REVIEW OF LITERATURE
HISTORICAL ASPECT

The history of cerebral palsy is more than a century old. In his historical treatise, William John Little (1962) for the first time gave a clear description of cerebral palsy and noted its relation with premature birth, difficult labour, and mechanical injuries, during parturition. Some earlier workers had also described cases of cerebral palsy—Andrey (1741), Dalpech (1628) and Haime (1860), quoted by Collier (1924). Mckert (1923) reported 10 cases of infantile hemiplegia, who mainly showed blood on the convexity of brain in each case. Cowars (1826) thought that most cases of cerebral palsy were due to brain damage at birth. Freud (1997) considered the etiology of generalized spasticity to be difficult and premature labour. Collier (1924) thought that the "essential anatomical cause of diplegia is a primary degeneration of cerebral neurones from causes which are at present elusive."
The ancient terminology of "Little's disease," thus originated at that time. But the whole concept of this condition has undergone complete metamorphosis since then, and now the term cerebral palsy has been universally accepted to denote brain damage during the phase of its development. It was in 1930 when Wintrop M. Phelps, grouped various forms of this condition, under a common heading. He coined the term cerebral palsy to indicate that common denominator is a paralysis (Palsy) due to impairment of central nervous system (Cerebral). The old terminology of "Little's disease" is just one aspect of related disorder and now is synonym of cerebral diplegia only (Illingworth and Felani 1958).

**DEFINITION**

Several attempts had been made to define the complete syndrome of cerebral palsy but none had been successful, due to diverse etiologies and manifestations. This had long been a disputed point that what is the maximum age of permanent brain damage which can lead to cerebral palsy or in other words, till what age does the brain grow as cerebral
palsy occurs during the phase of brain's development only. Perlstein et al. (1956) set the limit of eight years, earlier which if any thing goes wrong with maturing brain may terminate into cerebral palsy. However the American academy of cerebral palsy has arbitrarily fixed years as the upper limit for brain damage to occur (Down and Hill, 1980).

Low and Carter (1982) defined cerebral palsy as a group of diverse non-progressive syndromes affecting brain, manifesting as impairment in motor functions and presumed to have had their onset during gestation, during parturition or in childhood. According to Prabhakar and Kumar (1983) cerebral palsy is a persistent but not unchanging disorder of posture and movement due to dysfunction of brain, present before its growth and development are completed. However cerebral palsy is not a static condition and its clinical pattern changes as brain maturation continues throughout the childhood, resulting in dynamic clinical pattern despite a static pathology (Brown and Fulford, 1984).
INCIDENCE AND PREVALENCE

Cases of cerebral palsy constitute 0.66% of the total admission to pediatric ward and form 0.2% of total out patient pediatric cases in India (Srinivasan et al., 1973).

The incidence of cerebral palsy is highly variable as it depends entirely upon the availability of obstetric facilities, socio-economic and cultural status of the population under study. Its incidence is increasing now-a-day, basically due to two reasons—Firstly improving standard of neonatal intensive care units all over the world and Secondly paucity of proper obstetric care in remote areas of under developed and developing countries (Brown and Fulford, 1984).

Its incidence varies from 0.6 to 6.0 per 1000 live births according to various studies. According to a report of Ciba Foundation (1978) the prevalence rate of cerebral palsy in Britain is 1-2.4 per 1000. The overall average incidence is around 2.5 per 1000 live births (Brown and Fulford, 1984). Calculating at
an average rate of 2 per 1000 live births, with 21 million births every year in our country, nearly 42,000 new cases of cerebral palsy are added every year to our population (Prabhakar et al., 1983).

The condition is said to be slightly more common in males and male:female ratio is 58:42, but spastic paraplegia is more common in females, with male:female ratio of 47:53. This difference could be because of the fact that spastic paraplegia is very common in prematures who are more commonly females (Perlstein et al., 1955). In India the sex ratio reported by Sharma et al., (1981) was 50:23.

