Quantitative and Qualitative Analysis of Ambulatory Electroencephalography During Mild Traumatic Brain Injury

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Objectives: To characterize the neurophysiological changes in a patient with mild traumatic brain injury (mTBI) and to compare these changes with a small cohort of patients with neurocardiogenic syncope, an analogous cause of transient neurological dysfunction.

Design: Case report and quantitative analysis of a small electroencephalography (EEG) cohort.

Setting: University-affiliated teaching hospital.

Patients: A 64-year-old man with mTBI recorded on ambulatory EEG. The comparison group was 4 patients with spontaneous neurocardiogenic syncope during continuous video EEG recording.

Intervention: Quantitative and qualitative analysis of EEG.

Main Outcome Measures: Changes in quantitative EEG measurements between the patient with mTBI and the comparison group.

Results: In the patient with mTBI, there was an abrupt decrease in high-frequency (beta) power and alpha-delta ratio immediately after the injury and a corresponding increase in lower-frequency (alpha, theta, delta) power. The change in beta power resolved within 5 minutes of the injury, but the increases in low-frequency power persisted up to 20 minutes after the injury before resolving. Similar but smaller changes were seen in the patients with syncope, but these changes resolved within 5 minutes, with no intermediate or long-term changes.

Conclusions: The quantitative EEG changes in mTBI are initially similar to those in syncope, suggesting acute transient cortical dysfunction. However, there are longer-lasting increases in low-frequency power during mTBI, suggesting ongoing disruption of cortical-thalamic circuits.

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TRAUMATIC BRAIN INJURY IS DEFINED AS "an alteration in brain function, or other evidence of brain pathology, caused by an external force." In mild traumatic brain injury (mTBI), the evidence of brain pathology can be subtle and transient, and it is often difficult to document and quantify the immediate neurological manifestations. Attempts have been made to understand the pathophysiology of mTBI through the use of neurophysiological techniques such as electroencephalography (EEG). Because mTBI is an unexpected phenomenon, there have been no previously published reports of EEG during an event, to our knowledge. We report a case of a patient monitored by ambulatory EEG (AEEG) during a minor motor vehicle collision leading to mTBI. To put these EEG changes into context, we have compared changes in quantitative EEG measurements with a group of patients with reversible brain dysfunction due to cardionic syncope.

REPORT OF A CASE

Ambulatory EEG was performed on a 64-year-old man with epilepsy secondary to a right frontal pilocytic astrocytoma that was surgically resected in March 2009. He had no definite clinical seizures for more than a year prior to undergoing a 72-hour AEEG evaluation in January 2011 to rule out unrecognized seizures. On the first day of his AEEG evaluation, he had a motor vehicle collision while driving home. According to onlookers, another vehicle hit his car from the side as he entered an intersection, and the patient was determined not to be at fault in the collision. The patient did not recall the impact, and he did not remember the events of 2 to 3 minutes afterward. He is uncertain whether he lost consciousness, but he was fully alert and aware when he was evaluated by emergency medical technicians within a few minutes of the collision. He was brought to an emergency department, where he had a normal neurological examination and com-
puted tomography of the head. He fractured his shoulder but was otherwise symptom free with a normal neurological examination when he was evaluated by a neurologist 3 days later. The AEEG captured the moment of the collision and continued recording for 2 days afterward.

PATIENTS

Our EEG database was reviewed from January 2009 to March 2011 for all patients who had spontaneous neurocardiogenic syncope during video EEG recording. Patients were included if they had a loss of consciousness associated with a change in blood pressure and/or heart rate and typical EEG changes. Patients were excluded if they had a seizure prior to syncope (ictal arrhythmia) or if the patient was comatose or asleep at baseline. The study design was approved by the Columbia University institutional review board.

EEG ANALYSIS

Raw EEG tracings were reviewed manually to identify general changes in EEG morphology and possible sources of artifact. The start time of each event was determined by identifying the first definite qualitative change in EEG background. Quantitative EEG evaluation was performed using magic marker functions in Insight II Software (Persyst Development Corporation, Prescott, Arizona). Power values for each frequency band were obtained using fast Fourier transform calculations on 4-second epochs of EEG, with 236 data points per second. Values were combined into 4 frequency bands: beta (13-30 Hz), alpha (8-13 Hz), theta (4-8 Hz), and delta (<4 Hz). Relative power ratios (eg, alpha-delta ratio) were also calculated for each epoch. All calculations were made for anterior (F3-C3/F4-C4) and posterior (P3-O1/P4-O1) head regions.

The EEG epochs with muscle and movement artifact were rejected by the Insight II artifact rejecter using a threshold of 0.2. Further data points with prominent high-frequency muscle or electrode artifacts were excluded based on gamma range power (30-64 Hz) greater than 2.1 µV−2/Hz. This threshold was selected through comparing power distribution of segments of EEG with prominent movement artifacts and those without evident artifacts.

Quantitative measurements were calculated for 5 minutes prior to the event and for at least 1 hour after the event. Only awake recordings were included in the analysis, and sleep was excluded by manual review of raw EEG tracings. For graphical representation, data points for each quantitative measurement were generated at 1-minute intervals by calculating the mean of 15 artifact-filtered 4-second epochs. Ninety-five percent confidence intervals were calculated assuming a normal distribution.

