

Multifocal visual evoked potential in optic neuritis, ischemic optic neuropathy and compressive optic neuropathy

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Purpose: To investigate the effect of optic neuritis (ON), ischemic optic neuropathy (ION) and compressive optic neuropathy (CON) on multifocal visual evoked potential (mfVEP) amplitudes and latencies, and to compare the parameters among three optic nerve disorders. **Materials and Methods:** mfVEP was recorded for 71 eyes of controls and 48 eyes of optic nerve disorders with subgroups of optic neuritis (ON, $n = 21$ eyes), ischemic optic neuropathy (ION, $n = 14$ eyes), and compressive optic neuropathy (CON, $n = 13$ eyes). The size of defect in mfVEP amplitude probability plots and relative latency plots were analyzed. The pattern of the defect in amplitude probability plot was classified according to the visual field profile of optic neuritis treatment trail (ONTT). **Results:** Median of mfVEP amplitude (log SNR) averaged across 60 sectors were reduced in ON (0.17 (0.13-0.33)), ION (0.14 (0.12-0.21)) and CON (0.21 (0.14-0.30)) when compared to controls. The median mfVEP relative latencies compared to controls were significantly prolonged in ON and CON group of 10.53 (2.62-15.50) ms and 5.73 (2.67-14.14) ms respectively compared to ION group (2.06 (-4.09-13.02)). The common mfVEP amplitude defects observed in probability plots were diffuse pattern in ON, inferior altitudinal defect in ION and temporal hemianopia in CON eyes. **Conclusions:** Optic nerve disorders cause reduction in mfVEP amplitudes. The extent of delayed latency noted in ischemic optic neuropathy was significantly lesser compared to subjects with optic neuritis and compressive optic neuropathy. mfVEP amplitudes can be used to objectively assess the topography of the visual field defect.

Key words: Compressive optic neuropathy, humphrey visual field, ischemic optic neuropathy, multifocal visual evoked potential, optic neuritis

Optic nerve disorder is a functional impairment of the optic nerve, which may be a manifestation of most common pathological processes such as inflammation, ischemia or compressive lesions that can be life threatening.^[1] The study of incidence of neuro-ophthalmology diseases in Singapore reported that optic nerve disorders, such as ischemic optic neuropathy (ION) and optic neuritis (ON), were found to be the one of the most common conditions that affects 1.08 per 100,000 and 0.83 per 100,000 population, respectively.^[2] The classic clinical signs of optic neuropathy are acute or chronic visual loss with visual field defect, dyschromatopsia, and presence of relative afferent pupillary defect (RAPD).^[3-7] Visual field testing is an important component of neuro-ophthalmic examination in diagnosing and monitoring the progression of optic nerve diseases. There is a considerable overlap of clinical features between various entities of optic neuropathy. Evaluation with imaging studies,^[8] electrophysiological,^[9,10] and serological and genetic testings^[6] are essential for establishing the correct diagnosis. The functional visual disturbances may manifest without neuro-radiological change.^[11,12] Both visual field and electrophysiological test can provide evidence of optic nerve/ganglion cell functional disturbances. Though automated perimetry testing remains the reliable means of assessing the visual field, it requires the full co-operation of the subject because of its subjective nature.^[13,14]

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Sutter (1991)^[15] and Baseler *et al.*, (1994)^[16] designed multifocal visual evoked potential (mfVEP) that evokes multiple locations of V1 area in response to pattern reversal stimulation. This explores an electrophysiological topographical map of the V1 area of the visual cortex.^[17,18] Amplitude decrease and prolonged latency have been reported in many disorders involving the impairment of conductivity along the optic nerve.^[9,10] To the best of our knowledge, there is limited research that has focused on the comparison of mfVEP parameters among different optic nerve disorders.

This study aims at investigating the effect of optic neuritis, ischemic optic neuropathy and compressive optic neuropathy on amplitudes and latencies of mfVEP and to compare them among these three optic nerve disorders.

