“ Neurophysiologic findings and clinico-radiologic correlates in children with bilateral spastic cerebral palsy”

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BACKGROUND

Cerebral palsy (CP) represents the group of permanent non-progressive motor disorders. Somatosensory system which was not suggested to be involved in CP children has interestingly been reported to be associated with motor disorders in cerebral palsy [1-3]. Somatosensory evoked potential (SSEPs) as non-invasive method of investigating central as well as peripheral nervous system can be particularly useful in infants and young children in whom the clinical sensory examination is often difficult and unreliable.

Disruption of sensory tracts, if present might indicate the presence of sensory impairment and also sensory-motor integration deficits in children with cerebral palsy. The radiological investigation can provide the information about the nature and timing of the brain lesion in cerebral palsy [4]. However, the region of present research despite with higher rates of disability in the country and considerable incidence rates of CP, lacks substantial data investigating the same [5-7]. Most of the data emerge from the developed countries [8, 9].

The present study findings can provide insights into the extent of the brain damage and possible pathophysiologic mechanism contributing to the same, which in turn may help in planning current physical therapy-based treatment approaches (sensory integration therapy/occupational therapy).

OBJECTIVES

The present study aimed at obtaining SSEP, visual evoked potential (VEP), brainstem auditory evoked potentials (BAEP) and electroencephalography (EEG) in children with bilateral spastic cerebral palsy and to compare the records with clinical and radiological variables.

MATERIALS AND METHODS

60 participants (30 children with spastic CP and 30 controls, age-range: 6 months-10 years) were studied for a period of one year. Sample size calculation was done based on the differences in the mean (effect size) from the previous similar study, power of 80% and 1.96 as the level of statistical significance [10]. The inclusion criteria for the study group were the patients with bilateral spastic cerebral palsy in the age group of (6 months-10 years). The exclusion criteria for the study group were the subjects with otological diseases and peripheral nerve injury. Patients underwent neurophysiological testing and brain magnetic resonance imaging (MRI).

SSEP, VEP, BAEP and EEG were recorded in 30 children with bilateral spastic cerebral palsy and were compared with those with normal age, sex and height-matched controls. Clinical variables were analysed. Presence of associated comorbidities and risk factors were studied.

SSEP, VEP and BAEP were performed on Neuro-MEPω EMG and EP digital neurophysiological system software in Neurophysiology laboratory, Department of Physiology, AIIMS, Gorakhpur *(figure 1)*. A written informed consent from the parents was obtained from the children before the test. Methodology for the test was as recommended by guidelines by American Clinical Neurophysiology society [11-13]. Children were sedated with oral chloral hydrate (25-75 mg/kg). Preparation of scalp skin was done prior to the electrode application. Standard disc surface electrodes were placed according to the International 10/20 system of electrode placement.

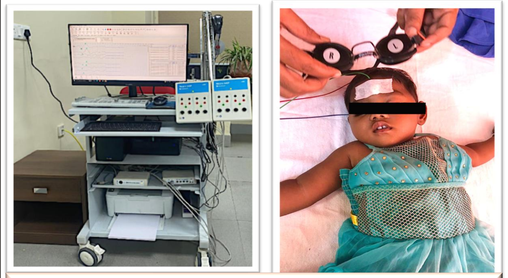

Figure 1: Neuro-MEP 8 machine (8-channel NCS, EMG and multi-modality EP system) (Neurophysiology Laboratory, Department of Physiology, AIIMS Gorakhpur) (left), Flash VEP test preparation in a normal female child (right).

SSEP

Median and tibial somatosensory evoked potentials were recorded. Recordings in response to unilateral median nerve stimulation was obtained from contralateral cortex CP3 referred to Fz. All the components (N9, 13, 18 and 20) were measured via four channel recording. The cortical component N20 was evaluated. For tibial SSEP, P37 (Cpz referred to Fpz) was evaluated.

VEP

Flash VEP was recorded with active electrode at Oz, reference electrode at Cz and ground electrode at Fpz. The method of presentation of the stimuli was by means of goggles (monocular stimulation).

