Research Article

DOI: 10.5455/2349-3933.ijam20140506

A study of visual evoked potentials and effect of relaxation technique in patients with migraine

Dalia A. Biswas*, Meghana A. Gaikwad

Department of Physiology, Jawaharlal Nehru Medical College, Sawangi (Meghe), Wardha, Maharashtra, India

Received: 25 April 2014 Accepted: 8 May 2014

*Correspondence:

Dr. Dalia A. Biswas,

E-mail: dalia_biswas@yahoo.co.in

© 2014 Biswas DA et al. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

It is a cross-sectional comparative study in which comparison was done between 1) group A comprising of 15 cases of migraine (without aura) on medication only, 2) group B comprising of 15 cases of migraine (without aura) and practicing Rajyoga meditation and deep breathing and 3) control group who were 15 age matched normal subjects. This study was conducted in the interictal period. The most striking finding was - A decrease in P_{100} (ms) at post-test in both the eyes in group B, (though this finding is not significant), in contrast to group A which showed a significant increase in P_{100} (ms) at post-test. Both the groups showed an increase in P_{100} - N_{75} (μv) & this finding was not significant in both. Multiple comparisons using Tukey test and one way ANOVA showed significant finding between groups and within groups for P_{100} and N_{145} latencies for the right eye. No such difference was seen with left eye. We conclude that Rajyoga meditation and deep breathing can be used as adjuncts to routine antimigraine therapy. We advocate the continuous practice of these interventions which might decrease the frequency of attacks & finally the elimination of this problem.

Keywords: Rajyoga meditation, Migraine, Antimigraine therapy

INTRODUCTION

Migraine is a common disorder seen in the neurology outpatient department, all over the world. It is a much under-diagnosed and under-managed disorder. These cases are usually passed off as headache without characterization. In view of inadequate and correct diagnosis, such cases are deprived of specific treatment. Absence of empathy and accuracy of diagnosis by the physician are partly responsible for the recurrence of episodes of headache leading to anxiety and depression. Various causes of this problem have been hypothesized based on family researches. There are a number of genetic loci, which have been speculated as the primary defects in migraine patients. Familial migraine is the only type of migraine having a definite genetic basis. Among the families with a history of headache, a large number of

cases showed inheritance from the maternal side.¹ Stress is a well-known trigger factor for migraine.² There is lack of data regarding the epidemiology of migraine in India, specially the family researches. Literature review shows that migraine is more frequent in the female population. An epidemiological report from United States showed migraine in 18% women and 6% men with a female:male ratio of 3:1.³

The international headache society criteria classification⁴ shows majority of the patients of migraine did not have aura during the attacks³ and the prevalence was 83.8%. Migraine with aura was less prevalent while complications due to migraine were rare.

Irrespective of its genetic basis, the incidence and severity of the headache is modified by environmental

factors. The most common factors are travel, hunger, mental tension, lack of sleep, watching television and various odor producing substances ranging from perfumes and incense to petrol, cigarette smoke and body odor.⁵

Visual symptoms and photophobia are common features of migraine, but are not exclusively confined to attacks. Hypersensitivity to light and grating patterns of definite spatial frequency have been shown to persist even between attacks. Recent researches have shown that, differences in information processing are not limited to the visual system only but are also associated with central hypersensitivity, which may be genetic to some extent. The important aspect of central information-processing dysfunction is found to be reduced habituation to stimuli, specially, for Visual Evoked Potentials (VEP). A widely accepted and standardized test to assess excitability in the occipital cortex is the Visual Evoked Potential (VEP).

Lifestyle and behavior modification programmes can significantly lower the frequency and severity of migraines in some people. Stress management, relaxation techniques, and cognitive behavioral therapy don't work for all people for migraine prevention. However, these techniques can be pursued without fear of unwanted side effects. By reducing stress levels, many individuals also reduce muscle tension in the neck and shoulder area, which can both aggravate and cause headaches. Thus, this study was taken up to check the hypothesis that relaxation method like Rajyoga meditation has beneficial effects in migraine patients (without aura) as assessed by Pattern Reversal Visual Evoked Potentials (PRVEP).

METHODS

This study was conducted in the neuro-physiology lab of department of physiology, Jawaharlal Nehru medical college, Sawangi (Meghe), Wardha.