**CLASSIFICATION**

Since the time of advent, attempts are being made to classify various types of presentations of cerebral palsy. First attempt in this direction was made by Perlstein et al., whose classification with relative frequency is as follows (Perlstein et al., 1935).
A. Spastic Type
   Hemiplegia      68%
   Quadriplegia    33%
   Paraplegia      24%
   Triplegia       17%
   Monoplegia      3.7%
B. Dyskinetic Type
   28%
C. Ataxic Type
   4%

Another detailed classification was produced just one year later by Balf and Ingram (1956):-

<table>
<thead>
<tr>
<th>Neurological Diagnosis</th>
<th>Extent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemiplegia</td>
<td>Right</td>
</tr>
<tr>
<td>Bilateral Hemiplegia</td>
<td>Left</td>
</tr>
<tr>
<td>Diplegia</td>
<td></td>
</tr>
<tr>
<td>- Hypotonic</td>
<td>Paraplegia</td>
</tr>
<tr>
<td>- Dystonic</td>
<td>Triplegia</td>
</tr>
<tr>
<td>- Spastic/Rigid</td>
<td>Tetrplegia</td>
</tr>
<tr>
<td>Ataxic Diplegia</td>
<td>Paraplegia</td>
</tr>
<tr>
<td>- Hypotonic</td>
<td>Triplegia</td>
</tr>
<tr>
<td>- Spastic</td>
<td>Tetrplegia</td>
</tr>
<tr>
<td>Ataxia</td>
<td>Predominantly Unilateral</td>
</tr>
<tr>
<td></td>
<td>Bilateral</td>
</tr>
</tbody>
</table>
6. Dyskinesia
   - Dystonic
   - Chorea
   - Athetoid
   - Tension
   - Tremor

7. Mixed

The most universally accepted classification is that produced by American Academy of Cerebral Palsy (Down and Hill, 1980):

A - Type of Motor disorder -
   (i) Spasticity
   (ii) Rigidity
   (iii) Athetosis
   (iv) Ataxia
   (v) Tremor
   (vi) Hypotonia
   (vii) Mixed

B - Anatomical Distribution -
   (i) Tetraplegia
   (ii) Paraplegia
   (iii) Triplegia
   (iv) Double Hemiplegia
   (v) Hemiplegia
   (vi) Monoplegia
C-Degree of Severity -

(i) Mild
(ii) Moderate
(iii) Severe

The distribution of different types of cerebral palsy had been quite variable. Soon after Perlstein et al., 1955 (above), Salomonsen and Skatvedt produced following figures (Salomonsen and Skatvedt, 1955) -

1- SPASTIC 95.3%
   Hemiplegia 20.3%
   Monoplegia 20.3%
   Others 35%

2- Pure ATETOSIS 14.4%

3- Pure Ataxia 3%

4- Mixed 23.6%

The distribution pattern reported by Srinivasan et al., reveals that Diplegia is the commonest pattern

1- Diplegia 62.7%
2- Atonic Diplegia 7.5%
3- Hemiplegia 20%
4- Dystonic 3%
5- Minimum Brain dysfunction 6.5%
Later Sharma, M. et al., reported same pattern of distribution but with slight different figures (Sharma, M. et al., 1987):

1- Spastic 82.7%
   -Diplegia 45.3%
   -Hemiplegia 34.6%
   -Others 19.9%
2- Athetoid 4.1%
3- Ataxic 0.9%
4- Atonic 5.0%
5- Mixed 7.3%

ETIOLOGY

As it is well known that incidence of cerebral palsy is correlated with abnormal-pregnancy/delivery/birth, the etiology may be classified into prenatal, natal and postnatal causes which are as follows (Carg and Srivastava, 1965).

A- Prenatal Factors - These are responsible for about 30% cases.

1) Genetic factors are-Hemolytic disease of new born and kernicterus, hereditary paraplegia, Familial Tremors, Hereditary athetosis, Familial spastic
paraplegia, Familial paroxysmal choroathetosis.

(ii) **MATERNAL AGE**: Mitchell (1959) found cerebral diplegia to be commoner in children of younger mothers and athetosis and mixed type being commoner in children of older mothers.

(iii) **PARITY**: Skatvedt (1958) found a very high incidence of cerebral palsy in first born and similar was the observation of Garg and Srivastava (1965). But Eastman and Deleon (1955) negated such a relationship.

(iv) **PREMATURITY**: Prematurity stands out to be the single most common factor in the causation of cerebral palsy. Eastman et al. (1955), Churchill (1974), and Prabhakar et al. (1983) reported that the incidence of prematurity in cerebral palsy is three to five times more common than the general population. They all observed that nearly 35% cerebral palsied children are preterm at birth. A premature baby is more liable to birth injury, partial separation of placenta, and other types of antepartum hemorrhage may lead both to premature labour and foetal anoxia thus terminating into cerebral palsy (Garg and Srivastava, 1965).
(v) **MATERNAL-ILLNESS:** Garg et al. (1965) reported various antenatal problems like pre-eclampsia (16.9%), antepartum hemorrhage (11.3%), hyperemesis, vaginal discharge etc. (27.2%). Faber (1947) also reported about 15% cases with history of antepartum hemorrhage.