Materials and Methods

Subjects

A total of 119 eyes (48 eyes of optic nerve disorders and 71 eyes of age matched controls) were recruited. Right eye of all controls and subjects with bilateral diseases were used for mfVEP analysis. Subjects, who reported to the clinic from the time of onset of symptoms, were found to be median (17.5 (13.15-51.85) days). Subjects who were diagnosed based on acute clinical signs and symptoms with 21 eyes of optic neuritis (ON), 14 eyes of non-arteritic anterior ischemic optic neuropathy (NAAION), and 13 eyes of compressive optic neuropathy (CON) were included as cases. Among CON subjects, there were six eyes of meningioma and seven eyes of pituitary adenoma^[19] based on MRI imaging were recruited as CON subjects. Patients with a known history of glaucomatous optic neuropathy, other optic nerve disorders and retinal diseases, which are known to affect the mfVEP responses, were not included for the study. All the

subjects had BCVA better than 20/600 in the affected eyes to allow a stable fixation throughout the multifocal visual evoked potential testing. Subjects, who had best corrected visual acuity of 20/20 or better and were found to have no ocular disease after comprehensive eye examination, were included as control group. All the subjects underwent a complete eye examination which included detailed medical and ocular history, best corrected visual acuity (BCVA) assessment, color vision assessment using Ishihara pseudoisochromatic plates, pupillary examination, slit lamp biomicroscopy, applanation tonometry, and dilated fundus evaluation. All subjects underwent Swedish Interactive Threshold Algorithm (SITA) FAST 30-2 or 24-2 protocol in Humphrey visual field (HVF) analyzer. mfVEP and HVF were performed within a week from the time of diagnosis. The study follows the principles of the Declaration of Helsinki and was done after consent from institutional review board was obtained. An informed consent was obtained from subjects prior to mfVEP testing.

mfVEP recording

The mfVEP stimulus was elicited using cortically scaled dartboard pattern with 60 sectors stimulus subtends an angle of 44.5° which was available in VERIS 5.2.2X software as shown in Fig. 1. Subjects viewed the stimulus on the CRT monitor (Nortech Imaging Technologies) monocularly without pupillary dilatation at 53 cm. Each sector contained eight black and eight white checks. Each sector underwent pattern reversal stimulation independently and simultaneously by a binary m sequence at the frame rate of 75 Hz. A band pass filter was set between 3 and 100 Hz and was sampled at 1200 Hz.

The mfVEP three channels recording model of Hood and Greenstein^[17] with Gold Disc electrodes (Grass Model F-E5GH, Astro-Med, Inc., West Warwick, RI) was followed. Three active electrodes were placed at 4 cm above the inion and at 4 cm lateral to and 1 cm above the inion on either side. The common reference electrode was placed on the inion and ground electrode on the forehead.

Data analysis

The first slice of second order kernel responses with highest signal-to-noise ratio (log SNR) was extracted from three recorded and three additional derived channels using mfVEP processing program from Professor Don Hood's

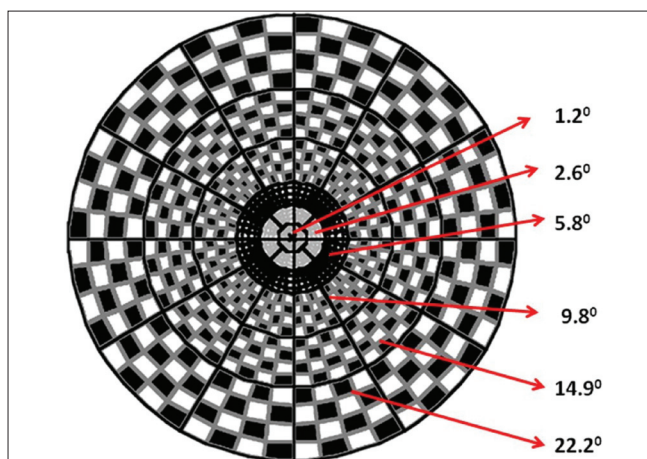


Figure 1: mfVEP stimulus

Laboratory in MATLAB 7 software (The Mathworks, Inc, Natick, MA, USA).^[17,20] Signal-to-noise ratios for each sector (log SNR) were calculated by dividing the root mean square (RMS) of the amplitude for each mfVEP response over a time interval of 45 to 150 ms by the average of the 60 RMS values of the noise-only window.^[21,22] The monocular amplitudes were represented in terms of signal-to-noise ratio (SNR).^[20-22] The monocular latency represented the relative delay in latency in comparison with the controls using cross-correlation function built in software.^[23,24]

