

Ictal and Electroencephalographic Sequelae of Head Injuries*

DIHARAMPAL,* S. N. PATHAK,** P. N. TANDON***, BALDEV SINGH****

Ictal and electroencephalographic sequelae of head injuries have been described in 60 cases. Only two parameters have been studied; the correlation of the type of clinical fits left as long term effect, to the time taken by the emergence of first fit after the head injury and the type as well as the frequency of fits and EEG findings in relation to the duration of initial unconsciousness after the brain trauma.

It has been pointed out that the tentative inference drawn from these findings is that, (i) shorter the time interval between the head injury and emergence of first fit, the higher the incidence of focal fits, (ii) if the interval is between 1 and 5 years, there is more likelihood of generalized fits, and (iii) with longer time interval, the possibilities are greater than chance factors that temporal lobe seizures will be manifested. Finally if more than 15 years elapse after head injury before the fits appear, the incidence of post-traumatic epilepsy becomes negligible.

It was further noted that the longer the duration of initial unconsciousness the greater the variety and frequency of fits and more complicated the epileptiform EEG pattern.

The tentative pathophysiology has been surmized on the basis of concussion or contusional damage to the brain leading to regional derangement of cerebral circulation, injury to brain stem or other neural circuits in various permutations and combinations. The effects of neurotransmitters on the damaged brain has been emphasized.

Post-traumatic epilepsy as an after effect of head injuries has been recognized for centuries. This subject however has assumed great importance recently because of increased incidence of accidents due to travel by automobiles, railways and air. Extensive industrial undertakings and

other hazardous occupations as that of builders, miners and divers have further added to the risk of injuries of the head and therefore of the brain.

This paper deals with only two parameters, i.e., the ictus and EEG changes

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* Research Officer, PL-480 Epilepsy scheme, Dept. of Neurology, AIIMS, New Delhi

** Professor & Head of Dept. of Neurology, AIIMS, New Delhi

*** Professor & Head of Dept. of Neurosurgery, AIIMS, New Delhi

**** Emeritus Professor, Dept. of Physiology, AIIMS, New Delhi

seen as sequelae in head injury patients examined in the special epilepsy clinic at All India Institute of Medical Sciences, run with the financial aid from PL-480 under agreement No. 01477-2 dated 2-9-69. The 600 epileptics seen during the three years in this clinic has 60 patients in whom the epileptic phenomenon could be attributed as sequelae to the head injury. The selection of these patients was made according to the criteria laid down by Walker², i.e.,

1. The attacks were bonafide epileptic. Functional spells were carefully scrutinized and excluded.
2. There was no evidence of pretraumatic convulsive manifestations.
3. No associated systemic or cerebral disease.
4. Only those patients who had either radiological evidence of fracture skull or had authenticated history of unconsciousness lasting for varying period of time or both were included.
5. By and large electroencephalographic changes and type of epilepsy showed fairly good correlation with each other.

Out of the 60 patients, 41 were male and 19 female. There were 19 patients below the age of 14 years and 41 above this age. Most of the children had ages between 5 to 14 years and the adults 14 years to 45 years. From the point of view of studying ictal and EEG sequelae these 60 cases were analysed in the following context :

I. Relationship between time elapsed after head injury when first fit appeared and the type of clinical fit recorded at the time of examination in the clinic. Only 56 cases could be taken up for this purpose. Four cases had to be excluded because of the vague history of the recorded time.

II. Relationship between duration of unconsciousness due to the head injury and :

- a) the type of clinical fits,
- b) frequency of fits in relation to the duration of unconsciousness due to head injury and
- c) EEG findings.

Only 40 cases out of 60 could be included in this group. In 20 cases the duration of unconsciousness was not reliable.

I. Relationship between time elapsed after head injury when first fit appeared and the type of clinical fit as recorded when examined in the clinic (Table I).

The time elapsed after head injury before the first fit appeared was arbitrarily divided into, 12 hours to one year, one year to 5 years, 5 years to 10 years, 10 years to 15 years, 15 years to 20 years, 20 years to 25 years, 25 years to 30 years and 30 years to 35 years. The clinical fits chosen were, generalized i.e. grand mal, temporal lobe fits and focal fits other than temporal lobe fits. Statistical validity was found only in the first four time period groups. The trends indicated were :

- a) With time duration of 12 hours to 1 year before the first fit, generalized and temporal lobe fits were significantly less when compared to the focal group.
- b) In the group between 1 to 5 years the generalized fits were higher in number when compared to temporal lobe and focal fits.
- c) With time lapse of more than 6 years upto 10 years the temporal lobe fits showed higher incidence as compared to the generalized attacks.
- d) If the first fit did not appear for fifteen years or longer after head injury, the chances of post-traumatic epilepsy were considerably reduced.

II. Relationship of the duration of initial unconsciousness after head injury to (a) type of clinical fits (b) frequency of fits and (c) EEG activity respectively (Tables II a, b & c)

It has been found that when unconsciousness lasted for less than 4 minutes (4 cases) the focal fits which became generalized predominated, the frequency of fits was mostly once in 3 to 6 months and the EEG records showed either no abnormality or just high voltage paroxysmal activity or hemispheric voltage asymmetry. When unconsciousness lasted from 5 minutes to 6 hours largely from 30 min-

utes to 2 hours (26 cases) generalized grand mal fits were the commonest type (12 cases) whilst focal becoming generalized and focal appeared in that order i.e. in 8 and 6 cases respectively. The frequency of fits in this group was one or more fits per week in fifteen per cent, 3 to 4 fits per year in fifteen per cent and markedly variable fits in the rest of the cases. The EEG activity predominantly was epileptogenic e. g. spikes or spike and slow wave, or irregular spikes and slow waves respectively (18 cases). With increased duration of unconsciousness i.e. from 6 hours to several days the clinical fits were either generalized or multicentric i.e. more than one variety of fits, frequency of these fits were once or more in a week whilst the EEG abnormality consisted of nearly 90% epileptogenic variety.