BAEP

BAEP was recorded with active electrode placed at mastoid (M1 or M2), reference electrode at Cz and ground electrode at Fpz. with Monaural stimulation was done with intensities ranging from 110 to 20 decibel SPL (sound pressure level). Stimulation was delivered by head-phones. BAEP records for absolute and interpeak latencies and amplitudes were evaluated.

EEG

EEG was conducted with bipolar and referential montages. The responsiveness of the background activity was assessed. EEG response to activation procedures (photic stimulation and hyperventilation) recorded.

***MRI*:** MRI was recorded in axial T1, T2, FLAIR, sagittal T1,coronal FLAIR and diffusion-weighted imaging sequences.

Statistical analysis

Chi^2 test and t-test were used for the statistical correlation with the qualitative and quantitative variables respectively. Independent sample t-test was employed for comparing group means. $p<0.05$ was considered as statistically significant.

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RESULTS

Tibial SSEPs and median SSEPs revealed abnormal cortical response in 76.6 % (23 of 30) and 66.66 % (20 of 30) respectively. Abnormal BAEP recordings were observed in 4 (13.33 %) and abnormal VEP recordings in 15 (50 %) patients. Abnormal EEG was found in 12 patients (40 %). Abnormal MRI (93.33 %) had periventricular leukomalacia as major finding *(figure 2)*. Abnormal cortical tibial SSEP response were statistically correlated with abnormal EEG (p= 0.009) and perinatal asphyxia (p= 0.012) *(table 2)*. Abnormal cortical median SSEPs were statistically correlated with abnormal VEP and perinatal asphyxia (p<0.05) (Chi-square test) *(table 3; figure 3 and 4)*.

Table1: Demographic and clinical variables of the study participants (total n=30)

S. No.	Variable		n	%
1	Age	6 months-5 years	17	56.66
		6-10 years	13	43.33
2	Gender	Male	16	53.33
		Female	14	46.66
3	Spastic CP type	Quadriplegic	20	66.66
		Diplegic	10	33.33
4	Functional level of motor skills (GMFCS)	Level I	3	10
		Level II	4	13.33
		Level III	4	13.33
		Level IV	6	20
		Level V	13	43.33
5	Risk factors	Prematurity	15	50
		Perinatal asphyxia	17	56.66
		Perinatal infection	4	13.33
6	Comorbidities	Epilepsy	15	50
		Abnormal IQ	25	83.33
		Visual dysfunction	15	50
		Hearing disorders	4	13.33
		Orthopaedic disorders	12	40

n: number; %: percentage; CP: cerebral palsy; GMFCS: Gross motor function classification system; IQ: Intelligence Quotient.

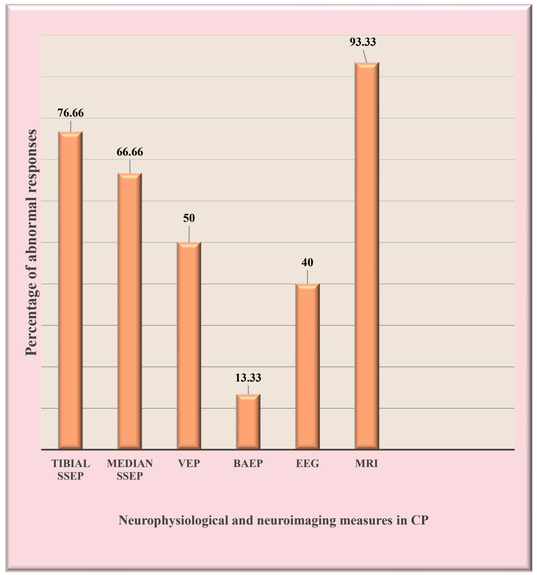

Figure 2: Abnormal neurophysiological and neuroimaging findings in patients with CP

SSEP: somatosensory evoked potentials; VEP: visual evoked potentials; BAEP: brainstem auditory evoked potentials; MRI: magnetic resonance imaging; CP: cerebral palsy

Table 2: Statistical association of abnormal *tibial SSEPs* with other neurophysiological, radiological and clinical variables

S. No	Variable	Chi-square value	p-value
1	Abnormal EEG	6.81	0.009**
2	Abnormal VEP	3.35	0.067
3	Abnormal BAEP	1.77	0.18
4	Abnormal MRI	0.02	0.87
5	Perinatal asphyxia	6.43	0.012*
6	Prematurity	3.47	0.06
7	Perinatal infection	1.33	0.25

