DISCUSSION
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The study of cerebral palsy has gained a great impetus, due to the development of specific research projects in this field. One of them is the application of electroencephalography in these patients. Electroencephalography, in that it affords data on cerebral physiology, is of considerable significance in the conditions, associated with cerebral pathology and abnormal physiology. This is especially true in those cerebral palsied children whose condition is complicated by convulsive disorders. Considering the problem of epilepsy in cerebral palsy and newer modalities in diagnostic field, it deemed worth while to perform an electro-clinical study on these patients.

The present study was conducted on 50 patients with various presentations of cerebral palsy, who attended the out patient department of Pediatrics, M.L.B. Medical College, Jhansi, from September 1987 to August 1988.

Male dominance in the patients of cerebral palsy is a universally known fact (Portstein et al., 1955, Garg et al., 1965, Sharma et al., 1981).
The reason for this male dominance is difficult to explain. However, Lennox (1953) attributed this predilection of males to their greater liability to congenital cerebral defects and birth injuries. We also experienced male preponderance, as male : female ratio in our study was 16:9.

Birth asphyxia was the commonest predisposing factor in our study, as 44% (22) of our patients were asphyxiated at birth. Out of these, 8% (4) patients were prematures while rest 36% (18) had suffered from birth asphyxia inspite of being full term. The reason could be poor obstetric care, home deliveries, mismanaged delivery and paucity of diagnostic and therapeutic modalities in this field. Udani (1963) and Garg et al., (1965) reported asphyxia in 30% and 33% cases respectively. Anderson (1952) stated that, third to half of all cases of cerebral palsy had evidence of anoxia at birth. Eastman and Delesen (1955) found foetal distress to be four times commoner in cerebral palsy than in general population. Bramm (1986) reported that Ulegeria and status marmoratus of basal ganglia are basic pathological changes in these asphyxiated babies. The lesions are principally in peripheral and dorsal areas of cerebral cortex, involving necrosis of gyri at the depth of sulci and the neuronal mantle of basal-ganglia and brain-stem.
Several other workers had reported prematurity as the principal risk factor (Eastman et al., 1955, Churchill 1974, Prabhakar 1983). The average incidence of prematurity in cerebral palsy, according to them is around 35%, thus prematurity is five times more common in cerebral palsy than general population. About one third (30%) of our patients too were premature though 8% of them had suffered from birth asphyxia also.

Prematurity principally causes subependymal-intraventricular hemorrhage. These lesions are located at the centre of hemisphere in germinal matrix along ventricular region, with sparing of cortical mantle (Bramm, 1966). Deep white matter of periventricular region at the angles of ventricles, may undergo cystic degeneration—Peri-ventricular Leukomalacia.

The reported incidence of cerebral palsy due to hyperbilirubinemia is highly variable. This disparity could be due to the size of sample under study and occurrence of Rh (D) negative individuals in that community. The reported values range from 17.5% (Brandt et al., 1953) to 5.3% (Martin 1960). We found that 2 (4%) cases in our study had severe jaundice during postnatal period one developed athetosis and other one developed spastic diparesis, Garg et al. (1965)
could reveal history of jaundice in 14.5% and Sharma et al. (1981) elaborated history of jaundice in 7% of their patients. None of our patient had any major blood group incompatibility. Raised levels of unconjugated bilirubin in blood may cause its deposition in basal ganglia and other parts of brain, like Cerebellum and subcortical structures. These changes usually occur when serum level of unconjugated bilirubin is more than 18-30 mg%, however prematurity and anemia accentuate bilirubin neurotoxicity (Diamond et al., 1966).

Post infectious pathology is the next common factor in the causation of cerebral palsy. 16% (3) patients in our study material had post-infective etiology. Gauger (1951) observed 21.5%, Gibbs et al. (1963) 15%, Garg et al. (1965) 14.5%, and Sharma et al. (1981) reported 7% such cases. 6% (3) of them in our study had meningitis. Nearly similar figures had been reported by Garg et al. (1965) 6% and Sharma et al. (1981) 7%. Remaining 10% patients had encephalitis. Garg et al. also reported encephalitis in 6.4% cases. Various changes in cerebral vasculature, viz- vasculitis and artherosclerosis cause infarction of cortical structure, supplied by affected vessel. The offending agent may also cause direct destruction (neuronal-necrosis) of
neurons but location of most severe damage varies, some cases having predominantly cortical involvement while in others basal ganglia bear the brunt.

