Saudi Journal of Ophthalmology (2012) 26, 91–99

Original Article

Electrophysiological study of myopia

Mona Abdel Kader, MD *

Abstract

Purpose: To investigate the characteristics of retinal function in myopia using full-field electroretinogram (ERG) and multifocal ERG (MF-ERG) and to determine the correlation among MF-ERG, ocular axis length, retinal thickness and degree of myopia.
Methods: Twenty emmetropes (20) and sixty-eight myopes (68) underwent manifest refraction, A- and B-scan, fundus examination, fluorescein angiography (FA), optical coherence tomography (OCT), full field ERG and MF-ERG. The amplitudes and implicit times of ERG were determined. The results were further analyzed by comparing ocular axis length, refraction, retinal thickness, and macular function detected by ERG parameters.
Results: There was a significant difference in implicit times of MF-ERG of an emmetrope and a moderate and high myopia whereas implicit times of mild myopia patients and emmetropes were similar. There was a statistically significant difference in amplitude densities of first positive peak of MF-ERG P_1 wave between an emmetrope and a moderate and high myopia. In central ring and four quadrants, amplitude densities showed negative correlation to ocular axis length and diopter of myopia. There was no statistically significant difference between the average retinal thickness in emmetropic and physiological myopic eyes (low, medium, high), but there was significant difference between physiological and pathological myopia.
Conclusion: Decreased foveal function as determined by MF-ERG is associated with high degree of myopia. Retinal function impairment is correlated with increase in the diopter of myopia, decrease of corrected visual acuity (VA), elongation of ocular axis and increased macular degeneration.

Keywords: ERG, Myopia, OCT

doi:10.1016/j.sjopt.2011.08.002

Introduction

Myopia is a public health concern in many parts of the world where the prevalence of myopia has been reported to be as high as 80%.[1,2] Although myopia can be easily managed with an appropriate optical correction, it is a risk factor for a number of retinal pathologies, especially in high myopia, and may cause permanent visual impairment.[3]

Myopia occurs when the axial length of the eye is too long for its optical power, and the increased axial length is the principal anatomical feature that differentiates myopia from emmetropia.[4]

The axial elongation that accompanies myopia has been reported to produce retinal stretching,[5] thinning,[6] reduced retinal cell density and enlarged photoreceptor inner segments.[7] Such anatomical changes may result in impaired retinal function and ultimately alter the visual performance.[8,9]

Eyes with pathological myopia which is characterized by degenerative changes in the posterior segment have been shown to have thinner retina at the posterior pole and the periphery.[10] However results for biometry of retinal thickness in healthy myopic eyes are still controversial.[11,12] Optical coherence tomography (OCT) is an imaging technology with ophthalmologic applications based on the principle of laser

Received 20 March 2011; received in revised form 26 July 2011; accepted 7 August 2011; available online 16 August 2011.

Mansoura Ophthalmic Center, Faculty of Medicine, Mansoura University, Mansoura, Egypt

* Tel.: +20 50 2216440; fax: +20 50 2256104.
e-mail address: monaabdelkader78@yahoo.com

الجمعية السعودية لطب العيون
SAUDI OPHTHALMOLOGICAL SOCIETY

Peer review under responsibility of Saudi Ophthalmological Society, King Saud University

Production and hosting by Elsevier
Access this article online: www.saudiophthaljournal.com
www.sciencedirect.com

interferometry.[13] Its high depth resolution (10 nm) makes it possible to measure retinal thickness more accurately.[14] Moreover, measurements do not depend on the axial length or refraction.[15]

It is unclear whether the reduction of electroretinogram (ERG) response in myopia is due to retinal degenerative changes associated with myopia or a reflection of the myopia itself. To investigate these possibilities, the multifocal electroretinogram (MF-ERG) was used to assess retinal function affected by various degrees of myopia.

The purpose of the study is to identify retinal morphology and retinal function changes in myopic eyes and to analyze the relationships among retinal function, ocular axis length, retinal thickness and degree of myopia.