(vi) **MULTIPLE BIRTHS:** An increased incidence of multiple pregnancies has been reported by Asher et al. (1950) 5.4%, Shyh Jong (1953) 9%, Greenspan et al. (1953) 7.0%, Skatvedt (1958) 6.7% and Garg et al. (1965) 5.6%.

**B- NATAI CAUSES** - Responsible for about 60% (majority) cases. Abnormality of labour is quiet common in cerebral palsied patients, like prolonged labour, breech delivery, cord prolapse, transverse, lie, Caesarian section, precipitate labour. Association of cerebral palsy with these factors had been reported by various workers - Asher et al. (1950), Denhoff et al. (1951), Lilian field et al. (1955). 44% cases in Skatvedt (1958) series had history of abnormal labour while Garg et al. (1965) record such abnormality in one third of their cases.
In the neonatal period, common abnormalities are—asphyxia, cerebral irritation, Jaundice, convulsions and feeding problems, (Garg et al., 1965). Schreiber (1940) noted 70% cases due to Birth Asphyxia. Anderson (1952) stated that a third to a half of all cases of cerebral palsy had evidences of anoxia at birth. Eastman et al. (1955) found foetal-distress to be four times commoner in cerebral palsy than in controls. Garg et al. (1965) reported history of abnormality in neonatal period in 56.4% of their patients.

C- POSTNATAL-CAUSES:— Important postnatal problems associated with cerebral palsy are meningitis, encephalitis, Kernicterus. The incidence of cases of postnatal origin is somewhere between 10% (Perlstein et al., 1952) and 20% (Illingworth, 1953). Mitchell (1959) recorded 12% such cases and Garg et al. (1965) observed 23% patients due to postnatal causes.

Neonatal jaundice, kernicterus and athetosis have got an important correlation (Garg et al., 1965) Rh. incompatibility was observed in 17.5% cases by Brandt et al. (1958) and in 3.9% by Martin (1960). But cases of Rh incompatibility are very few in India due to very low incidence of Rh (D)—negative
individuals in our country (Carg et al., 1965). Circulating unconjugated Bilirubin in blood in the patients of hyper bilirubinemia gains access to blood brain barrier and deposits in Basal Ganglia and cerebellum. Anoxia further accentuates bilirubin toxicity (Dempsey et al., 1966).

**PATHOLOGY**

With the exception of spastic hemiplegia, the lesion in most of cases of cerebral palsy is probably deeply situated in the centre of brain, in the form of dilated III- ventricles as seen in pneumo-encephalograms (Skatvedt, et al., 1955). The intra cerebral hemorrhage (subependymal and intraventricular) is most important factor in spastic diplegia of prematurity (Churchill et al., 1974, Esmem, 1986). Three type of basic defects have been described which either act alone or in combination (Skatvedt et al., 1955).

(i) Cerebral malformations of genetic origin.

(ii) Cerebral developmental inhibition caused by damage to fetus in embryonic period.

(iii) Cerebral damage incurred during process of birth i.e., anoxic brain damage.
Pathological studies on the other hand have resulted in careful dissection of various cerebral abnormalities in patients with non progressive neurologic disorders and have led to attempts, often highly speculative, at formulating their cause. A combined clinical and pathological approach has demonstrated, however, that a given neurologic deficit can result from a cerebral malformation of prenatal origin, a destructive process of perinatal or early postnatal onset or malformation and perinatal trauma acting in concert various. Pathological changes seen are (Menkes, 1980).

(a) Wide spread transsural degenerations, which may be bilaterally symmetrical, involving thalamus and several brain stem nuclei viz. inferior colliculi, superior olive and lateral lemniscus.

(b) Periventricular-encephalomalacia – Bilateral necrosis in periventricular distribution which is accompanied by astrocytic and microglial proliferation,ependymal loss and multifocal subcortical degeneration.

(c) Water shed Infarction – due to sudden arterial hypotension causing cortical infarction of these areas which are supplied by most peripheral
branches of three large cerebral arteries.