The amplitude and latency probability plots of 60 sectors were obtained for each subject in comparison with normative database^[21] built in Multifocal VEP processing program. The monocular amplitude probability plots^[21,22] and latency probability plots^[23,24] were coded in square and circle respectively. The normal responses ($P > 0.05$) were marked in black. The depth of the defects for deviation from control with $P < 0.01$ and $P < 0.05$ was coded as saturated color and desaturated color, respectively. Blue and red color were used for right eye and left eye, respectively. The sectors with signal-to-noise ratio too small (i.e., < 1.7) in the monocular latency probability was indicated in grey circle.

mfVEP parameters analysis

The log SNR and relative latency of averaged over the 60 sectors of each monocular plots were compared among three optic nerve disorders and age matched controls. The number of adjacent abnormal points with $P < 0.05$ or $P < 0.01$ in amplitude and latency probability plots were counted to allow the topographical distribution of amplitude and latency defect in optic neuritis, ischemic optic neuropathy, and compressive optic neuropathy groups.

The pattern of the amplitude and latency defect in probability plots were classified based on the visual field profile of Optic Neuritis Treatment Trial (ONTT).^[25,26] The significant defects involving central 2 mfVEP rings have been classified as central scotoma, defect involving central four rings as cecentral scotoma, defect in upper or lower field of 6 mfVEP rings as altitudinal defect, temporal or nasal field defect of 6 mfVEP rings as hemianopia defect and defect involving all the quadrants in mfVEP six rings as diffuse or generalized defect.

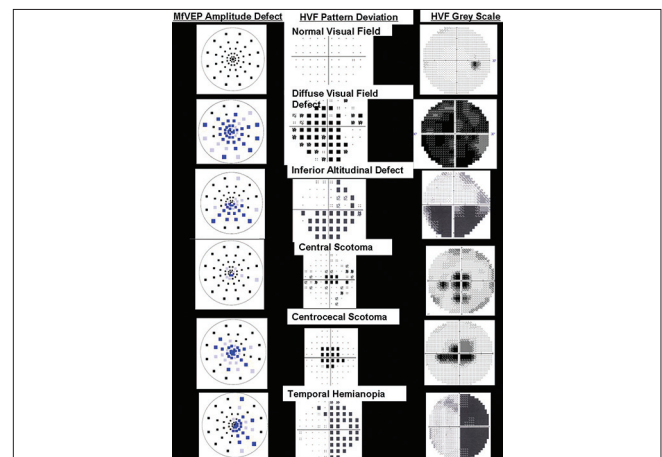


Figure 2: Monocular amplitude defect as per optic neuritis treatment trial visual field classification

Statistical analysis

The statistical analysis was performed using PASW (Predictive Analytics Software) Statistics version 18.0 for windows. Right eye of all the controls was used for analysis. Right eye of optic nerve disorders in cases of bilateral disease were recruited. Mann-Whitney U test was performed to assess the difference in mfVEP parameters between optic nerve disorders and age matched controls.

Results

A sample of 119 eyes (48 eyes of optic nerve disorders and 71 eyes of controls) was enrolled for the study during the period of April 2009-March 2010 after fulfilling the inclusion criteria. The demographic details of the cases with optic nerve disorders and age matched controls are summarized in Table 1. The median age of the three subgroups showed a significant difference (Kruskal-Wallis test, $P = 0.013$) and, hence, different sets of age matched controls were used for each optic nerve disorder.

Subjects with optic nerve disorder presented with unilateral or bilateral progressive visual acuity loss, color vision impairment and visual field defect. The median logMAR (logarithm of minimum angle of resolution) visual acuity in subjects with optic nerve disorders was 0.20 (interquartile range, 0.025-1.00. Subjects who were unable to identify the plates in color vision Ishihara Pseudisochromatic chart was observed in 45 out of 48 eyes. The median of HVF

MD (mean deviation) was -14.73 dB, $P < 0.05\%$ (interquartile range, -8.82 to -19.56 dB).

Comparison of multifocal VEP findings in optic nerve disorders

Mean mfVEP responses

The median amplitude (log SNR) and relative latency (ms) in optic neuritis, ischemic optic neuropathy and compressive optic neuropathy compared with age matched control are summarized in Table 2. This shows that mfVEP amplitude was significantly reduced among optic nerve disorder group. The relative latency was significantly prolonged in optic neuritis and compressive optic neuropathy group. However, the comparison of log SNR amplitude reduction and relative latency delay between optic neuritis and ischemic optic neuropathy, between optic neuritis and compressive optic neuropathy, and between ischemic optic neuropathy and compressive optic neuropathy was not significant as summarized in Table 3.