Subjects and methods

This study was carried out on patients attending the outpatient clinic of Mansoura Ophthalmic Center during the period from August 2009 to July 2010. Approval from the Human Subjects Committee of the University of Mansoura was obtained, and the study adhered to the Declaration of Helsinki. Informed consents were also obtained from all participating subjects after they were given an explanation of the study.

Eighty-eight subjects were included. Based on their refraction and retinal pathology, subjects were divided into groups of emmetropes, physiological myopia and pathological myopia. Physiological Myopia subjects were further subdivided into 3 sub-groups: low (mild) myopia with myopia (−0.5D to −3.00D), medium myopia (−3.25D to −6.00D) and high myopia greater than (−6.25D) with normal fundus (absence of myopic changes).

All subjects underwent complete ophthalmological examination including: refraction using (Canon auto-refractor), best corrected visual acuity (BCVA), slit lamp examination, fundus examination using direct, indirect ophthalmoscopy, and Goldman 3-mirror lens, ocular tension measurement using applanation tonometry, A- and B-scan, ultrasound echography (US), OCT, FA, ERG. Physiological myopia was diagnosed in subjects with myopia more than −0.25D with normal and tigroid or tessellated fundus. The optic discs varied from normal to just myopic crescent. Other signs of myopic retinal degeneration were absent in this group (as confirmed by FA, absence of any degeneration or abnormalities, OCT, absence of degenerative changes or abnormalities, and US regular ocular contour).

Pathological myopia was diagnosed as subjects with high myopia more than (−6.00D) with signs of myopic retinal degeneration (posterior staphyloma, central or peripheral degeneration). There were no retinoschsis (retinal splitting) in any patient with pathological myopia as diagnosed clinically and confirmed by OCT.

All subjects had cylindrical corrections of less than 1.00D. Subjects with glaucoma, diabetes, strabismus, hypertension, abnormal ocular media, and history of current or past photosensitive epilepsy, retinoschsis or inherent retinal pathology were excluded from the study.

Axial length measurement

The axial length of both eyes of each subject was measured using A- and B-scan ultrasonography (Sony Corporation, Kitashinagawa, Shinagawa, Ku-Tokyo, Japan).

Prior to measurement, the cornea was anesthetized with one drop of topical 0.4% benoxinate HCl. Ten readings were taken to derive an average value. The standard deviation was below 0.1 mm for each subject. The subjects underwent US B-scan to assess the posterior pole contour of the eye and measure the axial vitreous length supplementary to the customary axial A-scan.

Table 1. Number, age and sex among groups.

Group	Number of subjects	Number of eyes	Sex		Age (years)
			Female	Male	
Emmetrope	20	40	8	12	19–39
Physiologic myopia	44	88	24	20	20–41
Mild	10	20	6	4	22–39
Moderate	14	28	8	6	21–41
High	20	40	10	10	20–40
Pathological myopia	24	40	12	12	18–40
Posterior staphyloma	7	10	4	3	
Retinal degeneration	24	40	12	12	

Table 2. Axial length among groups in mm.

Groups	Axial length	
	Mean ± SD	Average (*P*)
Emmetrope	21 ± 0.9	20–22 (0.001)
Physiological myopia		
Mild	21.5 ± 0.5	20–23.5 (0.002)
Medium	24.6 ± 1.1	24–26.9 (0.002)
High	28 ± 1.8	27–29 (0.001)
Pathological myopia	29 ± 2.00	28–34 (0.001)

Table 3. Errors of refraction among groups ($P = 0.005$).

Groups	Refraction	
	Range	Mean ± SD
Emmetrope	+0.25 to −0.25	−0.1 ± −0.1
Physiological myopia		
Low	−0.5 to −3.00	−1.5 ± −0.75
Medium	−3.25 to −6.00	−4.00 ± −1.00
High	−6.25 to −15.00	−8.00 ± −5.00
Pathological myopia	−7.00 to −22.00	−10.55 ± −7.1

Table 4. MF-ERG parameters among groups (all traces grouping).