It is a cross-sectional comparative study. Group A and B consisted of patients attending the Acharya Vinoba Bhave Rural Hospital (A.V.B.R.H.) Sawangi medicine out-patient department. The diagnosis of migraine was confirmed at the time of patient recruitment based on criteria laid down by the International Headache Society (IHS) in 1988.

Subjects

All subjects had normal visual acuity and no visual abnormalities. Migraine frequency varied between 1-2 attacks per week to 1 attack per 6 mths. Group A consisted of 15 migraine subjects on medication only. Group B consisted of 15 migraine subjects on medication and practicing deep breathing and Rajyoga meditation. The control group were 15 ages matched normal subjects. This study was conducted in the interictal period. The subjects fulfilled the International Headache Society (IHS) criteria for diagnosis of migraine.⁴

Inclusion criteria for the study

- 1. Patients having episodes of migraine headaches for at least 2 years and suffered at least 2 attacks per month in the last quarter year.
- 2. Not taken preventive therapy for migraine during the preceding 6 months.
- 3. Occasional use of analgesics, ergotamine, sumatriptan and antiemetics for acute attack.
- 4. No other neurological, ophthalmological or systemic disease known to cause abnormalities in VEP.
- 5. With normal or corrected normal vision.

Exclusion criteria for study group

- Patients with any neurological, ophthalmological or systemic disease known to cause abnormalities in VEP.
- 2. Patients on preventive therapy for migraine during the preceding 6 months.

Study parameters

- 1. Peak P₁₀₀ latencies.
- 2. P_{100} amplitude (N_{75} - P_{100}).
- 3. Interocular latency difference.

Experimental design and recordings

Subjects were sited at a distance of 100 cms from a TV monitor, which was attached to a polywrite (RMS make). The TV monitor displayed a reversal checker board pattern with a fluctuating red point. The subjects were asked to concentrate on this red point. A 2 channel montage was used for recording VEP which is as follows - Channel 1 as O_z to Fp_z and channel 2 as O_z to linked ear.

The ground electrode was placed at the vertex labelled as Cz. The recording electrode was placed at Oz, using electrode paste. The reference electrode was placed at Fpz or 12 cm above the nasion. These positions were as per 10-20 international system of EEG electrode placements. The skin was prepared by shaving the appropriate parts of the scalp after its degreasing. The electrode impedance was kept below 5 K Ω . Monocular stimulation was used, to avoid masking of a unilateral conduction abnormality. The stimulus reversal rate was 2/sec and the visual angle subtended by the checker board was 60°. The fixation point for full field size was 15° by 12⁰. The size of pattern elements was 14"-16" with rate of stimuli at 6 Hz. The mean luminance of the central field was 50 cd/m², with a background luminance of 30 cd/m². These technicalities were as per technical

recommendation for VEP study (IFCN). The latency of N_{75} , P_{100} , N_{145} & P_{100} - N_{75} ($\mu\nu$) was measured.

Relaxation technique

Comprised of deep breathing and Rajyoga meditation.

Deep breathing

It is simple breathing through the nose, letting the stomach expand as much as possible. The hands are firmly and comfortably placed on the r stomach during the exercise. After deep inspiration, breath was held for a few seconds, and then expired slowly through the mouth. This procedure is repeated for 3 or 4 times a day.

Meditation

Rajyoga meditation was practiced in 3 steps namely-Initiation, Concentration & Realization.

Statistical Analysis

It was done using SPSS version 14 and significance was set at P <0.05. Student's t test, Tukey test and one way ANOVA was used for multiple comparisons.

RESULTS

The age group of subjects was from 11-40 years with a mean age of 26 years as seen from Table 1. 8 male subjects and 7 female subjects comprised both the groups with an age matched control group having 9 males and 6 females respectively (Table 2).

Table 1: Age wise distribution of patients in three groups.

Age group (years)	Group A	Group B	Control group
11-20	9 (60%)	9 (60%)	9 (60%)
21-30	0 (0%)	0 (0%)	2 (13.33%)
31-40	6 (40%)	6 (40%)	4 (26.67%)
Total	15 (100%)	15 (100%)	15 (100%)
Mean ± SD	25.80 ± 9.11	25.80 ± 9.11	24.80 ± 8.60

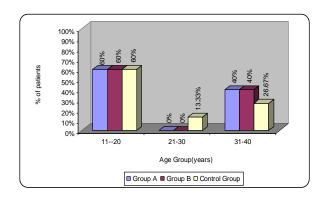


Figure 1: Age wise distribution of patients in three groups.

Table 2: Gender wise distribution of patients in three groups.

