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Cerebral Palsy is defined as a non progressive, neuromuscular disorder of cerebral origin and includes a group of heterogeneous disorders of variable severity, ranging from minor incapacity to total handicap. In India, it is the second commonest cause of crippling in childhood, after polio; with an approximate incidence of 1-2 per 1000 population. This means that nearly 1 baby out of every 200 live births is born with brain damage. In a survey it was observed, that out of every 1000 patients attending out patient department of pediatrics, nearly two were suffering from cerebral palsy (Prabhakar and Kumar 1983).

The incidence of cerebral palsy is increasing at an alarming pace, in developed, as well as developing or underdeveloped countries (Brown and Fulford 1984). Two basic reasons are responsible for this rising incidence, in developing or under developed countries – firstly the poor obstetric facilities available in rural and remote areas, where even the mild obstetric complications could not be managed suitably; and
secondly the improving standard of neonatal nurseries in affluent cities where more and more low birth weight and sick babies are being saved. But in developed countries, latter reason is solely responsible for rising incidence (Brown and Fulford, 1984).

First published study relating to cerebral palsy, dates back to late eighteenth century, when Little, in his historical article held the obstetric factors responsible, for the causation of cerebral palsy (Little, W.J., 1961). Since then several obstetric and perinatal factors had been discussed as the causative or predisposing factors; the important ones are - prematurity, Birth asphyxia, Birth trauma, Hyperbilirubinemia, Hypoglycemia and LBW - babies.

This had long been a disputed point that what is the maximum age of permanent brain damage for the causation of cerebral palsy. Perlstein et al fixed the ceiling of eight years for the permanent brain damage to occur (Perlstein et al, 1975). But still later the American Academy of cerebral palsy lowered this limit to five years of age (Davis and Hill 1980).
The diagnosis of cerebral palsy is clinical. Patients present with delayed mile stones and perinatal history usually reveal some abnormality either before, during or after delivery. The neurological examination reveals upper motor neurone signs, with or without abnormal movements. Most of the patients also have some associated defects like epilepsy, mental retardation or problems related to speech, hearing, vision or behaviour.

Epilepsy is one of the commonest associated problems in patients of cerebral palsy. The incidence of epilepsy in cerebral palsy is much higher than that in general population. While 7.3 per 1000 children, in general population, suffer from epilepsy (Kanwisk, A, and Sehgal, H, 1980), about 20-50% patients of cerebral palsy have epilepsy (Brown and Fulford, 1964). The diagnosis of epilepsy and its management is of paramount importance in patients of cerebral palsy as it adds further to a crippled baby. Moreover, if epilepsy is controlled further brain damage is at least avoided (Lennon, 1942 and Waterlain, 1978). Some patients with epilepsy are misdiagnosed as suffering from other conditions and therefore not treated properly,
while some patients are diagnosed as having epilepsy when in fact they have other problems viz. pseudo-seizures and other disorders simulating epilepsy (Walton, J. 1963) e.g. Breath holding spells, stokes-adams syndrome, carotid sinus syndrome and other cardiac dysrhythmias, syncope, migraine, vertigo, narcolepsy, rage-reaction, panic - attacks and emotional out bursts. On the other hand only abnormal behaviour, sometimes can be epilepsy. As the anticonvulsants, which are not free from side affects, if started, will have to be given for a long period, the diagnosis of epilepsy should be definite. Moreover anticonvulsant, like barbiturates further make a hyperkinetic baby worse.

Though the diagnosis of epilepsy is usually clinical but during the state of dilemma, the electroencephalography is the only investigative tool which may clear ones mind. Even the most sophisticated investigations of modern times like CT-scan, magnetic resonance imaging and positron emission tomography are usually not fruitful. It has also been seen that prognosis of patients of cerebral palsy with abnormal electroencephalographs, is poor, though vice versa is not true. (Gibbs and Gibbs et al, 1963).
No published study had been performed in India which used electroencephalography as the investigating procedure, to evaluate the abnormal EEG - findings in the patients of cerebral palsy. Thinking of its common occurrence, this study was performed on electro - clinical characteristics of cerebral palsy with following aims and objectives:

1. To find out the incidence of epilepsy in cerebral palsy.

2. To find out various electroencephalographic abnormalities in patients of cerebral palsy.

3. To categorize these abnormalities as per well defined neurological subtypes of cerebral palsy.

4. To correlate the electroencephalographic abnormalities with clinical epilepsy.

5. To correlate the Developmental - Quotient with EEG abnormalities in cerebral palsy.

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