The Electroencephalogram in Acute Fatal Head Injuries*

E. Rodin, J. Whelan, R. Taylor, T. Tomita, J. Grisell, L. M. Thomas, and E. S. Gurjian
Lafayette Clinic and Department of Neurology and Neurosurgery, Wayne State University School of Medicine, Detroit, Michigan

The earliest investigations dealing with large series of electroencephalographic (EEG) changes in patients with head injuries are those by Glaser and Sjaardema, Jasper et al., and Williams. The latter two reports followed a number of patients through their clinical recovery. It was observed that random slow wave activity dominated the tracings initially, normal frequencies were suppressed, and epileptiform discharges were sometimes noticeable. In addition to diffuse changes, focal areas of abnormality were seen when the general disturbance subsided. It was felt that improvement in the EEG correlated quite well with the clinical recovery of the patient. Further studies were carried out by Dow et al. who were able to obtain records within 30 minutes after injury. It was observed that a greater percentage of abnormalities was found in those records that were taken early than in cases where the recording was obtained later. A detailed study of more severe injuries was then reported by Dawson et al. with emphasis on serial recordings. In that study it was noted that, in contrast to Dow’s observations, the EEG might show little or no abnormality soon after the trauma, even though the patient had a definite brain injury, and the EEG was said to be a more valuable adjunct in the sub-acute and chronic phase of the illness. These differences in opinion are likely to be due to the difference in the patient population. Dow’s group being mildly injured while Dawson’s group was severely ill. Dawson also pointed out that general suppression of electrical activity was seen in some of the records; and when this phenomenon occurred within 30 hours after injury, the illness was uniformly fatal.

Further papers on large series of head injuries were then published by Meyer-Mickeleit, Whelan et al., Frantzen et al., Mensikova and Vrbik, Geets, Debandt, Grindel and Podgornaya, and Jellinger et al. An excellent clinical and electroencephalographic study dealing with acute head injuries in boxers, which includes also a detailed bibliography, was presented by Larson et al.

There is, therefore, a considerable amount of literature available on EEG changes in head injuries, but most reports deal with patients who ultimately recover and whose injuries were, therefore, not of the gravest severity. During 1961 and 1962 a study was undertaken by the department of neurosurgery at the Detroit Receiving Hospital to evaluate the effects of most intensive treatment of patients with severe head injuries. Serial EEGs were obtained as part of the general work-up. From the electroencephalographic point of view it was hoped to obtain answers to three main questions:

1) Does the EEG differentiate between severe head injuries that will be fatal and those in which the patient may survive?  
2) Do the EEG and clinical changes progressively deteriorate in fatal head injuries, or are there marked fluctuations in the course of the illness?  
3) What is the effect of hypothermia on the EEG of a person with severe brain injury?

Material and Methods

Prior to collection of the data, a coding system was prepared to include detailed clinical and electroencephalographic description of the patient’s state. This was done in order to be able to use general purpose digital computers for subsequent data analysis. The system was based on a similar one used for coding patients with convulsive disorders.

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Fifty-two patients were then examined by means of serial EEGs. The clinical state of the patient was coded by a neurosurgeon resident at the time of each EEG. A Grass model 8D EEG machine was employed and, as most of the patients had surgical dressings covering the scalp, needle electrodes were used in bi-polar montages. The patients were kept in an intensive treatment unit which provided facilities for hypothermia. The neurosurgical resident and a nurse were in constant attendance. The number of EEGs obtained varied depending on the clinical course of the patient. The most severely ill patients had studies at 1 to 2 hourly intervals, while others had daily EEGs. Hypothermia was applied by means of a cool room and cooling blanket.

Only 7 of the 52 patients survived the 6-month period of observation. It was, therefore, not feasible to perform statistical analyses involving differences between severe head injuries which can be survived by the patient and those which are uniformly fatal. Of the 45 patients who died, 3 had to be eliminated from the series either because the EEGs were of poor quality or because it was doubtful whether cause of death was indeed the head injury. The data was, therefore, analyzed on 42 patients and this report deals only with these findings. There were 7 females and 35 males in the group. The ages ranged from 24 to 85 years with a mean age of 54 years.

In 34 patients the injury was due to compression of the head, 3 had penetrating high velocity injuries, 2 penetrating low velocity injuries, and the type of injury had not been recorded in 3 others. Thirteen patients had linear skull fractures, 6 comminuted skull fractures, 4 depressed skull fractures and 11 had multiple skull fractures. In 8 instances intracranial blood clots were removed prior to the first EEG. The severity of the injury is best demonstrated by the state of consciousness of the patients at the time of first EEG. All patients were either stuporous or comatose.

Autopsies were performed on 26 of these patients. Nineteen were found to have subdural hematomas, 2 had epidural hematomas, 1 an intracerebral clot, 1 a subarachnoid hemorrhage, 1 a basilar artery infarct, and 1 had contusion of the brain. A number of patients had a combination of these findings.

A total of 283 EEGs was obtained. The first EEG was recorded within 6 hours after the injury in 15 patients, between 7 and 12 hours in 7 patients, between 13 and 24 hours in 7 patients, between 25 and 48 hours in 7 patients and between 49 and 93 hours in 6 patients.

Twenty patients died within 48 hours after injury and 21 died between 3 days and 28 days. One additional patient survived for 3½ months. His last EEG was recorded 88 days after injury.

In order to detect possible clinical and electroencephalographic differences between the group of patients who died early and those who survived the first 48 hours, the data dealing with the clinical and electroencephalographic features of the patient at the time of the first recording were compared. Seventy-eight different clinical and electroencephalographic variables were selected and a test of significance between means was done for all continuously distributed variables. For those variables dealing with either/or statements, like: presence or absence of sleep spindles in the recording, and presence or absence of multiple skull fractures on x-ray, the test of significance was chi square. In order to obtain information about the course of the illness, 28 patients were subsequently selected who had had at least 3 EEGs. Means were computed on 47 variables for the first, middle and last EEG. Only 7 patients were not subjected to hypothermia at any time during the course of their illness. These were then contrasted on a variety of selected EEG variables with patients who had been placed on hypothermia throughout the entire course of the study. As the number of individuals involved in this particular phase of the project is quite small, no computer studies were carried out.

Results

Differences Between Patients Dying Within 48 Hours and Those Who Died Subsequently. Table 1 lists the clinical and EEG variables that showed no statistically significant differences between the patients who died within the first 48 hours after injury and those who died later. The variables that do show statistically significant differences are recorded in Table 2. It can be seen that patients who die within the first 48 hours tend to be comatose with unreactive, dilated pupils and absent corneal reflexes and a lower diastolic blood pressure; they show a flatter EEG with less focal EEG abnormalities.

Clinical Variables and EEG Background Activity. The course of the illness of the 28 patients who had 3 or more EEGs is diagrammatically represented for some of the major variables in Fig. 1. The mean values are plotted, regardless of the length of time elapsed between EEGs, for the first, middle and last recording. The curves show essentially a steady deterioration of systolic blood pressure, diastolic blood pressure, pulse, and respiration. There is a gradual increase in