Groups	MF-ERG	
	P_1 amplitude	P_1 latency
Emmetropia	45.6 ± 6.00 ($P = 0.002$)	37.6 ± 0.80 ($P = 0.009$)
Physiological myopia		
Low	47.4 ± 4.00 ($P = 0.003$)	37.4 ± 0.90 ($P = 0.009$)
Medium	42.8 ± 2.00 ($P = 0.005$)	43 ± 1.50 ($P = 0.008$)
High	31.6 ± 6.00 ($P = 0.001$)	47 ± 1.50 ($P = 0.007$)
Pathological myopia	14.4 ± 7.00 ($P = 0.001$)	54.4 ± 200 ($P = 0.007$)

Optical coherence tomography (OCT)

OCT was done using Topcon, 3-Dimensional OCT-1000, USA. For each eye, six single-line OCT scans were oriented at equally spaced angular orientations in a radial spoke pattern centered on the foveal pit with a scan length of 6 mm. The subjects were asked to gaze at an internal fixation light within the machine. The retinal thickness was calculated as the distance between the two boundaries using automatic boundary detection software. The software automatically detects the vitreoretinal junction as the inner retinal boundary and chorio retinal junction as the outer retinal boundary. The thickness in three circular areas (A, B, C) centered on the central fovea with diameter 1 mm, 3 mm, 6 mm respectively were calculated automatically by the software. The axial length is adjusted in every patient before performing the OCT scanning. The retinal thickness was measured both manually after adjusting inner & outer retinal borders & automated

Fluorescein angiography (FA)

FA was done using (Topcon Corporation, 2000, TRC, 50 Π, Japan).

Electroretinogram (ERG)

Standard full field ERG and multifocal ERG (MF-ERG) were done using Roland Consult, Brandenburg, Germany system.

Responses were recorded monocularly by using Dawson Trick-Litzkow (DTL) thread electrode which was positioned on the inferior cornea along the lid margin and fixed temporally. The pupils were dilated with tropicamide 1%. Gold-Cup reference and surface electrodes were applied to the subject's temple and forehead, respectively.

Standard ERG

After dark adaption for 20 minutes, the subject put the head on Ganzfeld stimulator, 3 steps were recorded (rod response, combined response and oscillatory potential) then light adaptation for 10 minutes then 2 steps were recorded (cone response and 30 Hz flicker response).

MF-ERG

The visual stimulus array was driven on a monitor consisting of 61-scaled hexagons. The size of the hexagons was scaled with eccentricity to elicit approximately equal amplitude responses at all locations. Each hexagon was temporally modulated between black and white according to pseudo-random binary m-Sequence with luminance of 100 cd/m^2 in white hexagons and 2 cd/m^2 in black hexagons. Subjects were optically corrected for the viewing distance (50 cm) and were asked to maintain fixation on the red fixation target at the center of stimulus matrix and refrain from blinking. Recording segments containing ERG artifacts due to blinks or small eye movement were detected and discarded.

Each session of recording took approximately four minutes to complete, a break was given after each 30 seconds of recording. If more than 3 fixation losses occurred within the 30 seconds the measurement was redone. Data from two recording sessions were obtained for each subject and averaged. For each wave form, the amplitude and implicit time of first positive peak (P_1) were determined. P_1 amplitude was measured from trough of first negative wave to the peak of the positive peak while the implicit time was measured from stimulus onset to first prominent response peak. First-order response is derived from the average retinal response to focal flash and reflects activities from the outer to middle retinal layers especially the bipolar cells.[15]

Three grouping configurations were used, all traces, rings and quadrants. All traces grouping was a single wave form grouping response from stimulus hexagons. The five rings groupings were five wave form grouping responses from five concentric rings. Ring (1) is the most central hexagons with radius of about 0.5 mm (1.7)°. Rings 2, 3, 4, 5, were responses of increasingly eccentric annuli of stimulus.

The four quadrants grouping was four-wave form grouping response from superonasal, superotemporal, inferotemporal, inferonasal. Using averaging programs, all wave form amplitudes were scaled in nv/degree^2 (density – scaled aver-

Table 5. MF-ERG amplitudes over rings.