In order to know the prevalence of various clinical types of cerebral palsy we classified our patients according to classification proposed by American Academy of cerebral palsy (Down and Hill 1950). Spastic variety had been the most common type of cerebral palsy as far observed by various workers. Its incidence is reported to vary from 40% (Phelps 1942) to 60% (Harlitz et al. 1955). 76% of our cases had spastic type of defect, thus our figures fairly coincide with others - Asher et al. (1920) 83%, Dunsden (1952) 82%, Furststein (1933), Woods (1937) 70%, Ellingsworth (1939) 72%, Mitchell (1959) 78.3%, Garg et al. (1960) 74.2%, Sharma et al. (1981, 82.7%). But it is noteworthy that Phelps (1942) reported very low incidence of spastic variety as he noticed athetosis to be equally common.

Next in the occurrence was hypotonic variety which was diagnosed in 14% cases. Other workers reported it in 6.5% (Garg et al., 1965) and 5% (Sharma et al. 1981) patients. Relatively higher incidence of hypotonic type in present series could be the younger age of
our patients as some of them may later convert into
spastic or athetoid type (Cruethers and Paine 1999).

The incidence of athetosis reported so far,
varies, from 40% (Phelps, 1942) to 2% (Harlitz and
Redin 1955). We observed athetoid movements in
only 1 (2%) case. But other workers reported higher
incidence—Perlstein et al. (1955) 28%, Carg et al.
(1965) 14.3%, Sharma et al. (1981) 4.1%. This low
incidence in our study could be due to declining
incidence of kernicterus and hyper bile rubinemia,
as during earlier studies suitable management of
neonatal jaundice like phototherapy and Exchange-
transfusion, was not available. Phototherapy itself
was first used by Crumet al. in 1958.

2% (1) patient from present study material
had ataxic type of cerebral palsy. Our figures are
nearly similar to those reported by Sharma et al.
(1981) who observed ataxia in 0.9% (2) of their
patients, though Perlstein et al. (1955) observed
4% such cases. But it will not be wise to compare
these cases due to small sample of our study.

3 patients (6%) had rigidity and tremors both
in all the four limbs. They were categorized as mixed
type of cerebral palsy, Sharma et al. (1981) also reported nearly similar number of such cases (7.3%). But Fehl (1950) and Illingworth (1958) reported lower incidence (2%) of these type of cases.

The reported incidence of convulsive disorders in cerebral palsy ranges from 68% (Yunus, 1944) to 13% (Pirrie et al., 1977). 56% cases in our study suffered from epilepsy. Patients having febrile convulsions were not included. Gauger (1931) noticed seizures in about 40% of his patients while respective values reported by other workers are - Perlstein et al. (1933) 47% and Garg et al. (1969) 23.4%. Our values are slightly higher and the possible explanation for which could be the younger age of our patients, as 44% of our patients were below 2 years of age. The younger patients are more commonly victimized by seizures (Perlstein et al., 1933).

If epileptics are further dichotomized, then we see that generalized tonic-clonic was the commonest seizure type, observed in 43% cases. Our this observation is further ascend by Perlstein et al. (1933) and Garg et al. (1969) who observed that type of seizure in 53% and 60% epileptics respectively. 10 (23%) out
of total 12 patients suffering from this variety of seizures, were spastic and still half of them (41.5%) had spastic diplegia. Fairly identical figures had been reported by Garg et al. (1963) where 73% patients of grand mal type had spasticity.

Next in the occurrence was myoclonic seizure, as 18% of our patients exhibited this type only. Perlstein et al. (1955) observed myoclonus in only 7% of their material. The reason of this disparity could be the larger proportion of hypotonic patients in our study as they had highest incidence of such seizures (Bauer 1978).

We observed generalized tonic seizures in 10.7% patients. All the patients of generalized tonic type had spasticity. Perlstein et al. (1955) also had 14% patients of generalized epilepsy excepting grand mal type, in their study material and we included fairly similar number (14.2%) of patients of identical type in the form of generalized tonic (10.7%) and generalized clonic (3.7%) variety.