Gender	Group A	Group B	Control group
Male	8 (53.33%)	8 (53.33%)	9 (60%)
Female	7 (46.67%)	7 (46.67%)	6 (40%)
Total	15 (100%)	15 (100%)	15 (100%)

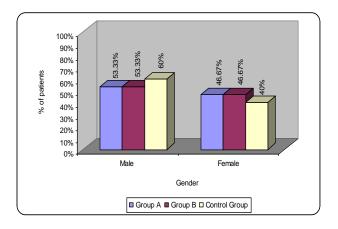


Figure 2: Gender wise distribution of patients in three groups.

A comparison of VEP parameters in group A at pre-test for right and left side showed a significant P_{100} (ms) finding whereas, post test data showed both P_{100} (ms) and N_{145} (ms) as significant finding. The P_{100} (ms) showed an increase in the left eye at post-test which indicates no beneficial effect (Table 3 & 4).

Table 3: Comparison of VEP parameters in groups A at pre-test for right and left side.

	Left	Right	t value	P value
N ₇₅ (ms)	84.24 ± 14.16	83.25 ± 17.81	0.217	0.831 NS, P > 0.05
P ₁₀₀ (ms)	103.82 ± 14.93	114.86 ± 12.36	2.394	0.031 S, P < 0.05
N ₁₄₅ (ms)	137.16 ± 13.73	145.34 ± 14.71	1.215	0.245 NS, P > 0.05
P ₁₀₀ -N ₇₅ (μv)	8.98 ± 0.89	9.00 ± 0.71	0.213	0.834 NS, P > 0.05

Table 4: Comparison of VEP parameters in groups A at post-test for right and left side.

	Left	Right	t value	P value
N ₇₅ (ms)	79.18 ± 14.87	76.90 ± 11.11	0.391	0.702 NS, P > 0.05
P ₁₀₀ (ms)	108.63 ± 14.87	92.05 ± 11.59	4.887	0.000 S, P < 0.05
N ₁₄₅ (ms)	134.20 ± 19.17	118.78 ± 15.93	2.170	0.048 S, P < 0.05
P_{100} - N_{75} (μv)	9.26 ± 0.81	9.37 ± 0.70	2.086	0.056 NS, P > 0.05

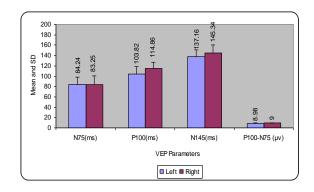


Figure 3: Comparison of VEP parameters in groups A at pre-test for right and left side.

Figure 4: Comparison of VEP parameters in groups A at post-test for right and left side.

Table 5: Comparison of VEP parameters in groups B at pre-test for right and left side.

	Left	Right	t value	P value
N ₇₅ (ms)	86.60 ± 12.69	81.07 ± 11.95	1.691	0.113 NS, P > 0.05
P ₁₀₀ (ms)	110.53 ± 14.07	114.88 ± 10.82	1.061	0.306 NS, P > 0.05
N ₁₄₅ (ms)	140.58 ± 13.12	144.30 ± 13.47	0.709	0.490 NS, P > 0.05
P_{100} - N_{75} (μv)	8.82 ± 0.99	8.84 ± 0.92	0.100	0.921 NS, P >0.05

Table 6: Comparison of VEP parameters in groups B at post-test for right and left side.

	Left	Right	t value	P value
N ₇₅ (ms)	84.78 ± 12.36	83.38 ± 15.49	0.365	0.721 NS, P > 0.05
P ₁₀₀ (ms)	109.10 ± 12.87	111.74 ± 13.26	0.784	0.446 NS, P > 0.05
N ₁₄₅ (ms)	139.38 ± 12.42	142.38 ± 13.61	0.650	0.526 NS, P > 0.05
P_{100} - N_{75} (μv)	8.97 ± 0.92	9.04 ± 0.87	0.496	0.628 NS, P > 0.05

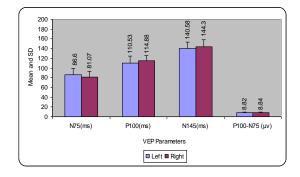


Figure 5: Comparison of VEP parameters in groups B at pre-test for right and left side.

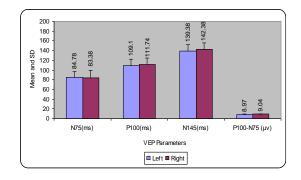


Figure 6: Comparison of VEP parameters in groups B at post-test for right and left side.

