



is essential that this symptom be scientifically analyzed to discover the underlying cause if any. Since sensory impairment can be one of the contributory factors of their performance, a thorough sensory examination is mandatory in these children. Visual evoked potential (VEP) is one of the non invasive tool which can assess the functional integrity of the visual pathway from the retina to the visual cortex. The VEP is primarily a reflection of central 3 degree to 6 degree of the visual field projecting onto surface of the occipital lobe. As VEP is mostly of foveal origin reflecting cone activity, any abnormality of fovea, cones or its projection to occipital lobe could be assessed by it (8). To the best of our knowledge the complete processing of visual pathway have not been studied earlier in slow learner group with unassociated specific learning disability or psychological problems. Hence the present study aims to investigate the possibility of deficits in central neural processing of visual pathway in slow learners.

#### MATERIALS AND METHODS

The study was conducted in the Electrophysiology Laboratory, Department of Physiology, University College of Medical Sciences, Delhi. The subjects were selected from a school for special children of Delhi. These subjects were referred for classroom behavioral or academic problems and they were tested by a psychological evaluation team. All those students not in the category of specific psychological disorders like learning disability (LD), attention deficit hyperactivity disorder (ADHD), mental retardation etc were selected for the study. Out of 31 children diagnosed as slow learners 5 could not participate in the study due to lack of consent from parents or prolonged

absenteeism from school, one had visual field defect and 8 had ADHD, dyslexia or other psychological problems and were excluded from the study. Hence 17 slow learners, 10 males and 7 females with mean age of  $10.59 \pm 1.87$  yrs participated in the present study. Fifteen age and sex matched controls (9males and 6 females) with mean age of  $10.87 \pm 2.94$  yrs who had good school performance, were recruited from an elementary school in the vicinity of our institution. The clearance from the Ethical Committee of the institution was taken and an informed written consent was taken from the parents, after the recording procedure was explained to them. The recording was done in the presence of either one of the parents or teacher.

A thorough optometric examination was conducted to rule out any non correctable visual deficits. A detailed history of clinical, academic, physical, psychological and neurological examinations was noted. The parents were interviewed to get specific information about their child's birth history, developmental history and other relevant behavioral problems. The clinical psychologist conducted the standard IQ test MISIC (Malin's Intelligence Scale for Indian Children) an Indian adaptation of Wechsler intelligence scale for children (WISIC). The counselor interviewed the parent (s) to get specific information about any behavior problems in the child.

#### Recording of visual evoked potential

The recording was done in a sound proof room and the subjects were given a trial session a day before the recording to familiarize them with the recording procedure. The PRVEP (Pattern reversal

visual evoked potentials) was recorded on Nihon Kohden Neuropack  $\mu$  MEB -9100, Japan using silver-silver chloride disk electrode placed at standard scalp locations according to the 10-20 International System. Subjects were told about the procedure before the test. The electrodes were placed at O<sub>1</sub>, O<sub>2</sub> (Active electrodes), A1 and A2 (reference electrode), Fpz (ground electrode) after cleaning the scalp or skin site with alcohol followed by Skinpure™ Skin preparation gel and EEG paste Elefix™. The skin electrode contact impedance was kept at less than 5 K $\Omega$ . The subject was seated comfortably in a quite darkened room, 1 meter away from the screen of a television and instructed to fixate on a small dot at its centre with one eye while the other eye was covered with a patch. The screen size measured 275 by 350 mm. The black and white checks (16x16 mm size) subtending an angle of 32 minutes of an arc were generated on the monitor by an electronic pattern generator. Luminance of dark checks was 6.31 ft-L and of the light checks was 31.6 ft-L giving contrast between black and white checks of 67%. The checks were made to reverse at a rate of 1 Hz and 200 responses were recorded and averaged by the computer of the evoked potential recorder with low and high frequency filters 1-200 Hz. Two trials of 200 responses were averaged for each eye. The absolute latencies of positive and negative waves were recorded.

#### Statistical analysis

The data obtained was analyzed using SPSS software (Version 13.0). The average of left and right eye was taken and analyzed. Unpaired 't' test was used to compare between the controls and the subjects. Results were expressed as mean $\pm$ SD.

## RESULTS

The mean IQ of slow learners was 81.26 $\pm$ 4.23 which was significantly lower as compared to that of controls having mean IQ of 99.73 $\pm$ 8.63. The age, gender and anthropometric measurements are given in Table I. There was no significant difference in these parameters between the two groups. It was observed that 76.5% of the subjects have delayed milestone/development history, 29.4% did not cry immediately after birth, 29.4% had history of convulsions, 23.5% had premature birth 17.6% had delayed speech, 17.6% of the subjects were kept in incubator after birth, 11.8% had history of post term delivery, 5.9% had history of nocturnal bedwetting, 5.9% had history of caesarean delivery, 5.9% had history of forceps delivery (Table II).