*p<0.05, **p<0.01
SSEP: somatosensory evoked potentials; EEG: electroencephalography; VEP: visual evoked potential; BAEP: brainstem-auditory evoked potentials; MRI: magnetic resonance imaging

Table 3: Statistical association of abnormal *median SSEPs* with other neurophysiological, radiological and clinical variables

S. No	Variable	Chi-square value	p-value
1	Abnormal EEG	3.47	0.06
2	Abnormal VEP	6.56	0.01*
3	Abnormal BAEP	0.97	0.32
4	Abnormal MRI	0.02	0.87
5	Perinatal asphyxia	4.54	0.033*
6	Prematurity	1.2	0.27
7	Perinatal infection	0.69	0.40

*p<0.05
SSEP: somatosensory evoked potentials; EEG: electroencephalography; VEP: visual evoked potential; BAEP: brainstem-auditory evoked potentials; MRI: magnetic resonance imaging

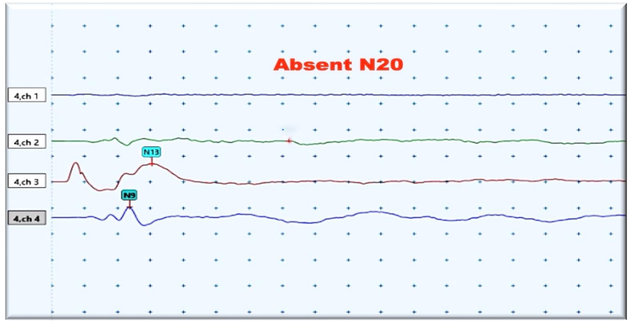

Figure 3: Representative median SSEP record of a 2-year-old male with spastic CP depicting absent cortical response.

sensitivity: 5µv, sweep speed: 2.5 ms.
µv: microvolt; ms: milliseconds; SSEP; somatosensory evoked potentials

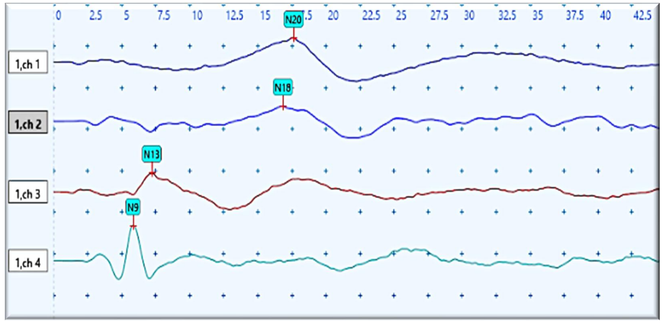

Figure 4: Representative median SSEP record of a 2-year-old normal male child depicting normal cortical response.

sensitivity: 5µv, sweep speed: 2.5 ms.
µv: microvolt; ms: milliseconds; SSEP; somatosensory evoked potentials

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DISCUSSION

High proportion of abnormal SSEP documented in the study is in concordance with the literature [8-10,15]. Somatosensory tracts in close alignment to the susceptible brain areas may explain the findings [14]. Also, influence of spasticity on the cortical somatosensory evoked potential responses has been implicated [15].

Correlation of abnormal VEP and SSEP has also been explained on the basis of involvement of parietal lobe and internal capsule fibers in CP [10]. Correlation of abnormal cortical SSEP and EEG strengthens the evidence of the damage in the cortex. Both VEP and EEG have previously been found to be valuable tools to assess the neurological outcome in high-risk neonates. Hence, it seems plausible that evaluating cortical SSEP in children with abnormal EEG and abnormal VEP may be valuable in assessing sensory impairment and also in assessing the neurological outcome in CP in follow-up studies.

CONCLUSIONS

The study provides objective evidences for impairment of central perception of the sensory stimulus in children with spastic CP. Evidences of sensory cortical involvement in cerebral palsy can also help in designing a better treating plan based on sensory integration therapy/occupational therapy.

Follow-up studies evaluating cortical SSEPs in children with spastic CP may further assess the potential utility of cortical SSEP as a prognostic tool in children with CP.

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