Table 7: Comparison of VEP parameters in control group right and left side.

	Left	Right	t value	P value
N ₇₅ (ms)	82.80 ± 12.73	82.92 ± 12.83	0.491	0.631 NS, P > 0.05
P ₁₀₀ (ms)	102.48 ± 15.25	103.68 ± 14.66	0.884	0.392 NS, P > 0.05
N ₁₄₅ (ms)	138.16 ± 16.87	139.81 ± 16.93	0.762	0.459 NS, P >0.05
P_{100} - N_{75} (μv)	8.86 ± 0.91	8.92 ± 0.81	1.058	0.308 NS, P > 0.05

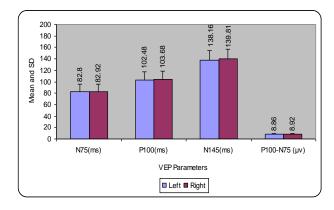


Figure 7: Comparison of VEP parameters in control group right and left side.

For group B, the pre-test and post-test for right and left eye showed no significant finding , though a decrease in the latency was observed regarding N_{75} , P_{100} and N_{145} (ms) at post-test. This decrease in latency at post test is an important finding (Table 5 & 6). Table 7, showed no interocular latency & amplitude difference in the control group.

The comparison of VEP parameters in group A at pre and post-test of left eye showed a significant P_{100} - N_{75} ($\mu\nu$) finding (Table 8). Whereas for right eye as per Table 9, a significant finding of 2 latencies namely P_{100} (ms) and N_{145} (ms) was seen. P_{100} - N_{75} ($\mu\nu$) finding was also significant (Table 9). When these same comparisons were done for group B of left & right eye, it was seen that the finding of P100- N_{75} ($\mu\nu$) was significant (Table 10 & 11).

Table 8: Comparison of VEP parameters in group A, pre and post-test, left eye.

	Pre-test	Post-test	t value	P value
N ₇₅ (ms)	84.24 ± 14.16	79.18 ± 14.87	0.959	0.354 NS, P > 0.05
P ₁₀₀ (ms)	103.82 ± 14.93	108.63 ± 14.87	0.854	0.407 NS, P > 0.05
N ₁₄₅ (ms)	137.16 ± 13.73	134.20 ± 19.17	0.510	0.618 NS, P > 0.05
P_{100} - N_{75} (μv)	8.98 ± 0.89	9.26 ± 0.81	3.400	0.004 S, P < 0.05

Table 9: Comparison of VEP parameters in group A, pre and post-test, right eye.

	Pre-test	Post-test	t value	P value
N ₇₅ (ms)	83.25 ± 17.81	76.90 ± 11.11	1.283	0.220 NS, P > 0.05
P ₁₀₀ (ms)	114.86 ± 12.36	92.05 ± 11.59	5.066	0.000 S, P < 0.05
N ₁₄₅ (ms)	145.34 ± 14.71	118.78 ± 15.93	5.710	0.000 S, P < 0.05
P_{100} - N_{75} (μv)	9.00 ± 0.71	9.37 ± 0.70	3.157	0.007 S, P < 0.05

Table 10: Comparison of VEP parameters in group B, pre and post-test, left eye.

	Pre-test	Post-test	t value	P value
N ₇₅ (ms)	86.60 ± 12.69	84.78 ± 12.36	1.469	0.164 NS, P > 0.05
P ₁₀₀ (ms)	110.53 ± 14.07	109.10 ± 12.87	1.452	0.169 NS, P > 0.05
N ₁₄₅ (ms)	140.58 ± 13.12	139.38 ± 12.42	1.061	0.306 NS, P > 0.05
P ₁₀₀ -N ₇₅ (μv)	8.82 ± 0.99	8.97 ± 0.92	2.323	0.036 S, P < 0.05

Table 11: Comparison of VEP parameters in group B, pre and post-test, right eye.

	Pre-test	Post-test	t value	P value
N ₇₅ (ms)	81.07 ± 11.95	83.38 ± 15.49	1.840	0.087 NS, P > 0.05
P ₁₀₀ (ms)	114.88 ± 10.82	111.74 ± 13.26	1.839	0.087 NS, P > 0.05
N ₁₄₅ (ms)	144.30 ± 13.47	142.38 ± 13.61	1.000	0.334 NS, P > 0.05
P_{100} - N_{75} (μv)	8.84 ± 0.92	9.04 ± 0.87	3.623	0.003 S, P < 0.05

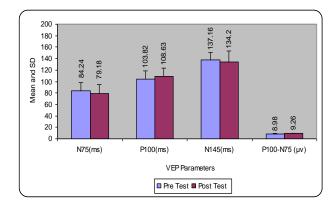


Figure 8: Comparison of VEP parameters in group A, pre and post-test, left eye.