Ulegyria and status Marmoratus of Basal Ganglia - This type of lesion is principally seen in full term asphyxiated babies. The lesions are located mainly in peripheral and basal areas of cerebral cortex, involving necrosis of gyri at the depth of sulci and the neuronal nuclei of basal ganglia and brain stem (Brown, 1986).

ASSOCIATED HANDICAPS

The child with damage to the motor mechanisms would be expected to have damage to other parts of the brain as well. Thus it is accepted that the child with cerebral palsy may suffer from a wide spectrum of other neurological disorders since cerebral palsy is merely one manifestation of brain damaged child (Brown and Fulford, 1984). Thus these patients may have associated - mental retardation, epilepsy, visual defects viz. hemianopia, squint, myopia, optic atrophy, cortical blindness, visuo-spatial and visuo-motor problems, gaze, palsy, Hearing defects, speech defects, learning problems, difficulties of cognitive functions, Behavioural problems like reversal of sleep pattern, irritability, slow at feeding, poor concentration span, decreased threshold for fight and flight; sensory problems like astereognosis, sensory in-attention and problems of
communication (Brown and Fulford, 1984, Carg et al., 1965).

Out of all these handicaps, the most significant is epilepsy as it is amenable to treatment. Epilepsy in cerebral palseid children is due to cerebral trauma (Woods, 1957). The overall incidence of epilepsy in cerebral palsy varies from 15% (Pirrie, 1997) to 68% (Yanmet, 1944). This figure does not include convulsions due to other problems, like febrile convulsions. While only 7.3 per 1000 children, in general population suffer from epilepsy (Kaushik et al., 1980), Geiger A.B. (1951) observed epilepsy in 43% of cerebral palseid children while Skatvedt, et al., (1955) 20.6%, Perlstein et al. (1955) 47% and Carg et al., (1965) reported that 23.4% patients in their series experienced epilepsy, in their life.

The correlation of type of cerebral palsy and incidence of epilepsy is disputed. Some workers (Gibbs and Gibbs, 1955) reported highest incidence in atonic diplegia (69%) while Aird and Cohen (1950), Perlstein et al. (1955) and Gibbs et al. (1965) observed
that spastic are more commonly affected by seizures. They reported the figures of 65%, 57% respectively. But it is well known that epilepsy is uncommon in athetoid and diplegic patients (Gibbs and Gibbs 1955), Gibbs et al., 1963 and Garg et al., 1963). According to Gibbs and Gibbs (1955) seizures are more common where arms are more commonly affected.

According to type of seizure, all the workers had reported that grand mal type seizures are the commonest, though incidence reported by them vary. The distribution of epileptics in series of Perlstein et al. (1955) was 53% grand mal type, 24% jacksonian type, 14% generalised type other than grand mal, 7% myoclonic type, 1.6% petit mal and rest 0.4% had psychomotor epilepsy. They also compared these figures with incidence of types of seizures in general population and observed that petit mal seizures were commonest (60%), followed by grand mal (25%), generalised but other than grand mal (10%), jacksonian seizures (3%), infantile myoclonus (1.5%) and psychomotor epilepsy (0.5%). The corresponding figures reported by Garg et al., (1963) are 69% grand mal, 20.7% petit mal and 10.3% jacksonian type of seizures, in patients of cerebral palsy.
ELECTROENCEPHALOGRAPHY

The electroencephalography is a continuous measurement of the constantly varying electrical potential difference between any two points on the scalp or between one point and an inactive reference electrode. First human EEG recording was published by Hans Berger (1929) although animal recordings had been made long before by Caton (1873) and Seck (1890), (Driver et al., 1982). The basic physiology underlying the EEG is largely unknown, but the electrical activity is probably derived primarily from post synaptic potentials in the dendrites of cortical neurones (Kiloh, 1972). The EEG has proven to be a useful tool for investigating specific problems of the CNS. First recording of human epileptic activity was published by Gibbs, Davis and Lennox in 1933 (Driver et al., 1982).

Pediatrics is an area in which EEG has much to contribute but in which a sound knowledge of the range of EEG norms seen in normal children is an essential prerequisite to interpretation. Such profitability has led to a proliferation of machines and several types of machines viz. 8, channel or 12 or 16 or 20 channel
recorders are available in the market and some also record ECG and/or EMG simultaneously. It is customary to operate all the channels at the same sensitivity and with same frequency characteristic (Kiloh et al., 1962).