Groups	Ring1	Ring 2	Ring3	Ring 4	Ring 5
Emmetropia ($P = 0.002$)	65 ± 10	53 ± 6	44 ± 7	35 ± 5	30 ± 4.00
Physiological myopia ($P = 0.001$)					
Low	64 ± 9.00	54 ± 6.00	52 ± 8.00	37 ± 5.00	30 ± 5 .00
Medium	50 ± 5.00	45 ± 3.00	40 ± 2.00	30 ± 4.00	25 ± 3.00
High	40 ± 6.00	39 ± 5.00	38 ± 4.00	27 ± 3.00	26 ± 5.00
Pathological myopia ($P = 0.000$)	20 ± 5.0	15 ± 7.0	16 ± 6.0	11 ± 5.00	10 ± 6.00

Table 6. MF-ERG amplitudes over quadrants.

Groups	SN	ST	IT	IN
Emmetropia	28 ± 8.00	27 ± 7.70	27.2 ± 7.00	26 ± 9.00
Physiological myopia ($P = 0.006$)				
Low	27 ± 8.00	27.1 ± 6.00	26 ± 6.00	26.6 ± 6.10
Medium	20 ± 5.00	21 ± 4.00	20 ± 3.00	19 ± 3.00
High	15 ± 2.00	16 ± 3.00	17 ± 3.00	16.6 ± 2.80
Pathological myopia	10 ± 5.00	11 ± 6.00	10.5 ± 6.00	9 ± 5.50

ST: superotemporal, IN: inferotemporal, SN: Superonasal, IT: inferotemporal

age: (degree^2) reflects the angular size of the stimulus hexagons that produced the response).

Statistical analysis

Statistical analysis of the data was conducted using the statistical packages for the social science (SPSS). Repeated measures analysis of variance (ANOVA) was performed to determine if there were differences in ERG and OCT responses between emmetropes and myopes. Spearmans Correlation Coefficient was used to calculate correlation between variables $P \leqslant 0.01$ was considered statistically significant, $r \geqslant 0.5$ was considered good correlation.

Results

The study included eighty-eight (88) subjects. Age and sex are included in Table 1. All pathological myopia subjects had chorioretinal degeneration while only (7) seven subjects (10eyes) had posterior staphyloma.