Briefly in this study of Ictal and Electroencephalographic sequelae of head injuries there are the following points brought out. (i) There is a time dependence for the type of clinical fits focal, temporal and generalized which is related to the duration after the head injury when the first fit appeared. (ii) The incidence of post-traumatic epilepsy became negligible if the first fit did not appear for 15 years after the head injury. (iii) The duration of unconsciousness was somewhat correlated to the type of clinical fits, their frequency and the associated EEG findings.

TABLE-I.
Relationship between Time Elapsed after Head Injury before the First Fit
Appeared and the Type of Clinical Fits which Emerged as Sequelae

| Time elapsed after head-injury before the first fit appeared | Type of fit which emerged as sequelae | | | |
|--|---------------------------------------|-------------------|------------|--------------|
| | Generalised attack | Temporal lobe fit | Focal fits | No. of cases |
| 12 hours to 1 year | 3 | 2 | 9 | 14 |
| 1 to 5 years | 9 | 4 | 5 | 18 |
| 5 to 10 years | 3 | 6 | 0 | 9 |
| 10 to 15 years | 3 | 3 | 2 | 8 |
| 15 to 20 years | 2 | 2 | 0 | 4 |
| 20 to 25 years | 1 | 0 | 1 | 2 |
| 25 to 30 years | 0 | 0 | 0 | 0 |
| 30 to 35 years | 0 | 0 | 1 | 1 |
| | 21 | 17 | 18 | 56 |

TABLE-II. a.
Relationship between Duration of Initial Unconsciousness after Head Injury and the Type of Clinical Fit

| Duration of initial unconsciousness after head-injury | Generalised fit | Focal becoming generalised | Focal | Multi-centric | No. of cases |
|--|-----------------|----------------------------|-------|---------------|--------------|
| Less than 5 minutes | 1 | 3 | 0 | 0 | 4 |
| From 5 minutes to 6 hours (Mostly 30 minutes to 2 hours) | 12 | 8 | 6 | 0 | 26 |
| From 6 hours to several days | 6 | 0 | 0 | 4 | 10 |
| Total | 19 | 11 | 6 | 4 | 40 |

TABLE—II b.
Frequency of Fits in Relation to the Duration of Initial Unconsciousness after Head Injury.

| Duration of initial unconsciousness after head injury | No. of Cases | Frequency of fits. |
|---|--------------|---|
| Less than 5 minutes | 4 | Mostly once in 3 to 6 Months |
| From 5 minutes to 6 hours (Mostly 30 minutes to 2 hours) | 26 | 50% cases had one or more fits per week 15% cases had 3 to 4 fits per year |
| From 6 hours to several days | 10 | Nearly all cases had at least one attack per week. |
| Total | 40 | |

TABLE—II c.
Relationship between Duration of Initial Unconsciousness after Head Injury and EEG Activity.

| Duration of initial unconsciousness after head injury | No abnormality | High voltage paroxysmal | Hemispheric EEG asymmetry | Epileptiform activity | No. of cases |
|---|----------------|-------------------------|---------------------------|-----------------------|--------------|
| Less than 5 minutes | 2 | 1 | 1 | 0 | 4 |
| From 5 minutes to 6 hours (Mostly 30 minutes to 2 hours) | 2 | 4 | 2 | 18 | 26 |
| From 6 hours to several days | 1 | 0 | 0 | 9 | 10 |
| Total | 5 | 5 | 3 | 27 | 40 |

Discussion

The two points taken up for discussion are :

a) The pathophysiology of the lesion which determines the type of ictal phenomenon as a sequelae and its relationship to the time elapsed when first fit appeared after the head injury. The actual data indicates that the emergence of focal, generalized and temporal lobe fits are time dependent in that sequence (loc cit). These clinical features are explainable on the basis of whether the head injury has produced a concussive effect or has lead to a contusional injury i.e. laceration or haemorrhages in the brain.¹ Concussion of brain has its effects on regional auto-regulation of circulation which precipitates convulsive activity early after head injury and subsequently leads to persistent regional damage giving rise to focal fits. More severe injury particularly if brain stem is involved delays the time of the first fit after head injury because of the prolonged unconsciousness and as a sequelae centrocerebral or grand mal fits appear comparatively more frequently. Temporal lobe fits take the longest time for appearance particularly in head injury during child hood because of functional maturity of this region taking longer time normally due to ontogenetic sequence of brain growth.

b) As to how the duration of initial unconsciousness in head injuries influences the type, the frequency of fits and EEG findings which emerge subsequently, is explainable by the work of Walker² on the depth electrode studies in human beings and in monkeys. He has described three distinct systems of preferential pathways which get involved in various combinations to give rise to varied clinical features and the EEG epileptogenic activity.

Firstly there is the frontal granular cortex, caudate nucleus and dorso-medial

thalamic nucleus circuit which forms a consistent interacting complex. Secondly there is the central cortex, the putamen and the lateral nuclear mass of the thalamus which is another closely related system. Thirdly the temporal cortex, the amygdala, hippocampus and septal nuclei which also is a functionally related group. Each of these can influence the other and increase the complexity. All the three systems in addition are under influence of the activity of the reticular tissue of the brain stem and the deep nuclei of the cerebellum. The picture in due course becomes extremely complex producing a variety of clinical manifestations in the form of type and frequency of fits and EEG manifestations such as spikes, spike and slow waves and multiple irregular spike and slow waves when the abnormal formation of neurotransmitters like nor-adrenaline and acetylcholine become operative in addition to the derangement in the neural circuitry.

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