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AUDITORY EVOKED POTENTIAL RESPONSES IN CHRONIC MALNOURISHED CHILDREN

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Abstract : Auditory evoked potential responses were recorded in 20 chronic malnourished children in age group 3–6 years and in 20 healthy age and sex matched controls using an 5200 Neuropack plus (Nihon Koden, Japan) evoked potential recorder. The absolute peak latencies, inter peak latencies and amplitude of waves I-V of brainstem auditory evoked potentials (BAEPs) were analyzed. The mid latency responses (MLRs) were also studied in these children. Malnutrition was characterized by stunting, which indicated chronicity of nutritional deprivation. The children with chronic Protein energy malnutrition (PEM) had prolonged peak latencies of waves I, II, III and IV. The interpeak latencies I-III and III-V were also prolonged. The amplitude of wave I and V did not show any significant difference as compared to controls. The middle latency responses were not significantly different from the controls. Thus malnutrition affects the peripheral developmental process of auditory pathways only in the brainstem and the central thalamocortical projections of these pathways are spared.

Key words : evoked potential children malnutrition auditory brain response middle latency responses

INTRODUCTION

Protein energy malnutrition is a pathological condition arising from coincidental lack of varying proportion of protein and calories occurring most frequently in infants and children. The effect of malnutrition on brain development depends on duration, type and severity of malnutrition. Previous study has shown that malnutrition affects the developmental process in rostral (pontomesencephalic) in infancy and in the caudal (medullopontine) regions during childhood (2–5 years) (1). Neuropharmological studies have shown changes in brain neural receptor function resulting from early episode of malnutrition (2). The age and range of these long term effects of malnutrition may be much greater and the minimal amount of malnutrition necessary to produce these long term alterations are unknown (3). Malnutrition imposed during early life not only reduces the growth of the brain but also leaves it permanently smaller in size, the phenomenon first observed in rats (4). A few

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earlier studies have shown that conduction in the peripheral nerves is affected in protein energy malnutrition (5, 6). It appears that long duration of continuous PEM slows or arrest the process of myelination thereby preventing the increase in caliber of myelinated nerve fibers. Perinatal undernutrition in rats produce severe retardation of fiber growth in caliber in sciatic nerve, spinal roots and optic nerve. Since the early waves form the peripheral part of auditory pathway, and nerve conduction velocity is related more to diameter than internodal length of nerve fibers (7). The conventional view, supported by pioneering work using DNA as an indicator of brain growth was that brain is most vulnerable to malnutrition during the critical period of brain growth and development. Several groups have studied the EEG which generally demonstrates nonspecific abnormality of diffuse slowing. The Computed Tomography (CT) shows cerebral atrophy, which resolves upon nutritional rehabilitation. Cerebral atrophy is also demonstrated by Magnetic Resonance Imaging (MRI). This study was therefore designed to provide an electrophysiological evidence of developmental abnormalities imposed by prolonged malnutrition specifically on auditory pathways in brain. This study also included MLR, a parameter of evoked potentials which was not incorporated in previous studies (2), to assess the functional integrity of Thalamocortical projection which represent the central part of auditory pathway, during prolonged malnutrition.

MATERIALS AND METHODS

The study was conducted in the clinical neurophysiology laboratory of department of Physiology, UCMS and GTB hospital, Delhi. India. 20 normal subjects and 20 chronic malnourished children in age group 3–6 years were recruited on the basis of anthropometrical parameters including height, weight, and mid arm circumference. Estimation of hemoglobin and serum albumin was done using standard kits.

Selection criteria : A measure of ‘height for age’ and ‘weight for age’ were used as selection criteria. A drop in Ht to age ratio points to a chronic malnourished condition-stunting, a low Wt for age with normal height indicates an acute condition-wasting or weight loss. A low height for age can be taken as one that is 1 standard deviation (SD) below the median height for age of the reference population given by National Center For Health Statistics (NCHS) (8), conversely a high height for age is more than 1 SD above the median Ht for age of the reference population. The same principle applies to the other indicator, Wt for age. Those with in 1 SD were taken as normal healthy controls.