The placing of electrodes for clinical electroencephalography is usually based on some anatomical landmarks on the external surface of the skull. Its relationship to the underlying parts of brain is of great practical importance, but can not always be easily defined (Hallström, 1963). Several types of electrodes had been used from time to time, notably sphenoidal Needle electrodes (Revit et al., 1960), Nasopharyngeal electrodes (Naylor et al., 1964), solder pellet electrodes embedded in bentonite paste (Taylor et al., 1969) and Naseethmoidal electrodes (Lehtinen et al., 1970). But chloridated silver electrodes with electrode jelly or saline as the contact medium are most satisfactory for general use. Chloriding enables the electrodes to present the same resistance to current flow in either direction, so that distortion of EEG signals does not occur such a electrode is said to be reversible or non polarizable (Kiloh et al., 1962). The interface between the subject and the electrode should
introduce the minimum possible impedance and for this purpose Bentonite paste is generally used (Kiloh et al., 1982).

This problem of electrode placement has been considered by, among others, pamphlons, who suggested in 1956, a system of electrode placement and illustrated their anatomical placement in relation to external skull and the internal cavities. He quoted Nettler's Neuroanatomy to delimit the position of central sulcus and sylvian fissure. In the report of committee on methods of clinical examination in electroencephalography (1957), in which the 10–20 system of international federation was outlined, which is based on the measurement from a landmark on the skull, (Jasper, 1958). Though this 10–20 system has not received universal acceptance, but the advantages of a standardized system far out weigh its short comings.

There are some universal acceptance of the methodology of EEG recording (Kiloh et al., 1982). It should preferably be carried out in recumbent posture, the contact resistances of electrodes should be minimized. A calibration signal, usually 100 µV, is then recorded simultaneously on all channels and the gains adjusted
until each channel gives a deflection of 1 cm. All EEG machines have an input switching unit, with a master selector, whereby the technician can choose from several prewired patterns and individual channel selectors whereby a pattern can be set up at will. These patterns of connection between electrodes and the recording channels are known as montages. In most investigations, the use of a few standard montages is sufficient, but in some cases it will be necessary for additional montage to be used. The type of montages used in EEG recording has a critical influence on appearance of a record, even when these are based on standard electrode placement. The International Federation has laid down following guidelines for design of montages (Jasper, 1958) —

(i) Recording channels should be connected in sequence to rows of equidistant electrodes that lie along antero-posterior or transverse lines.

(ii) The order of the channels, as read from top to the bottom of the recording paper, should in general be such that these recording from
right side of the head come before those recording from the left.

(iii) Channels recording anteriorly should come before those recording posteriorly.

The sense in which the recording pen reproduces the voltage fluctuations, between a given pair of electrodes depends upon the way in which the amplifier input leads are connected to them. Referring to those as lead 1 and lead 2, the convention is that when lead 1 becomes electronegative with respect to lead 2, the recording pen makes an upwards deflection. In diagrams of montages, leads 1 are drawn as full lines and leads 2 as broken lines, so that the relative polarity of a particular discharge can always be deduced. If an arrow between two electrodes is used to indicate the sense in which the channel is connected, it should point from lead 1 to lead 2. There are basically different types of montages — bipolar, unipolar and average reference (Kiloh et al., 1982).

Bipolar montages comprise of segmental linkage of channels along antero-posterior or transverse rows of equispaced electrodes (Jasper, 1958). Each channel is connected between a pair of active electrodes on the scalp and records the potential difference
between them. If a localized discharge occurs at or near the electrode common to two channels, these will deflect in opposite directions - a phenomenon known as phase reversals. This represents the presence of an underlying focus, from where the discharge is originating.

Each channel in unipolar montages records between one active electrode (lead 1) on scalp and one relatively indifferent electrode elsewhere (lead 2). The latter is common to all channels and is known as the common reference. Various sites of this common reference are ears, nose, shin or neck etc. But unfortunately, it is virtually impossible to find a truly neutral reference, as they will pick up certain amount of electrical activity from adjacent parts of brain, and may be contaminated by a variety of non-cerebral potentials, all of which will be registered in all channels for which this electrode is reference (Kiloh et al., 1962).