Incidence of Focal epilepsy was 10.7% in our patients. Two third patients of this variety had spasticity while remaining one third had hypotonia.
Perlstein et al. (1955) included 24% and Carg et al. (1965) included only 2.5% cases of focal epilepsy.

7.4% patients in this series had psychomotor epilepsy. They all presented with behavioural abnormality episodic onset. No one other than Perlstein et al. (1955), observed psychomotor epilepsy who reported 0.4% such cases. This disparity can be due to associated behavioural problems itself in cerebral palsy which may easily be missed or discounted. Half of these patients had spastic diplegia and rest half had mixed type of cerebral palsy.

Rest 7.4% of our patients had mixed type seizures (more than one variety). We did not observed any patient of Petit mal type. 1.6% of Perlstein et al. (1955) series and 4.3% of Carg et al. (1965) series had Petit mal seizures.

It is important to compare these figures with general population. Kaushik et al. (1980) stated that out of all the types of epilepsy in general population, Grand-mal was the most common type, observed in 66.3% cases, followed by focal seizures (25.3%) Psychomotor (2%), petit mal (2%) and mixed epilepsy (4%). The lesser occurrence of focal epilepsy in cerebral palsy could be
due to generalised, rather than localized, brain
damage in these patients.

It can further be added that majority (75%) of
epileptics belonged to spastic type of cerebral
palsy or in other words 55% patients of the spastic
group gave history of convulsions. Aird and Cohen
(1950) included 65% and Perlstein et al. (1955)
reported 50% epileptics. But Carg et al. (1965)
reported that only 21.7% of all their spastic
patients ever had seizures.

However incidence of seizures was highest
in hypotonic patients i.e. 85.7%. Bauer (1978) also
reported maximum incidence in atonic patients but
his figures (69%) are lower than ours. It is important
to mention here that all the patients of hypotonic
diparesis had epilepsy in our series i.e. an incidence
of 100%. None of our patients belonging to athetoid
or ataxic category was epileptic.

Microcephaly is one of the commonest associated
anomaly in the patients of cerebral palsy (Nenkes,
1980; Brown and Fulford, 1984). 31 (62%) of our patients
had microcephaly. The incidence of microcephaly was
highest in hypotonic type as all of them (100%) had
microcephaly and next followed by spasticity where 55% had associated microcephaly.

Delayed milestones is the commonest presentation of cerebral palsy. Psychologists and pediatricians differ in their approach to developmental assessment. While psychologists tend to base their score on the ability to pass a certain test or make score, the pediatricians want to know, not just whether the child can achieve a particular skill but the maturity of his response and therefore how long he has been able to do it. The clinician's basic aim is to determine how far the child has developed in relation to normal. One of the methods of expression of development, for the purpose of comparison, is to assess child's Developmental Quotient (D.Q.). Thus we have calculated developmental quotient of each of fifty patients according to method described by Prabhakar and Kumar (1963).

Average developmental quotient of our study was 34.9%. Maximum developmental retardation was observed in Athetosis (D.Q. = 11%) and spastic tripareisis (D.Q. 19%). Though much emphasis can't
be imparted to these figures as one patient only belonged to these categories.

Next in the sequence of developmental retardation are hypotonic, (D.Q. = 25.14%) and spastic quadriparetic (D.Q. = 26.6%) patients. While least developmental retardation was seen in ataxic (D.Q. = 69%), Spastic (left) hemiparetic (D.Q. = 59.7%) and Spastic (left crural) monoparetic (D.Q. = 37.0%) patients. Spastic diparetic which comprised the substantial amount of present study material, had developmental quotient of 35.3%, and overall D.Q. in all the spastics was 36.2%.

Electroencephalographic abnormalities are quiet frequent in cerebral palsied children, but these abnormalities become more apparent and frequent if epilepsy is also associated. All the patients were sedated before hand as they were not cooperative. However no appreciable difference is noted in sleep and awake recordings (Gauger, 1951).