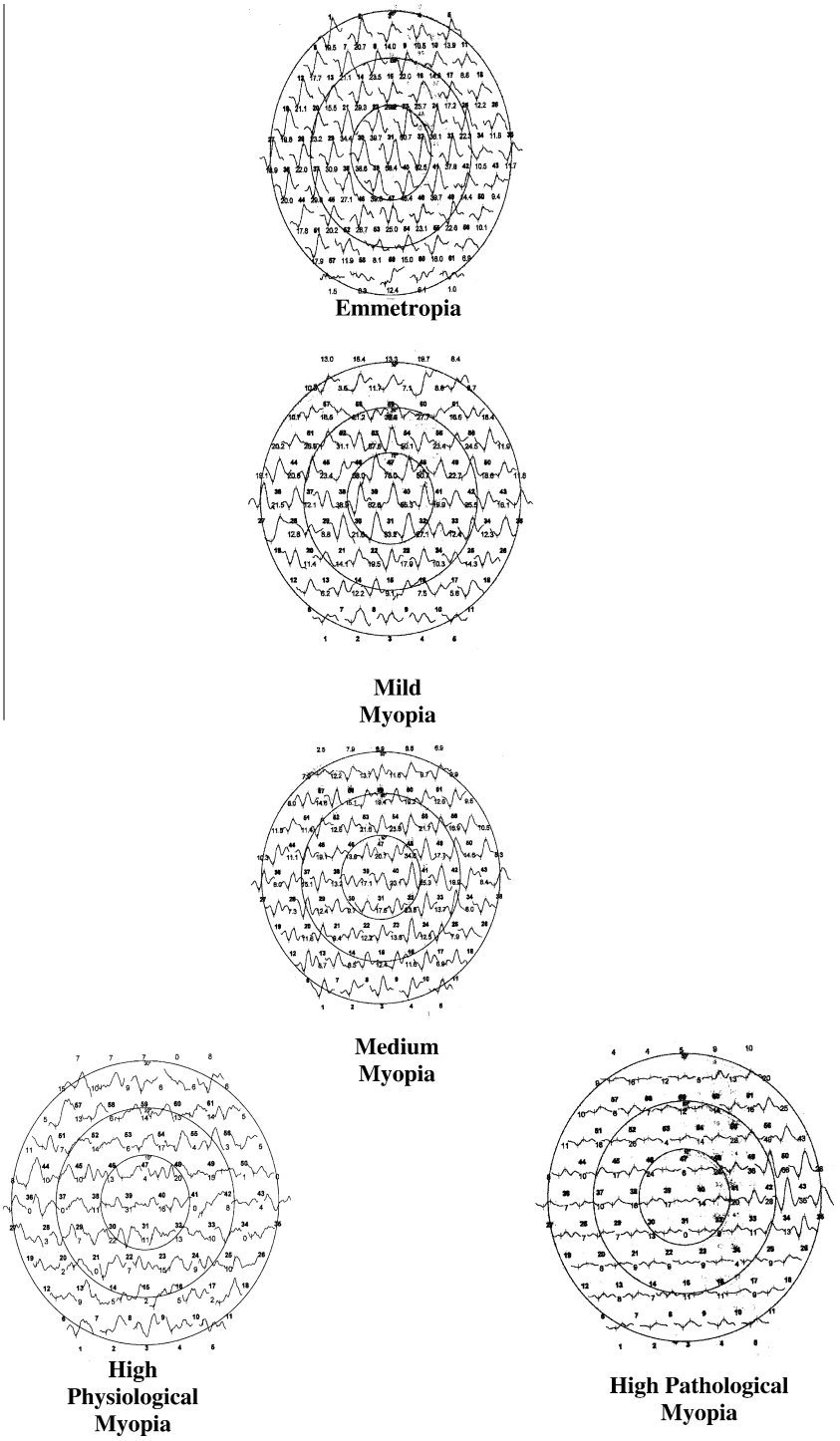

Figure 1. MF ERG trace array among groups.

The axial lengths among groups as measured by ultrasound were recorded in Table 2.

There were statistically significant differences among groups in the axial length. The axial length increases as the error of myopia increases. The refractive errors were significantly correlated with axial lengths $r = 0.95$ $P = 0.000$. Errors of refraction among groups are included in Table 3.

Full field ERG and MF-ERG were recorded. There were differences between MF-ERG parameters between emmetropes and moderate and high myopia. In addition, there were statistically significant differences between emmetropes and pathological myopia patients while there were no statistically significant difference between emmetropes and low myopia. (Tables 4–6), (Figs. 1–4).

The mean p_1 amplitudes of all trace grouping waves decreased significantly as refractive errors increased ($r = 0.70$) ($P = 0.001$).

In all areas, mean P_1 amplitudes for emmetropia and mild myopia were the largest and those of high myopia were the smallest. The mean P_1 amplitude was smaller in pathological myopia than in high physiological myopia. The difference in P_1 amplitudes was statistically significant between any two myopic groups. In the emmetropia, the more peripheral the tested areas, the lower the amplitudes. In myopia groups, this decrease was more exaggerated (the P_1 amplitudes were reduced more in high and moderate myopia) as the stimulus hexagons were located in the more peripheral area ($P = 0.007$).

In four quadrants grouping waves stimulation, the P_1 latencies and amplitudes were almost equal in the four quadrants.

The P_1 latencies of all traces group waves (ATG), ring group waves (RGW) were significantly different among groups (Table 7–9). P_1 latencies were significantly correlated with refractive errors ($r = 0.55$, $P = 0.005$).