In an average reference montage, each channel records between an electrode (via lead 1) on scalp and a common reference potential (via lead 2). The common reference potential is usually obtained by joining
all the electrodes on the scalp to a common point through high resistance of equal volume. This system was originally described by Offner (1950) and used by Gold man (1950), after whom it is sometimes known.

The usual standard practice is to record at a paper speed of 30 mm/sec and to adjust master gain control so that the pen excursions fluctuate between 0.5 and 2 cm. If high voltage discharges are anticipated, the gain should be reduced to an extent that allows their wave form to be seen without distortion due to mechanical restriction of pen deflection, (Kiloh et al., 1982). A disturbed or mentally retarded child may need some sedation and for which purpose drugs used are - Quinal barbitone, phenobarbitone, Dichloral phenazone, chlorpromazine, Trimipramine tartarate, promazine and paraldehyde (Kiloh et al., 1982). These drugs themselves also produce effect on the EEG tracing, like beta activity in the form of discrete runs or spindles and typically at a frequency of 18-24 Hz. This activity has fronto-central preponderance and more or less asymmetrical (Brazier et al., 1945).
The terminology committee of International Federation (1966) suggested that the features present in EEG record should be classified into waves, activities, rhythms and complexes and that each feature should be described in terms of its frequency or period, amplitude, phase reactions, quantity, morphology, topography, reactivity and variability (Kiloh et al., 1962).

The principal objective criteria by which a record is assessed are based upon the frequency, amplitude and shape of waves, of which it is composed, upon their spatial and temporal distributions and upon their reactivity to stimulus. An EEG record consists of components of three basic kinds (Kiloh et al., 1962).

(a) Those that are fairly continuous and very often rhythmic.
(b) Those that are transient.
(c) Those that comprise the background activity, upon which the two preceding kinds are superimposed.

As a matter of convenience, the EEG frequency spectrum is divided into bands that are designated as follows (Kiloh et al., 1962) —

delta : less than 4 Hz
theta : 4-less than 6 Hz
alpha : 8-13 Hz
beta : 14 Hz onwards.

A poly rhythmic record is one in which two or more clear rhythmic components are present, whereas the term polymorphic refers to irregular activity, the individual waves of which are of variable period.

A monomorphic wave of less than 80 m.sec, duration is called a spike whereas, one that is of 80-200 m.sec duration is called a sharp wave. The amplitude refers to peak-to-peak value in μV. Whenever it is desired to compare the amplitude of particular component in EEG, it is preferable that an appropriate monopolar montage should be used (Kiloh et al., 1962).

The EEG normally appears as sinusoidal like wave forms of varying frequencies in which the predominating frequency may be normally modified by many factors, for example, opening and closing of the eyes, state of consciousness, and drugs. One of the abnormalities that occurs is that of wave forms that stand out from the background frequency. Focal slowing, often, but not in variable, results from a local disturbance causing
destruction of brain tissue. This abnormality is often seen in tumors, hematomas, strokes, localized infectious or contusions of brain. In contrast, focal spikes are in general, manifestations of irritative lesions or old processes taking months or years to develop. Spikes are often seen with scars or cysts or less commonly, slow-growing tumors. Focal slowing by itself or combined with spikes should, therefore make the physician think about further studies (Lewis et al., 1977).

The interpretation of EEG findings in children is often difficult because of wide range of patterns that occur normally at any one age. During neonatal period, the clinical and biochemical state of the child at the time of recording may be a crucial factor in the interpretation of results. Even so, the visual analysis of EEG data in newborns enables quite an accurate estimation of gestational age to be made in prematurity (Dreyfus-Brisac, 1970). The EEG of a wakeful, few days old baby is of relatively low voltage, seldom exceeding 50 μV and is composed of irregular and asynchronous theta and delta components.
During deep sleep, discrete bursts of generalized delta activity occur, often associated with faster components of few seconds duration, a pattern named trace-alternant by Dreyfus-Brisac (1964).

Vertex sharp waves and K-complexes begin to appear during sleep at about 6 months of age and sleep spindles may then also appear in fronto-central regions, though their frequency may be slightly lower than 14 Hz, typically seen in adults. During first few months of life there is progressive and relatively rapid increase in voltage of low frequencies in alert state and a tendency for these to become more rhythmic. The dominant frequency also gradually increases with age.