Exclusion criteria : A thorough history and physical examination was done to rule out genetic, visceral and endocrinal causes of short stature. Children with any ear pathology were excluded. An informed consent was taken from all the subjects. A baseline recording BAEPs and MLRs was taken in all children. A comparison was done between control and study group using Tukey test.

Measurement Protocols : BAEPs – Subjects were lying comfortably in a reclining bed with their eyes closed and completely relaxed. Apprehensive children were given promethzine in dose Of 0.5 mg/kg body weight orally. The subjects were informed about the procedure. BAEPs were recorded using standard techniques (9.10). Click stimuli (average count 2048) of intensity 70 dB above the normal hearing

threshold, at the rate of 10/s and 0.1 ms duration were presented to each ear independently. The other ear was masked by pure white noise –40dBHL. These clicks were generated by passings 0.1 ms square pulses through the shielded headphones with alternating polarity. The active (+ve) electrode was placed over vertex (Cz), reference electrode was placed at the ear lobules separately (A1, A2) and the ground electrodes on the forehead. The skin to electrode impedance was kept below 5 Kohm. The signal picked up by these electrodes was filtered, amplified, averaged and displayed on the screen of Neuropack 5200 plus potential recorder (Nihon Kohden, Japan). Absolute peak latencies of waves I and V and interpeak latencies I-III, III-V, l-V were recorded for each ear separately.

MLRs : Same procedure was repeated with the same electrodes and with the same latency range between 10–50 ms. Alternating rarefaction and condensation clicks were generated by an acoustic stimulator with the duration of 0.1 ms and interstimulus interval of 70 ms delivered through the headphones monaurally/ binaurally at the intensity 60dBHL (8). With the stimulus rate 0.5 Hz, total of 256 stimuli were given and evoked responses of these were recorded. Latency of each component wave MLRs i.e. N0, P0, Na, Pa, Nb, Pb was calculated. Comparison between the two groups was done using a Tukey test at 5% level of significance.

RESULTS

The mean and the standard deviations of absolute peak latency and interpeak latency in milliseconds of BAEPs are shown in Table I. A significant prolongation in the absolute peak latencies of waves I to IV was observed in the study group as compared to

TABLE I : Showing values of various BAEPs in control and malnourished groups.

Group	*APLs*					*IPLs*			*Amp.*	
	I	*II*	*III*	*IV*	*V*	*I-III*	*III-IV*	*I-V*	*I*	*V*
Control	1.56±0.07	2.54±0.12	3.75±0.10	4.46±0.24	5.61±0.21	2.25±0.21	1.04±0.19	4.05±0.16	0.36±0.09	0.30±0.11
Study	1.68±0.12*	2.67±0.13**	3.78±0.16**	4.55±0.34**	5.64±0.07	2.01±0.17*	1.94±0.14*	3.93±0.10*	0.35±0.10	0.30±0.09

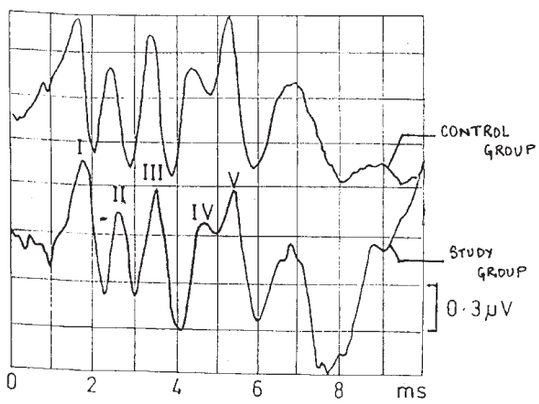
*P<0.05; **P<0.01

TABLE II : Value of MLRs in control and malnourished groups.