There were significant correlation between axial length and implicit time ($R = 0.5$, $P = 0.001$) and between amplitudes measures and axial length ($R = 0.49$, $P = 0.01$). (The in-

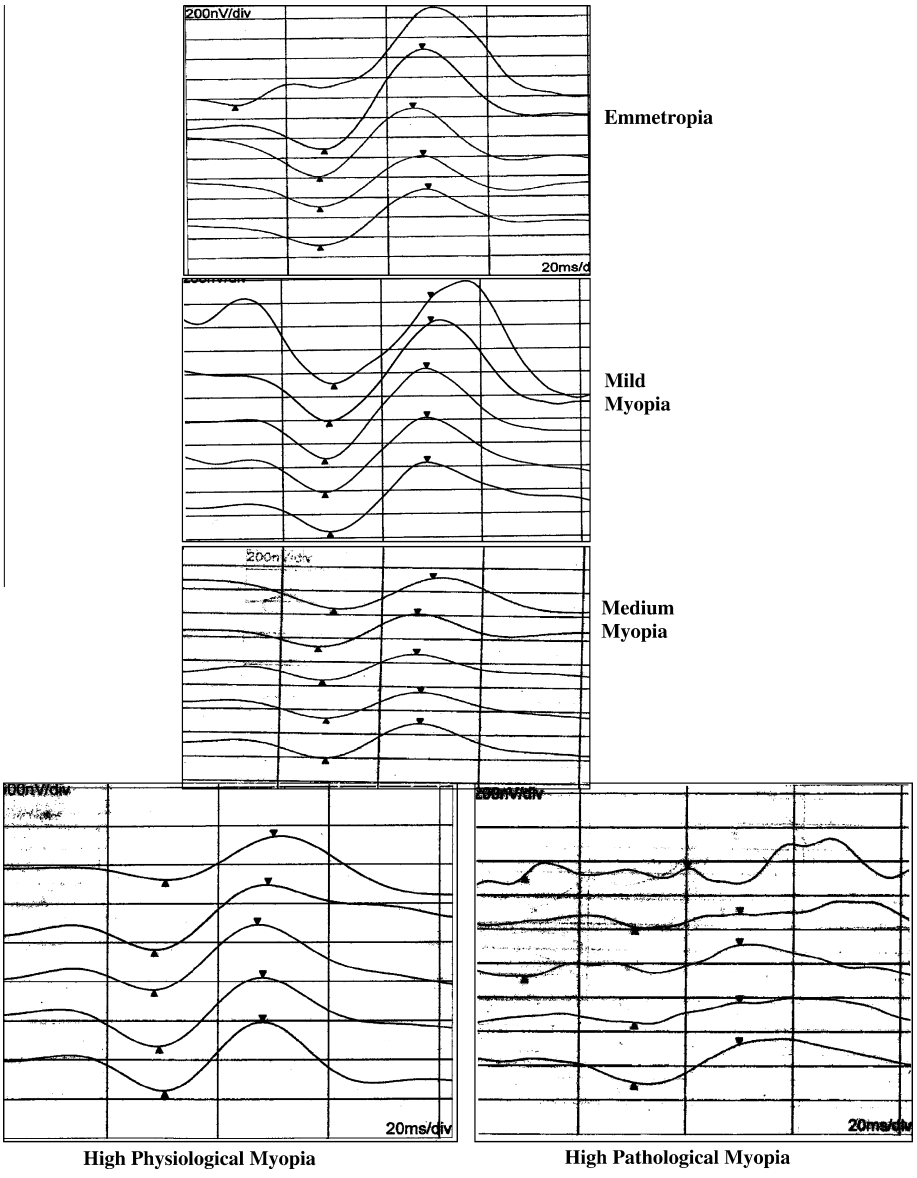

Figure 2. MF-ERG over rings among groups.