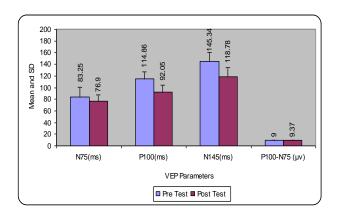


Figure 9: Comparison of VEP parameters in group A, pre and post-test, right eye.

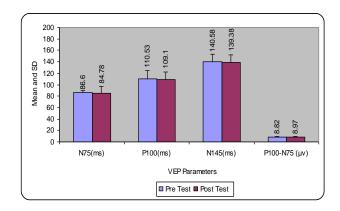


Figure 10: Comparison of VEP parameters in group B, pre and post-test, left eye.

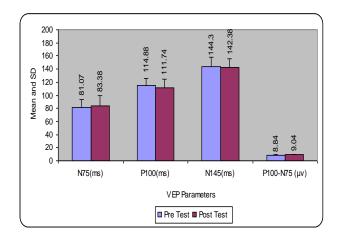


Figure 11: Comparison of VEP parameters in group B, pre and post-test, right eye.

Thus it was seen that the group B showed an important finding of decrease in P_{100} latency at post-test in both the eyes in contrast to left eye finding of group A.

The multiple comparison using Tukey test between and within groups for left eye showed non-significant finding (Table 12), but for right eye a significant finding was observed in relation to P_{100} & N_{145} latencies (Table 14).

Table 12: Comparison of N_{75} , P_{100} , N_{145} and P_{100} - N_{75} in three groups: left eye (Multiple comparison: Tukey test) descriptive statistics.

		N	Mean ± SD	Std. error
	Group A	15	79.18 ± 14.87	3.84
N ₇₅	Group B	15	84.78 ± 12.36	3.19
	Control	15	82.80 ± 12.73	3.28
	Group A	15	108.63 ± 14.87	3.84
P_{100}	Group B	15	109.10 ± 12.87	3.32
	Control	15	102.48 ± 15.25	3.93
	Group A	15	134.20 ± 19.17	4.95
N ₁₄₅	Group B	15	139.38 ± 12.42	3.20
	Control	15	138.16 ± 16.87	4.35
	Group A	15	9.26 ± 0.81	0.20
P_{100} - N_{75}	Group B	15	8.97 ± 0.92	0.23
	Control	15	8.86 ± 0.91	0.23

Table 13: One way ANOVA.

	Source of variation	Sum of squares	df	Mean square	F	P value	
	Between groups	242.034	2	121.017		0.514 NS, P >0.05	
N ₇₅	Within groups	7506.517	42	178.727	0.677		
	Total	7748.551	44				
	Between groups	408.677	2	204.339		0.380 NS, P >0.05	
P ₁₀₀	Within groups	8675.411	42	206.557	0.989		
	Total	9084.088	44		-		
	Between groups	219.830	2	109.915		0.667 NS, P >0.05	
N ₁₄₅	Within groups	11293.083	42	268.883	0.409		
	Total	11512.912	44				
	Between groups	1.321	2	0.661			
P ₁₀₀ -N ₇₅	Within groups	32.839	42	0.782	0.845	0.437 NS, P > 0.05	
	Total	34.160	44		-		

Note: F-value is insignificant; Tukey multiple comparison test cannot be applied

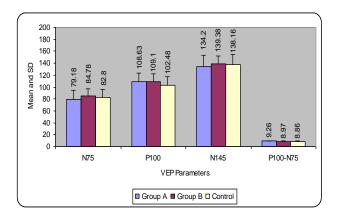


Figure 12: Comparison of N_{75} , P_{100} , N_{145} and P_{100} - N_{75} in three groups: left eye.

Table 14: Comparison of N_{75} , P_{100} , N_{145} and P_{100} - N_{75} in three groups: right eye (Multiple comparison: Tukey test) descriptive statistics.