TABLE I: Age, gender and anthropometric parameters of controls and slow learners.

	Control (n=15)	Slow learners (n=17)
Age	10.87 $\pm$ 2.94	10.59 $\pm$ 1.87
Gender:		
Males	9	10
Females	6	7
Height (cms)	148 $\pm$ 9.86	145 $\pm$ 12.30
Weight (kg)	45 $\pm$ 10.23	42 $\pm$ 12.56
BMI (kg/m <sup>2</sup> )	20.5 $\pm$ 2.74	19.86 $\pm$ 2.98

TABLE II: Clinical History and findings of Slow Learners (n=17).

Clinical history	Number of Subjects (%)
Delayed developmental history	13 (76.5%)
Hospital delivery	7 (41.2%)
Home delivery	6 (35.3%)
History of convulsions	5 (29.4%)
Did not cry after birth	5 (29.4%)
Born prematurely	4 (23.5%)
Delayed speech	3 (17.6%)
Kept in incubator after birth	3 (17.6%)
Post term delivery	2 (11.8%)
Nocturnal enuresis	1 (5.9%)
Caesarean delivery	1 (5.9%)
Forceps delivery	1 (5.9%)
Delayed walking	1 (5.9%)
Hydrocephalus	1 (5.9%)

comparable to the values obtained earlier in our laboratory (10).

Theories relating visual dysfunction and visual processing problems to poor school performance have been discussed in the literature earlier (11). Four general classes of visual abnormality have been proposed to account for learning problems.

1. Blurred or double vision associated with uncorrected refractive error, abnormal binocular vision or abnormal accommodation.
2. Poor eye movement or fixation control during reading.
3. Abnormal central neural processing, recent theories of which suggest deficiencies in magnocellular pathway (12–14).
4. Problems in mechanisms of perceptual or visual information processing.

Prior to recording the VEP our subjects had undergone a complete optometric examination hence the first two visual defects were absent in them. Deficiencies in central neural processing of visual information can be one of the contributing factors in the performance of our subjects which has never been reported earlier in slow learner group. Studies with transient stimuli have found reduced sensitivity attributable to magnocellular deficits in individuals with learning disability (12–14). Differences have been reported between individuals with and without dyslexia in contrast sensitivity (15), flicker thresholds (16), VEP (12, 17) and anatomy in magnocellular layers of cadaver brains (16). These results have given support to the theory that weak saccadic suppression leading to problematic visual persistence during reading might be a causal element in

learning or reading disorders.

It was observed that the response of magnocellular visual pathway is slowed in reading disabled children, who do not have a general slowing visual response (18). An abnormality of magnocellular pathway was confirmed anatomically by the demonstration that cells in magnocellular layers of the dorsal lateral geniculate nucleus of the brains of reading disabled persons were smaller than those in normal readers (19).

Our findings of a significantly prolonged latency of N75 component of VEP is consistent with delay in visual performance at very early stage of processing, such as that accomplished by M cells. VEP's to pattern reversal stimulation as used in the present study are evoked predominantly from the central 10 degree of the visual field and therefore reflect the function of the macular area and related pathways (4, 20, 21). The visual system processes information along multiple parallel channels (22). The separation of visual information starts at the neuronal circuitry of retina. The magnocellular system is involved primarily with motion analysis. The parvocellular system is associated with colour selectivity and shows a preference for high spatial frequency stimuli (22). The findings in the present study is suggestive of some abnormality in the geniculate afferents to V1 which is consistent with a defect in the magnocellular pathway at the level of Visual Area 1 or earlier indicating a loss of visual performance at a very early stages of visual processing in the slow learners which may be one of the contributing factors of the child's failure to perform. Electrophysiological methods may reveal underlying immaturity or abnormal brainstem timing and may serve as reliable tool in individuals with learning difficulties.

Other areas of information processing like auditory and central processing needs to be explored in this group to identify them at the earliest so that proper adjustment can be made for them.

#### ACKNOWLEDGEMENTS

We are grateful to Dr. Pusplata,

Psychologist, Child Guidance Clinic, Amar Jyoti School, Delhi for conducting the psychological evaluation of our subjects. We are also grateful to Amar Jyoti Institute, Delhi, for their full co-operation during the study. We would like to thank Mr. Manjhi for the technical assistance provided by him during the study.

