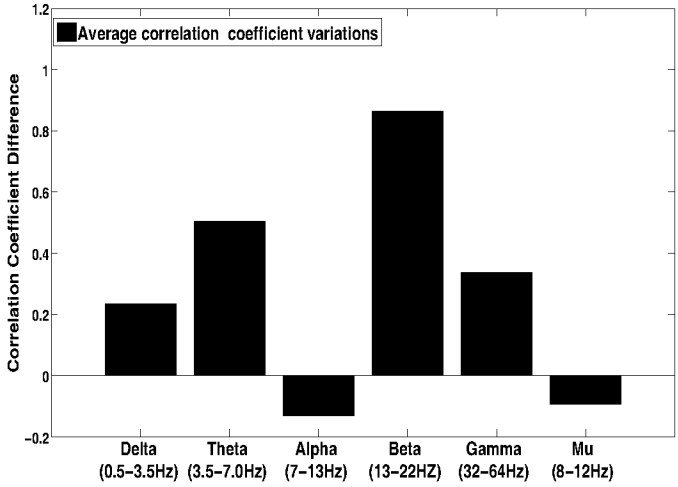

Figure 3: Average correlation coefficient variations of the instantaneous phase shifts, in clinical frequency bands, before vs. after the impact

The correlation coefficients before and after impact, in the Theta band, are respectively 0.23 and 0.88, as shown in Figure 4. In the Beta band, they are 0.05 and 0.85, as shown in Figure 5. The data indicate increased in-phase activity in the two respective regions of the brain.

As previously mentioned, we acquired EEG data from rats over different days and under different behavioral conditions. Given the EEG high dependency on subject's activity, we considered and analyzed, for the purpose of the presented analysis, only data acquired when the rat was awake.

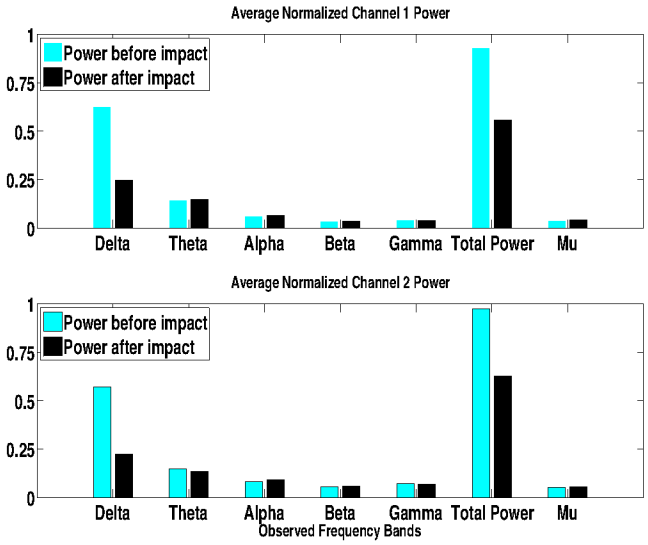

Figure 2: Average normalized power for both channels, in the clinical frequency bands, before vs. after impact

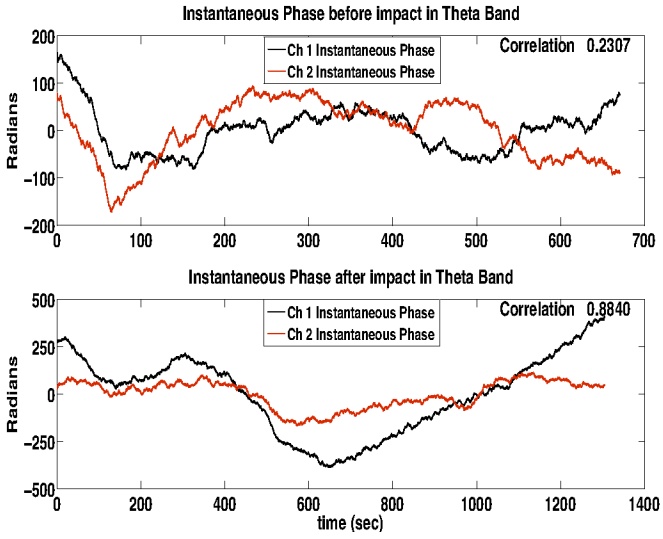

Figure 4: Unwrapped and detrended Instantaneous Phase of the two channels before and after the crash in Theta frequency band

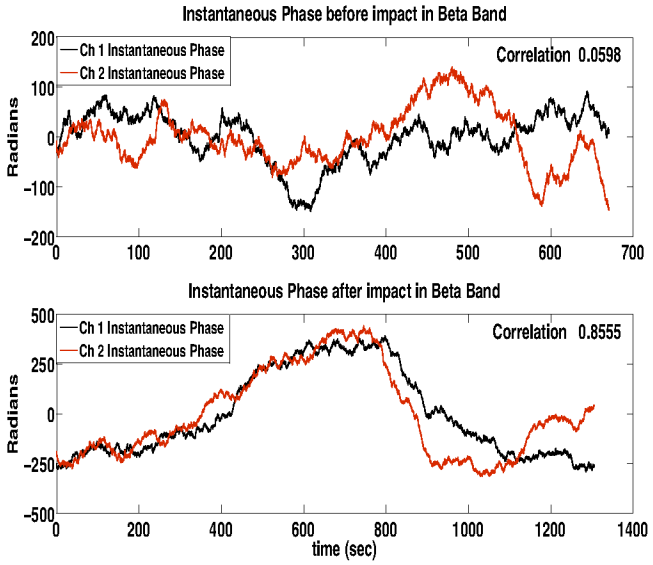

Figure 5: Unwrapped and detrended Instantaneous Phase of the two channels before and after the crash in Beta frequency band

IV. Discussion

Over the last few years, several studies have proved the efficacy of EEG power spectral analysis in studying and assessing traumatic brain injury in humans [1,4]. In contrast, the rat model we are developing will allow us to precisely control the severity, number, and spacing of the impact conditions of every experimental subject. This will allow us to correlate the observed EEG spectral variations to traumatic brain injury in order to better quantify the induced damage. These results can then be correlated to measures of neural tissue damage (i.e. brain slice analysis or diffusion tensor imaging).

Our findings indicate a decrease in Delta band power after impact as well as an increase in cortical synchrony in the Beta and Theta bands. Future work will develop this relationship to determine how EEG can be used as a quantitative assessment tool for measuring traumatic brain injury.

V. Conclusion

The results of this preliminary study demonstrate that EEG analysis can be a successful tool to assess brain injuries. In particular, we have found that EEG spectral power in clinical frequency bands, displayed evident changes that could be used to identify the presence of traumatic brain injury.C We also found that changes in the EEG channels' instantaneous phases could be used as a measure of brain's synchrony variations that were caused by a traumatic impact.

Showing that EEG analysis can be effectively used as a powerful tool to identify and assess brain related injuries, is the first step towards extending this technology to human subjects, where non-invasive techniques can be easily used to record EEG signals, making the proposed method a valuable fast, non-invasive and non-harmful diagnostic tool for quantifying brain injuries.

VI. Future Work

In future work, we plan on using the proposed processing techniques as part of a more complete EEG analysis tool, which will aim to fully assess and quantify mTBIs.

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