		N	Mean ± SD	Std. error
	Group A	15	76.90 ± 11.11	2.86
N ₇₅	Group B	15	83.38 ± 15.49	4.00
	Control	15	82.92 ± 12.83	3.31
	Group A	15	92.05 ± 11.59	2.99
P ₁₀₀	Group B	15	111.74 ± 13.26	3.42
	Control	15	103.68 ± 14.66	3.78
	Group A	15	118.78 ± 15.93	4.11
N ₁₄₅	Group B	15	142.38 ± 13.61	3.51
	Control	15	139.81 ± 16.93	4.37
	Group A	15	9.37 ± 0.70	0.18
P_{100} - N_{75}	Group B	15	9.04 ± 0.87	0.22
	Control	15	8.92 ± 0.81	0.21

Table 15: One way ANOVA.

	Source of variation	Sum of squares	df	Mean square	F	P value	
N ₇₅	Between groups	393.04	2	196.52			
	Within groups	7398.52	42	176.15	1.11	0.337 NS, P > 0.05	
	Total	7791.57	44				
P ₁₀₀	Between groups	2940.62	2	1470.31		0.001 S, P < 0.05	
	Within groups	7353.87	42	175.09	8.39		
	Total	10294.49	44		•		
N ₁₄₅	Between groups	5029.74	2	2514.87			
	Within groups	10160.88	42	241.92	10.39	0.000 S, P < 0.05	
	Total	15190.63	44				
P ₁₀₀ -N ₇₅	Between groups	1.65	2	0.82			
	Within groups	27.00	42	0.64	1.28	0.287 NS, P >0.05	
	Total	28.66	44				

Group		Mean difference	Std.	P value	95% confidence interval		
			(I-J)	error	1 value	Lower bound	Upper bound
P ₁₀₀	Group A	Group B	-19.69	4.83	0.001 S, P < 0.05	-31.43	-7.95
		Control	-11.63	4.83	0.053 NS, P > 0.05	-23.37	0.10
	Group B	Control	8.06	4.83	0.229 NS, P > 0.05	-3.67	19.79
N ₁₄₅	Group A	Group B	-23.60	5.67	0.000 S, P < 0.05	-37.39	-9.80
		Control	-21.03	5.67	0.002 S, P < 0.05	-34.83	-7.23
	Group B	Control	2.56	5.67	0.894 NS, P > 0.05	-11.23	16.36

Table 16: Multiple comparison: Tukey test.

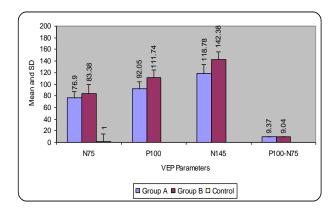


Figure 13: Comparison of N_{75} , P_{100} , N_{145} and P_{100} - N_{75} in three groups: right eye.

DISCUSSION

This study was conducted in the interictal period. The most striking finding was-

- A decrease in P₁₀₀ (ms) at post-test in both the eyes in Group B though this finding is not significant. In contrast in Group A there was a significant increase in P₁₀₀ (ms) at post-test.
- Both the groups showed an increase in P_{100} - N_{75} ($\mu\nu$) & this finding was not significant in both.
- Multiple comparisons using Tukey test and one way ANOVA showed significant finding between groups and within Groups for P₁₀₀ and N₁₄₅ latencies for the right eye. No such difference was seen with left eye.

Abnormalities of visual evoked potentials were first reported by Kennard et al using pattern reversal method in migraine patients. He found that the latency of P₁₀₀ was greater and its amplitude was larger in migraine patients. Our prolonged latency findings at pre-test is in line with findings of other workers. Hennard et al. suggested that repeated attacks cause ischemic damage which might cause the increase in latency. In clinical setups many migraine patients are disturbed by noises

and bright lights. In fact, in many cases, noises and bright light have precipitated migraine attacks. 11,12 If we test these patients neurophysiologically we would get decreased latencies but what we found was increased latencies, which might be due to synaptic delays. Also, it may suggest that cortex is hypoexcitable in between attacks. Rajyoga meditation causes shifting of autonomic balance in favour of parasympathetic instead of sympathetic. This explains the decrease in the latencies at post-test. There are no specific researches available explaining the effects of Rajyoga meditation on VEP in migraine patients but the above mentioned reason explains its overall beneficial effect on the body.