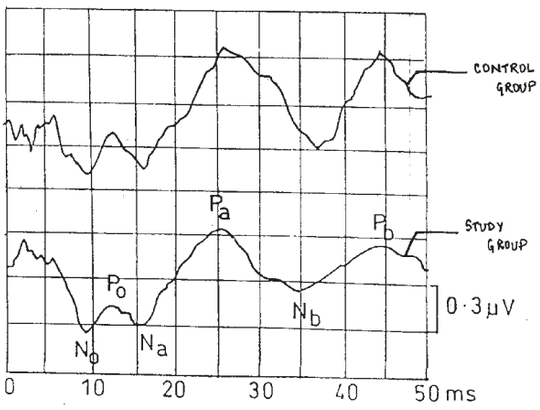
Group	*No*	*Po*	*Na*	*Pa*	*Nb*	*Pb*
Control	11.69±0.83	16.85±2.38	22.33±4.95	31.23±6.19	37.25±5.99	41.94±4.79
Study	11.62±0.72	16.48±2.26	24.53±2.14	31.76±3.10	37.30±2.94	44.11±1.99

the control group. No significant difference was found in the amplitudes of waves V and I in the study group as compared to the control group. No significant difference was found in the latency of the components No to Pb MLR in the study group as compared to the controls. The values of the components of MLR are given in Table II.

REPRESENTATIVE TRACING OF ABR



REPRESENTATIVE TRACINGS OF MLR



DISCUSSION

The present study has revealed changes in absolute and interpeak latencies of Brainstem auditory evoked potentials in Protein energy malnutrition (PEM). A few pervious studies have also reported a significant reduction of nerve conduction velocity in children with PEM(1). The neural conduction in auditory pathways in the brainstem in the PEM children is significantly different from those without deficiency, indicating that the deficiency has contributed to altered neural conduction. The absolute latencies of waves l-V in the present study for the control group and the study group are shown in table 1, which are similar to the latencies reported in an earlier study conducted by Tandon et al (2), with malnourished children showing absolute latencies of waves I-V as 1.72 ± 9.18 ms, 2.63 ± 0.15 ms, 3.84 ± 0.17 ms, 5.00 ± 0.21 ms, 5.91 ± 0.29 ms. In this study the children who are malnourished and show a height deficit representing chronicity of PEM exhibit a significant increase in absolute latencies of the early waves except wave V of BAEPs. The earlier studies on auditory evoked potentials by Tandon et al (1989) on 96 malnourished children under six years of age showed abnormal interpeak latencies in 32 children (33.3%) and 8 children had abnormal morphology characterized by absent wave patterns. It was observed that the malnutrition affected the development process in the rostral (pontomesencephalic)

pathway in infancy and the caudal (medullopontine) regions during childhood. But in previous study no significant difference in absolute latencies in three grades of malnutrition was observed categorized on the basis weight for age, suggesting that in the present study the increased absolute latencies are related to the chronicity of PEM. Thus effect of malnutrition on BAEPs depends on the duration, type and severity of malnutrition. It appears that a long duration of PEM slows or arrest the process of myelination in the peripheral nerves thereby preventing the increase in caliber of myelinated fibers (9). Since the early waves form the peripheral part of the auditory pathway, and the nerve conduction velocity is related to diameter rather than internodal length of the nerve fibers, this study suggest that PEM from an early stage possibly" beginning perinatally leads to persistence of small diameter myelinated nerve fibers and the presence of segmental demyelination and improper remyelination. Thus PEM seems to be responsible for reduced conduction velocity in chronic malnutrition. The various interpeak latencies reflect developmental changes in the time sequence of' myelination and myclin composition along the auditory pathway. Neurohistological evidence of improper myelination and small caliber nerve fibers as a cause of abnormal BAEPs in PEM still needs to be demonstrated since BAEPs have limitations in their interpretation as their abnormality can not separate inadequacy of axonal, dendritic or synaptic events. Thus the cellular or intercellular events occurring during chronic PEM at the sites of generation of these waves in the brainstem cannot be determined. The middle latency components of auditory evoked responses reflect thalamocortical projections having generators in the primary cortex of the temporal lobe and seem to be resistant to malnutrition effects. Whether this is permanent damage or is reversible with nutritional rehabilitation remains to be investigated.

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