#### REFERENCES

1. Tannock R. Reading Disorder. In: Sadock BJ, Sadock VA, eds. *Kaplan & Sadock's Comprehensive Textbook of Psychiatry*. Lippincott Williams & Wilkins 2005; 3107-3116.
2. Pratinidhi AK, Kurulkar PV, Garad SG, Dalal M. Epidemiological aspects of school Dropouts in Children Between 7-15 Years in Rural Maharashtra. *Ind J Pediatr* 1992; 59: 423-427.
3. Masi G, Marcheschi M, Pfanner P. Adolescents with borderline intellectual functioning: psychopathological risk. *Adolescence* 1998; 33: 415-424.
4. Karande S, Kanchan S, Kulkarni M. Clinical and Psychoeducational profile of children with borderline intellectual Functioning. *Ind J Pediatr* 2008; 75: 795-800.
5. Karande S, Kulkarni M. Poor school performance. *Ind J Pediatr* 2005; 72: 961-967.
6. Chaudhari S, Bhalerao MR, Chitale A, Pandit AN, Nene U. Pune low birth weight study a six year follow up. *Ind J Pediatr* 1990; 36: 669-676.
7. Ivanovic DM, Perez HT, Olivares MG, Diaz NS, Leyton BD, Ivanovic RM. Scholastic achievement: a multivariate analysis of nutritional, intellectual, socioeconomic, sociocultural, familial, and demographic variables in Chilean school age children. *Nutrition* 2004; 20: 878-889.
8. Celesia GG. Visual Evoked Potentials in Clinical Neurology. In: Aminoff MJ, eds. *Electrodiagnosis in Clinical Neurology*. New York, Churchill Livingstone 1992; 467-489.
9. Kavale KA, Forness SR. History, definition and diagnosis. In Singh NN and Beale IL eds. *Learning disabilities: Nature theory and treatment*. New York Springer-Verlag 1992; 3-43.
10. Tandon OP, Ram D. Visual Evoked Responses to pattern reversal in Children. *Ind J Physiol Pharmacol* 1991; 35: 175-179.
11. Watson CS, Kidd GR, Horner DG, Connell PJ, Lowther A, Eddins DA et al. Sensory, Cognitive, and Linguistic Factors in the Early Academic Performance of Elementary School Children. *J Learn Disabil* 2003; 36: 165-197.
12. Lehmkuhle S, Garzia RP, Turner L, Hash T, Baro JA. A defective visual pathway in children with reading disability. *N Engl J Med* 1993; 328: 989-996.
13. Galaburda A, Livingstone M. Evidence for a Magnocellular Defect in Developmental Dyslexia. *Ann NY Acad Sci* 1993; 682: 70-82.
14. Kubova Z, Kuba M, Peregrin J, Novakova V. Visual evoked potential evidence for magnocellular system deficit in dyslexia. *Physiol Res* 1995; 44: 87-89.
15. Mason A, Cornelissen P, Fowler S, Stein J. Contrast sensitivity, ocular dominance and specific reading disability. *Clin Vision Sci* 1993; 8: 345-353.
16. Martin F, Lovegrove W. Flicker contrast sensitivity in normal and specifically disabled readers. *Perception* 1987; 16: 215-221.
17. Livingstone MS, Rosen GD, Drislane FW, Galaburda AM. Physiological and anatomical evidence for a magnocellular defect in developmental dyslexia. *Proc Natl Acad Sci USA* 1991; 88: 7943-7947.
18. Johannes S, Kussmaul CF, Munte TF, Mangun GR. Developmental dyslexia: Passive visual stimulation provides no evidence for a magnocellular processing defect. *Neuropsychologia* 1996; 34: 1123-1127.
19. Celesia GG, Tobimatsu S. Electroretinograms to flash and to patterned visual stimuli in retinal and optic nerve disorders. In: Desmedt JE, eds. *Visual Evoked Potentials*. Amsterdam, Elsevier 1990; 45.
20. Bodis-Wollner I, Brannan JR, Ghilardi MF, Mulin LH. The importance of physiology to visual evoked potentials. In: Desmedt JE, eds: *Visual Evoked Potentials*. Amsterdam, Elsevier 1990; 1.
21. Lennie P, Trevarthen C, Van Essen D, Wassle H. Parallel processing of visual information. In: Spillmann L, Werner JS, eds. *Visual Perception*. San Diego: *The Neurophysiological Foundations Academic Press* 1990; 103.
22. Schiller PH, Logothetis NK. The color-opponent and broad-band channels of the primate visual system. *TINS* 1990; 13: 392.