Studies of the cortical VEP have shown contradictory results. Review by Schoenen1992 showed inability of researchers to demonstrate differences between migrainers and control group. ¹⁶ Increase in P_{100} (μv) was seen by Diener et al 1984 & Khalil 1991 for subjects having migraine less than 10 years. 17,18 Skuse & Burke 1992¹⁹ have found that prolonged checker board stimulation causes a progressive decrease in P_{100} - N_{75} (μv) in the absence of drowsiness, when examined by reaction time task. This is due to habituation. Thus, habituation of VEP is a physiological phenomenon in the visual cortex, which is absent in migraine patients in between attacks, and thus a large amplitude is obtained. . This finding is in line with our study at pre-test. Similar finding was also seen at post-test which indicates that recovery of cortical damage is not complete with these interventions.

The habituation activity of cortex depends on certain neurotransmitters like serotonin, dopamine, histamine, noradrenaline and acetylcholine. These transmitters have diffuse innervations of the sensory cortices mainly the layer IV pyramidal cells and interneurons. The serotoninergic neurons in the raphe nuclei play a main modulatory role in cortical information processing. Perview by Ferrari 1992, and in his topic on migraine pathogenesis reports the role of serotonin in causing low interictal activity in the raphe-cortical-serotoninergic pathway which could be responsible for a low preactivation level of sensory cortices which could cause both increased detection thresholds and a wide range of suprathreshold activation before reaching a saturation or

"ceiling" effect.24 Thus this deficient habituation again might be the cause of larger amplitudes in migraine patients. Our finding of increased amplitude even after intervention might also point to the role of abnormal genes as found in a study by Ophoff et al. 1996. His hypothesis is that the major functional outcome in the brain activity can be changed by neurotransmitter system also includes the subcortical-corticalwhich serotoninergic pathway since the identified genes codes, for the ionophore of a P/Q calcium channel that regulates transmitter release, which is responsible for lack of habituation.²⁵ Another cause for this increase in the amplitude could be an excessive increase in cortical lactate levels which may induce a metabolical instability like lactate accumulation triggering a spreading depression.²⁶ Recent study by Watanabe et al. 1996 showed increased lactate levels in the occipital cortex in migraine patients.

Significant interocular latency difference as regards to P_{100} and N_{145} was seen in right eye as compared to left eye. No specific data is available to explain such difference. However, Ipata et al. assessed the interhemispheric visual transfer of information in humans. They found that more anterior locations of electrode sites yielded shorter values and overall transfer time which tended to be shorter for N_{75} component than for P_{100} component. 27,28

A further study can be taken up on a larger group of subjects comparing duration of migraine and latency, repetitive attacks and amount of ischemic damage, physiological and biochemical parameters, location of electrode site and migraine attacks and interhemispheric transfer of visual information as compared to right and left eye.

CONCLUSION

Thus, we can conclude that Rajyoga meditation along with deep breathing can be used as adjuncts to routine antimigraine therapy. We advocate the continuous practice of these interventions which might decrease the frequency of attacks and finally the elimination of this problem.

ACKNOWLEDGEMENTS

The authors wish to acknowledge the help rendered by the staff of Neurophysiology section of physiology department at Jawaharlal Nehru Medical College, Wardha, the subjects and the staff of ophthalmology and medicine departments of AVBR Hospital, Wardha.