RESULTS

Qualitative AEEG during mTBI revealed approximately 3 seconds of diffuse movement artifact followed by an increase in diffuse theta and delta slowing that lasted several minutes (Figure 1). During prior evaluations, this patient’s typical seizures were associated with a prolonged high-voltage ictal rhythm in the right frontal region. There was no such ictal rhythm on AEEG prior to the presumed mTBI. The remainder of the 48-hour AEEG showed intermittent right frontal slowing but no epileptiform discharges and no seizures.

During the study period, there were 10 patients with spontaneous neurocardiogenic syncope recorded on video EEG. Six recordings were excluded because they occurred during coma or sleep or because of insufficient quality or duration of the recording. The 4 patients included in the analy-
sis were aged 32 to 60 years. Three patients were female and 1 was male, and the cause of the syncope was primary bradydyscardia in 2 patients and vasovagal syncope in 2 patients. Two patients had epilepsy but did not have seizures at the time of the syncopal event. None of the patients were taking medications that would have a significant effect on EEG background.

Selected quantitative measurements for each hemisphere are displayed in Figure 2. For the patient with mTBI, there was an increase in delta power in the posterior head regions

Figure 2. Selected quantitative measurements for 5 minutes before and 35 minutes after mild traumatic brain injury (mTBI) and syncope. Error bars represent 95% confidence intervals for 4 patients with syncope.
immediately after the head trauma that persisted for 15 to 20 minutes before returning to the preinjury baseline. There was a smaller increase in delta power in patients with syncope, and this resolved within approximately 5 minutes. Alpha power also increased after mTBI, but the peak was slightly delayed compared with the peak in delta power. There was a persistent increase in alpha power after the collision, and on review of the raw EEG tracing, there were more frequent periods when the patient had his eyes closed after the mTBI. There was only a slight increase in alpha power after syncope, and this lasted less than 5 minutes. Similar changes in alpha and delta power were seen in the anterior head regions, and the changes in theta power were similar to the changes in delta power, and so these are not displayed.

There were subtle, brief (1-3 minutes) decreases in the alpha-delta ratio during mTBI, and similar changes can be seen during syncope. Beta power in the anterior head region decreased slightly during both mTBI and syncope but returned to baseline within 1 to 3 minutes. Similar changes were seen in beta power in the posterior head region, and so these are not displayed.

Early reports of EEG within 24 hours after traumatic brain injury revealed that most patients with mild head injuries had a normal EEG. Some patients had subtle slowing of the background EEG within 15 to 30 minutes of injury, but this generally resolved after 30 minutes. Quantitative changes in EEG after mTBI have included reduction in alpha mean frequency and increase in the theta-alpha ratio, and these changes gradually resolved over weeks to months. Other authors have used a composite index of multiple quantitative EEG characteristics (including power and coherence analysis) to demonstrate significant differences from baseline EEG in football players with concussion that persisted for at least 8 days after injury. In our patient, we did not see definite quantitative or qualitative changes beyond 15 to 20 minutes after the injury. However, because this was an AEEG, it contained significant movement and muscle artifact, and it was not possible to appreciate subtle abnormalities more easily observed during a routine EEG in an immobilized, resting patient.

Decreased high-frequency power and increased delta power are nonspecific, and similar changes are seen in a variety of disorders, including syncope. There were several similarities between our patient and the patients with syncope, including a transient increase in low-frequency activity and a transient decrease in high-frequency activity, as well as a transient decrease in the alpha-delta ratio. However, the EEG of patients with syncope returned to baseline within 5 minutes, while our patient had a persistent increase in low-frequency power for an additional 15 to 20 minutes. There was also a relative preservation (or even increase) in alpha power after the injury in our patient, possibly due to more frequent periods of eye closure.

Mild traumatic brain injury is thought to be caused by shearing forces generated by sudden acceleration, deceleration, and rotation of the brain, causing transient neurological dysfunction. In general, higher-frequency EEG rhythms (beta and gamma) are produced by cortical generators, while lower-frequency rhythms (delta) are the result of cortical-thalamic synchrony and can become more prominent when cortical-thalamic connections are disrupted. We propose the following neurophysiological mechanism for EEG changes in our patient. The initial sudden acceleration and deceleration caused mild, reversible diffuse cortical dysfunction, leading to a brief, transient decrease in cortically generated high-frequency rhythms, resulting in a decrease in beta power. This injury was mild, and so there was a rapid return to normal beta power within 5 minutes. At the same time, shearing forces and rotational acceleration resulted in acute disruption of cortical-thalamic connections that was longer lasting than the cortical dysfunction and led to a more prolonged increase in delta and theta power. As this dysfunction resolved, the delta and theta power returned to baseline.

In conclusion, the EEG in acute mTBI demonstrates neurophysiological changes that are consistent with acute transient cortical dysfunction and longer-lasting disruption of cortical-subcortical connections. Compared with syncope, the changes associated with mTBI are longer lasting, confirming that even mTBI can result in widespread, potentially long-lasting effects.

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REFERENCES


