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It is well known to all that accidental injuries are the leading cause of morbidity and mortality all over the world and the leading cause of death in individuals between 1 and 44 years of age. Head injuries are present in more than 50 percent of trauma related deaths. In most countries primary and continuing care of patients sustaining a head injury is the responsibility of general surgeons rather than neurosurgeons. Despite the expansion of neurosurgical services this situation is likely to continue and therefore a general surgeon requires a knowledge of principles and practice in this field. Since most of the patients do not require any neurosurgery, the major steps in the care of such patients are medical, diagnostic and nursing, and it is these steps which will determine the outcome far more frequently than any surgical manoeuvres.

The final neurological status of the patient, who has sustained brain trauma is the sum of irreversible damage acquired at the time of initial injury and the damage that is the consequence of secondary insults. At the time of initial injury one portion of brain may sustain irreversible damage from which it will recover over a period of months. Secondary insults that result
in worsening of patient's neurological deficits include:

1. Systemic disorders as hypoxia or hypotension.
2. An expanding intracranial mass as subdural, epidural or rarely an intraparenchymal haematoma.
3. Sustained raised intracranial pressure.

Several forms of intervention have been proposed to enhance brain's normal repair process but little can be done about it, only swift recognition or prevention of these secondary insults offers the best chance of improving the prognosis of patients who have sustained a brain injury.

Patients with mild head injuries and brief loss of consciousness are often expected to make an uneventful recovery. In fact, these patients are found to have a surprising degree of post injury disability in the form of persistent headaches, memory deficits, and difficulties with activities of daily living that persist for months following the accident. Rimel, Giordani, and Barth (1981) reported that one third of patients who had sustained minor head injuries had not returned to gainful employment in 3 months following injury. Patients who have sustained mild head injuries but have a severe headache, lethargy or restlessness should be observed for 24 hours. If the patient shows any deterioration in neurological status or demonstrates any signs of a focal neurological lesion on examination, CT scan should be performed. Patients who
have sustained a moderate head injury are likely to be lethargic, stuporous, or combative when they first regain consciousness. Ten to fifteen percent of patients entering the hospital with a moderate head injury are found to have a focal intracranial lesion. Nearly all patients who sustain injury of this degree suffer from persistent headaches, memory difficulties and difficulties of daily living for months following injury. Three months following a moderate head injury, two thirds of patients still do not return to their normal work.

A severe head injury is defined by a score of eight or less on Glasgow coma scale. Almost forty percent of patients sustaining a severe brain injury have a focal intracranial mass lesion. Signs of severe neurologic dysfunction, such as an abnormal motor response, abnormal oculocephalic reflexes or bilateral fixed pupils are common in this group of patients. Each of these brain stem reflexes can be demonstrated in approximately one third of severely injured patients and is associated with an increased mortality. Mortality has also been shown to be proportional to patients' age. Other stated factors for poor outcome include presence of focal intracranial lesion or elevated intracranial pressure. Frequent neurological examination is necessary to detect any change in patient's neurological status.
There has long been felt a necessity for a drug which may affect recovery from craniocerebral injury in a positive way and minimize the sequelae thereof. Chemicals such as magnesium pemoline, methylphenidate and Pyritinol appeared on therapeutic scene. But of these Pyritinol is the only drug that is still the subject of continuing research using modern and sophisticated methods. Substantiated reports of therapeutic efficacy of pyritinol have appeared as recently as 1980. So it is increasingly used in more than 70-80 countries around the world for the management of various neurological disorders, including sequelae of brain trauma.

Pyritinol was first introduced in India in 1967 and was considered a neurotropic compound. It is said to enhance glucose uptake and utilisation in neuronal cells and also to have some effect in GABA metabolism in brain, stimulate nucleic acid metabolism and protein synthesis, activates cerebral cortex, limbic system and reticular activating system as well (last three effects are due to increased uptake of GABA).

Molecule of Pyritinol is derived from a combination of two molecules of pyridoxine (Vit B₆). But the actions of the two compounds are dissimilar. The short chemical name is pyritinol or Pyrithioxine.
Pyritinol molecule was first synthesised in 1957 and subjected to detailed investigation which confirmed absence of any serious side effects or teratogenic effect in animals. Many people still challenge its role in neurological abnormalities including sequelae cranioencebral injuries and say that is empirical.

So the present study was undertaken to see and compare the development and duration of sequelae of cranioencebral injury using pyritinol and comparing them with control group of similar patients.