Funding: No funding sources Conflict of interest: None declared

Ethical approval: The study was approved by the

institutional ethics committee

REFERENCES

- 1. Samhita Panda, Manjari Tripathi. Clinical profile of migraineurs in a referral centre in India. J Assoc Physicians India. 2005 Feb;53:111-5.
- Gianluca Coppola, Antonio Curra, Simona Liliana Sava, Alessia Alibardi Vincenzo Parisi, Francesco Pierelli et al. Changes in visual-evoked potential habituation induced by hyperventilation in migraine. J Headache Pain. 2010;11:497-503.
- 3. Stewart WF, Lipton RB, Celentano DD, Reed ML. Prevalence of migraine in the United States. Relation to age, income, race and other sociodemographic factors. J Am Med Assoc. 1992;267:64-9.
- Headache Classification Committee of the International Headache Society. Classification and diagnostic criteria for headache disorders, cranial neuralgias and facial pain. Cephalalgia. 1988;8(Suppl 7):1-96.
- 5. Ottman R, Hong S, Lipton RB. Validity of family history data on severe headache and migraine. Neurol. 1993;43:1954-60.
- 6. Migraine prevention: techniques are available, but underutilized. New guidelines emphasize ways to reduce the number of migraine attacks. Duke Med Health News. 2012 Jul;18(7):6-7.
- KennardC, Gawel M, Rudolph Nde M, Rose FC. Visual evoked potentials in migraine subjects. Res Clin Stud Headache. 1987;6;73-80.
- 8. Winter A. Electrophysiological studies of the visual system of people with classical migraine. In: Winter A, eds. Ph.D. thesis. Surrey, UK: University of Surrey, 1985.
- 9. Polich J, Ehlers CL, Dalassio DJ. Pattern shift visual evoked responses and EEG in migraine. Headache. 1986;26(9):451-6.
- 10. MarianiE, MoschiniV, Pastorino GC et al. Pattern reversal visual evoked potential (VEP-PR) in migraine subjects with visual aura. Headache. 1990;30(7):435-8.
- 11. Wilkins A, Nimmo-Smith I, Tait A, McManus C, Della Sala S, Tilley A et al. A neurological basis for visual discomfort. Brain. 1984;107:989-1017.
- 12. Marcus DA, Soso MJ. Migraine and stripe induced visual discomfort. Arch Neurol. 1989;46:1129-32.
- Headache Classification Committee of the International Headache Society. Classification and diagnostic criteria for headache disorder, cranial neuralgias and facial pain. Cephalalgia. 1988;8(Suppl 7):1-96.
- 14. Afra J, Proietti Cecchini A, De Pasqua V, Albert A, Schoenen J. Visual evoked potentials during long periods of pattern reversal stimulation in migraine. Brain. 1998;121:233-41.
- Neelam D. Sukhsohale, Mrinal S. Phatak. Effect of short term & long term Brahmakumaris Rajayoga meditation on Physiological variables. Indian J Physiol Pharmacol. 2012;56(4):388-92.

- Schoenen J. Abnormal cortical information processing between migraine attacks. In: Sandler M, Ferrari M, Harnett S, eds. Migraine: Pharmacology and Genetics. 1st ed. London: Altman; 1996: 233-253.
- Diener HC, Ndosi NK, Koletzki E, Langohr HD. Visual evoked potentials in migraine. In: Pfaffenrath V, Lundberg PJ, Sjaastad O, eds. Updating in Headache. 2nd ed. Berlin: Springer-Verlag; 1984: 439-465.
- 18. Khalil NM. Investigations of visual function in migraine using visual evoked potentials and visual psychological tests. In: Khalil NM, eds. PhD thesis. London: University of London; 1991.
- 19. Skuse NF, Burke D. Sequence dependent deterioration in the visual evoked potential in the absence of drowsiness. Electroencephalogr Clin neurophysiol. 1992;84:20-5.
- Judit Afra, Alberto Proietti Cecchini, Victor De Pasqua, Adelin Alberta, Jean Schoenen. Visual evoked potentials during long periods of pattern reversal stimulation in migraine. Brain. 1998;121:233-41.
- 21. Mesulam MM. Large-scale neurocognitive networks and distributed processing for attention, language and memory. Ann Neurol. 1990;28:597-613.

- 22. Jacobs BL, Azmitia EC. Structure and function of the brain serotonin system. Physiol Rev. 1992;72:165-229.
- 23. Ferrari MD. Biochemistry of migraine. Pathol Biol (Paris). 1992;40:284-92.
- 24. Knott JR, Irwin DA. Anxiety, stress, and the contingent negative variation. Arch Gen Psychiatr. 1973; 29:538-41.
- 25. Ophoff RA, Terwindt GM, Vergouwe MN, Eijk R van, Oefner PJ, Hoffman SM et al. Familial hemiplegic migraine and episodic ataxia type 2 are caused by mutations in the Ca²⁺ channel gene Cacnlia4. Cell. 1996;87:543-52.
- Lauritzen M. Pathophysiology of the migraine aura.
 The spreading depression theory. Brain.
 1994:117:199-210.
- 27. Ipata A, Girelli M, Miniussi C, et al. Interhemispheric transfer of visual information in humans: the role of different callosal channels. Arch Ital Biol. 1997 Mar;135(2):169-82.
- 28. Andrew B. Evans, Selim R. Benbadis et al. Clinical utility of evoked potentials, 2014. Available at http://emedicine.medscape.com/article/1137451-overview. Accessed 18 March 2014.

DOI: 10.5455/2349-3933.ijam20140506 **Cite this article as:** Biswas DA, Gaikwad MA. A study of visual evoked potentials and effect of relaxation technique in patients with migraine. Int J Adv Med 2014;1:24-33.