Topographical probability map

Amplitude probability plot

The size and the pattern of the amplitude defects in optic nerve disorders are shown in Table 4 and Fig. 2.

Latency probability plot

The number of contiguous abnormal latency points in probability was counted for optic nerve disorder group. The latency probability did not follow any specific pattern of prolonged latency. In the case of optic neuritis and compressive optic neuropathy, 3 out of 9 measurable points and 3 out of 11 points (SNR >0.23) showed delayed latency respectively. One out of six measurable locations showed a delayed latency in ischemic optic neuropathy eyes.

Discussion

The value of mfVEP parameters in objective assessment of the visual function in optic nerve disorder is illustrated in this study. To the best of our knowledge, limited numbers of studies have compared the PVEP^[9,10,27-30] and mfVEP findings^[31-34] in optic nerve disorders. In our study, the mean amplitude (log SNR) of 60 mfVEP responses was significantly reduced in all optic nerve disorder groups compared to the age matched controls. Although the mfVEP latency was prolonged in all optic nerve disorder groups, the optic neuritis and compressive optic neuropathy group showed a more prolonged latency than ischemic optic neuropathy group. However, comparison of

Table 1: Demographic details of the subjects

Group	No. of eyes	No. of males: females	Median (Interquartile range) Age (Years)
Optic nerve disorders	48	25:23	47.0 (28.0-54.0)
Control	71	34:37	43.0 (30.0-52.0)
ON	21	7:14	28.0 (23.0-44.5)
Control for ON	30	7:23	28.5 (22.0 -37.0)
ION	14	11:13	52.5 (47.8-58.0)
Control for ION	18	15:3	51.0 (45.8-54.3)
CON	13	7:6	52.0 (35.3-59.0)
Control for CON	23	12:11	49.0 (36.0-55.0)

ON: Optic neuritis, ION: Ischemic optic neuropathy, CON: Compressive optic neuropathy

Table 2: Multifocal VEP amplitudes among optic nerve disorders and controls

Optic nerve disorders	mfVEP amplitude (log SNR)		mfVEP relative latency (ms)	
	Median (Interquartile range)	Mann-Whitney U test, P value	Median (Interquartile range)	Mann-Whitney U test, P value
ON	0.17 (0.13-0.33)	<0.001	10.53 (2.62-15.50)	<0.001
Control for ON	0.50 (0.44-0.56)		0.72 (-2.06-2.94)	
ION	0.14 (0.12-0.21)	<0.001	2.06 (-4.09-13.02)	0.569
Control for ION	0.42 (0.37-0.48)		3.66 (2.37-7.91)	
CON	0.21 (0.14-0.30)	<0.001	5.73 (2.67-14.14)	0.007
Control for CON	0.46 (0.37-0.50)		1.69 (0.36-3.84)	

VEP: Visual evoked potential, mfVEP: Multifocal visual evoked potential, ON: Optic neuritis, ION: Ischemic optic neuropathy, CON: Compressive optic neuropathy

mfVEP amplitude (log SNR) and relative latency between optic neuritis and ischemic optic neuropathy, ischemic optic neuropathy and compressive optic neuropathy, compressive optic neuropathy and optic neuritis was not significant in our study. This could be due to significant difference in median age of three groups in our study.^[21]

These results were similar to PVEP findings in optic neuritis and ischemic optic neuropathy subjects reported by Wilson,^[27] Thompson *et al.*,^[28] Veselinovic and Duric,^[29] Mukartihal *et al.*,^[30] Holder GE,^[9] and Holder *et al.*,^[10] They reported that the extent of delayed latency in PVEP responses for ischemic optic neuropathy was significantly small compared to optic neuritis. Our findings of amplitude reduction and delayed latency are consistent with those of Sanders *et al.*,^[35] and Wildberger *et al.*,^[36] findings on pathophysiology of disease mechanisms and its effect on VEP parameters. They have shown that amplitude reduction is because of the axonal loss in optic nerve diseases and delayed latency indicates the extent of demyelination. The edema in ON,^[28,29] ischemia in ION^[28,29] and demyelination in CON^[36] explains the noted amplitude reduction in mfVEP responses. The demyelination of axons in ON and CON eyes contributes to the delayed latency.^[28,29,36]

Table 3: Comparison of mfVEP log SNR and relative latency among optic nerve disorders