At about 18 months, intermittent low frequency alpha components may begin to appear and there may be quite a marked reduction in amount of occipital activity on eye opening. During 2-6 years the EEG is usually polyrhythmic, different components waxing and waning independently of each other. Even some degree of asynchrony or asymmetry may be normal up to the age of 5 years. Theta rhythms are present mainly at central,
temporal and posterior temporal regions. There is a tendency for all components to become more evident over posterior part of head as the child matures. From 5-15 years of age the alpha frequency increases from 8 to about 10 Hz in average subject. It cannot be emphasized too strongly that this outline of EEG maturation from infancy to adolescence is but an average picture, that the rate of evolution will vary from child to child and that many children will depart from it in any age group.

**EEG IN CEREBRAL PALSY**

Electroencephalographic abnormalities in the patients of cerebral palsy are very common. First attempt in this direction was made by Perlstein et al. (1946) who recorded EEG of 212 consecutive cases of cerebral palsy. They reported that the incidence of EEG abnormality was 82% in seizure group and 45% in non seizure group of cerebral palseid children.

Still later, in 1951, Aird and Cohen performed EEG recording of 167 patients and they observed that 85% of spastics and 60% of athetoid patients had essentially abnormal electroencephalograms. Out of these, the
incidence of focal abnormality was 62% and 32% in spastic and athetoids respectively. They also observed that more severe the clinical involvement, more are the chances of EEG abnormality. However the chances of EEG abnormalities are higher in the patients complicated by epilepsy. They reported that if there has been no history of seizures, the finding of a normal electroencephalogram gives more assurance that clinically evident seizures will not occur, the chances being approximately eight to one in favour of their non-appearance.

One year later, Caugier (1951) recorded EEG in 83 cerebral palsied patients and seconded the view of Aird and Cohen (above) that greater the severity of clinical involvement, more marked is the dysrhythmia in EEG. About 80% of his patients had abnormal EEG tracings.

Skatvedt, N. (1955) observed abnormal electroencephalograms in 59% patients of cerebral palsy. He recorded epileptogenic discharges in 35% patients while only 20.6% had clinical epilepsy.

In a massive study over 1500 patients, Perlstein et al. (1955) noted abnormal EEG, in 90%
patients with epilepsy and in 44% patients without epilepsy, however general character of EEG in both the groups was much the same. They recorded maximum EEG abnormalities in spastics, excluding paraplegics. About 44% of their patients had seizure discharges although they were not having clinical epilepsy. The most frequent seizure discharge in their patients was of petit mal type (45%), followed by spikes (30%). In the asymmetric forms (hemiplegia etc.) they observed either unilateral or predominantly unilateral EEG abnormalities. 66% patients of right hemiplegia had left sided abnormalities while 64% of left hemiplegia had right sided defects. In the asymmetrical recordings the common abnormalities were absence or great reduction in the amount of spindle activity, absence or great reduction of parietal humps in one hemisphere and alteration of normal frequencies from side to side.

Still later in 1963 Gibbs and Gibbs reported various EEG abnormalities in these patients viz: Hypsarhythmia, Bilateral multifocal spikes, unilateral wide spread spikes and localised spikes, 3 per sec. spikes, 14 and 6 per sec. positive spikes, irregular diffuse spikes and slow waves. Out of these, multifocal
bilateral spikes constituted commonest abnormality in 0–1 year age group. Occipital spikes were also very common while temporal spikes and petit mal type discharges were rare. They also recorded asymmetry in EEG in patients with asymmetrical clinical involvement. Slow-waves with or without spikes are common in hemiplegics (52%) thus suggesting localized cortical damage. They observed lateralizing EEG in 47% of hemiplegics and in 97% of their, lateralization was correct. Suppression of voltage production was a reliable localizing sign in EEG according to them.

Same year in 1963, Gibbs and Gibbs performed an interesting study to predict epilepsy in cerebral palsy with help of EEG. While doing follow-up of patients with negative spikes but no epilepsy (below 2 years) they noted that during 2–9 years of age 56% of them had developed seizures. They observed no seizure in follow-up of the patients having normal EEG after the age of 3 years.

Later Bauer, H. (1978) recorded 61.3% abnormal EEGs in cerebral palsy. Highest incidence of EEG abnormality was in atonic diplegia (71%). Among all the diplegias,
EEG abnormality was more frequent if mental retardation was associated (56.4%) than in the patients with normal I.Q. (29.1%). They reported that majority had focal or multifocal defects (58%) in EEG while remaining patients either had generalized changes (22.5%) or unclassifiable and borderline tracings (19.7%).