Reported values of abnormal EEG tracings in cerebral palsy varies from 65% (Aird et al. 1950, Perlstein et al. 1955) to 80% (Gauger 1951). However
60% of our patients had essentially abnormal EEG records. Bauer (1973) found fairly similar number of patients (61.3%) having abnormal electroencephalograms.

If these patients with abnormal electroencephalograms are further subclassified then it has been observed that maximum abnormality is seen in spastic patients. Aird and Cohen (1950) noted 55% EEG abnormality in his study material and Perlstein et al. (1955) noted it in about 72% patients. Though still lower figures (61%) had been observed by Bauer (1973). 55% of our spastics had abnormal electroencephalograms.

In contrast, we recorded maximum EEG abnormality in Hypotonic patients (70%) but our figures fairly coincide with that of Bauer (1973) corresponding figure is 71%. Moreover 66.6% of mixed type of cerebral palsy in our study had abnormal EEG. But it is important to note here that mixed type of cerebral palsy was observed in only 6% (3) patients by us. So it will be difficult to comment on them. Same holds true for ataxic and athetoid patients. None of the athetoid patients (2%) had any electroencephalographic abnormality.
This seems reasonable, because athetosis is caused by extrapyramidal lesions, and injuries to extrapyramidal structures (being deep seated) do not usually produce abnormality in electroencephalograms.

Epilepsy, being an episodic electrical abnormality of brain, quite frequently gives rise to some electroencephalographic abnormality, either in the form of inter-ictal discharges or as a manifestation of ictal-brain damage. Out of all the 60% patients having abnormal electroencephalograms, only two third had epilepsy while rest had EEG abnormality without ever experiencing seizures. Gibbs et al., (1963) however observed that normal electroencephalograms are far more common among non-epileptics than epileptics. In our study incidence of abnormal EEG in epileptics was 77% while among non epileptics only 30% had abnormal electroencephalograms. But the general character of electroencephalographic findings in epileptic and non epileptic groups in much the same, except for much higher incidence of normal records in non-seizure group while much frequent occurrence of spike foci in seizure group.
High degree of concordance exists between laterality of clinical involvement and the laterality of electroencephalographic abnormality in cerebral palsy. In symmetrical forms of cerebral palsy, the electroencephalographic abnormalities when present are usually bilaterally symmetrical but in asymmetric forms, EEG findings are either unilateral or predominantly on contralateral side (Ferliein et al. 1955).

About one quarter (26.6%) of our patients had focal changes in EEG and in 62.5% of these cases, the lateralization was correct i.e. lateralized to the side opposite of motor deficit. Maximum lateralization was seen in hemiparetic patients as all (100%) the patients with left hemiparesis revealed right hemispherical damage while two third patients of right hemiparesis had only left sided and rest one third had predominantly left sided abnormalities. None of our patients with bilateral affection clinically, had asymmetrical EEG.

Reduced voltage production and asymmetry of sleep spindles were the most significant abnormalities in lateralising EEG, Gibbs et al. (1963) noted lateralization in 43% cases and out of that, in 97% the lateralization was correct. But this should not mean that brain damage is exclusively unilateral in hemiplegic patients (Gibbs et al. 1963).
In most of the patients there was evidence of intermittent generalised slowing suggestive of diffuse brain damage while next common abnormality was spikes or spike-and-slow-wave complexes which however were common in epileptics.

In the last we have tried to compare EEG abnormality and developmental quotient. In our series average developmental quotient was 34.9% and majority of our patients (76%) had developmental quotient below 90%. The developmental retardation was more severe in the patients with abnormal EEG than in the patients with normal EEG. This was statistically significant as 'p' value is ≤ 0.05. This suggests that more is the developmental-retardation, more are the chances of abnormality in electroencephalograms. We could not find any study correlating developmental quotient and EEG findings cerebral palsy but studies conducted, where, intelligence-quotient has been correlated with EEG finding in these patients, report that more severe the mental retardation more are the chances of EEG abnormality (Gauger 1957, Gibbs et al. 1963). We also observed significantly more
chances of developmental retardation in epileptics, as of all the epileptics more than 85% had D.W. below 50%. This could be due to further brain damage in epileptics (Lennox 1942, Waterlain 1970).