Optic nerve disorders	mfVEP amplitude (log SNR) Mann-Whitney U test, P value	mfVEP relative latency (ms) Mann-Whitney U test, P value
Optic neuritis	0.3044	0.1727
Ischemic optic neuropathy		
Optic neuritis	0.4895	0.4895
Compressive optic neuropathy		
Compressive optic neuropathy	0.0850	0.3255
Ischemic optic neuropathy		

mfVEP: Multifocal visual evoked potential

Several studies compared the mfVEP amplitude and HVF thresholds and reported that amplitude probability plots are similar to HVF total deviation map.^[34,37-41] In this study, we have analyzed the size of the amplitude reduction and its pattern in mfVEP topographical amplitude deviation map among optic nerve disorders using the visual field profile classification of ONTT as shown in Fig. 1. In our analysis in optic neuritis group, a diffuse pattern of amplitude loss was noted in 10/21 (47.62%) eyes and central or centrocecal defect in 10/21 (47.62%) of the optic neuritis eyes. Rinnaldussi *et al.*,^[42] performed PVEP stimulations of central, temporal and nasal regions and concluded that central fibers were more affected by demyelination in optic neuritis than peripheral fibers. Pakrou *et al.*,^[43] reported that only 8.3% of the 16 optic neuritis subjects showed a central or centrocecal mfVEP defect using cluster criteria. Laron *et al.*,^[37] analyzed the mfVEP amplitude and latency abnormality using cluster criteria in MS-ON eyes. They reported that although abnormal sectors were diffusely distributed, the central 10° was more affected than periphery.

In this study, we have illustrated that the mfVEP amplitude can be used to objectively assess the topography of the visual field defect similar to that obtained in subjective HVF testing. Several studies in optic neuritis,^[37-41,43-46] compressive optic neuropathy,^[34,47] and ischemic optic neuropathy^[48] have reported that mfVEP amplitude provides a valuable diagnostic tool in detecting visual field loss. Distribution of delayed latencies in latency probability plot did not follow any specific pattern, although delayed latencies were noted in optic neuritis and compressive optic neuropathy. This could be due to the acute stage of disease which showed large regions with SNR <0.23. Hence latencies could not be measured in those locations in our study.

We emphasize that pattern of amplitude defect in combination with delayed latency can be used to differentiate optic neuritis and compressive optic neuropathy from ischemic optic neuropathy. These results were consistent with the findings of Gih *et al.*,^[31] Odel *et al.*,^[32] and Wenick *et al.*,^[33] They reported the findings in ION and ON subjects that delayed latency combined with abnormal points in cluster criteria can help to discriminate the ON from ION eyes. Semela *et al.*,^[34] also reported delayed latency in series of meningioma subjects and this delayed latency was similar to that seen in studies of optic neuritis subjects and larger than ischemic optic neuropathy and glaucoma subjects.

Table 4: Pattern of amplitude defect among optic nerve disorder eyes

	Size of amplitude defect (Out of 60 sectors) Median (Interquartile range)	Pattern of amplitude defect (Number of eyes)
Control	3 (1-5)	Normal
Optic neuritis	43 (34-45)	Diffuse field loss (10/21) Central scotoma (6/21) Centrocecal scotoma (4/21) Superior defect (1/21)
Ischemic optic neuropathy	45 (42-49)	Inferior altitudinal defect (9/14) Superior altitudinal defect (1/14) Diffuse field loss (2/14)
Compressive optic neuropathy - pituitary adenoma	33 (25-36)	Temporal hemianopia (7/7)
Compressive optic neuropathy - Meningioma	44 (42-47)	Inferior altitudinal defect (4/6) Diffuse field loss (1/6) Central Scotoma (1/6)

The two limitations of the study are the findings have been reported in small sample size and the reproducibility of the mfVEP parameters has not been assessed.

In conclusion, optic neuritis, ischemic optic neuropathy and compressive optic neuropathy cause the reduction in mfVEP amplitude. The extent of delayed latency noted in ischemic optic neuropathy was significantly lesser compared to subjects with optic neuritis and compressive optic neuropathy. Thus, mfVEP amplitudes can be used to objectively assess the topography of the visual field defect. However, it is not possible to differentiate the ischemic optic neuropathy, optic neuritis, and compressive optic neuropathy based on our study. The limitation of the study is point to point comparison of mfVEP amplitude with HVF is not analyzed. There is a scope of future studies on mfVEP responses in differentiating the subgroups of optic nerve disorders.

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