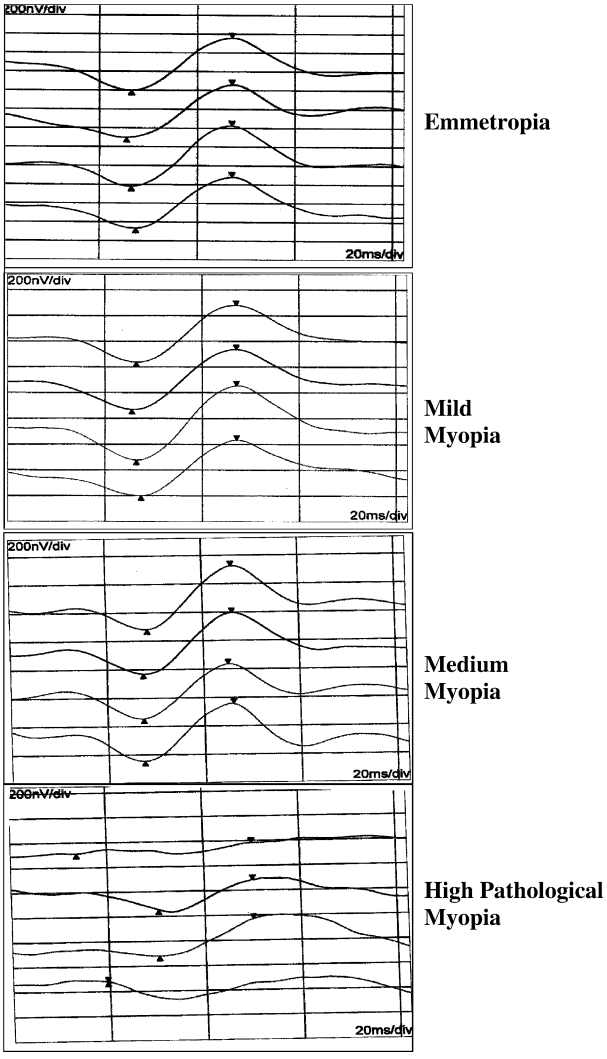

Figure 3. MF-ERG over quadrants among groups.

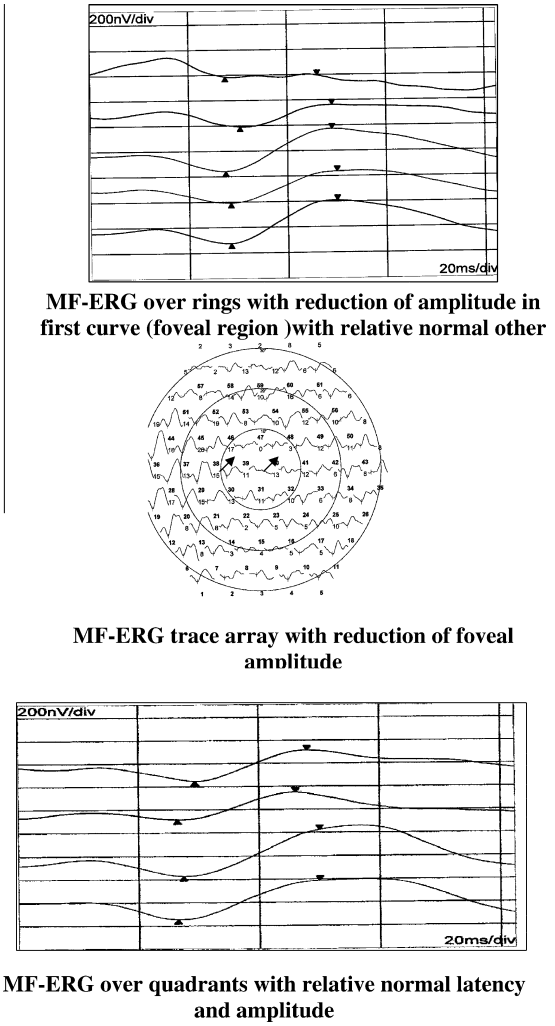

Figure 4. High myopia with central myopic degeneration.

crease in axial lengths is accompanied with decrease in amplitudes and increase in latencies.)

Full field ERG responses were significantly different between emmetropia and high myopia ($P = 0.001$), while no significant difference was found between emmetropia and mild and moderate myopia. ($P = 0.2$) (Table 10; Fig. 5). The decrease of b-wave amplitude was proportional to the axial length.

As regards the retinal thickness, there was no significant difference in emmetropia, low myopia, moderate myopia, high myopia in three circular areas ($P = 0.1$). There was no significant change in the retinal thickness with increasing axial length of the eye. While there was significant decrease in retinal thickness in pathological myopia (Table 11), there was no statistical significant correlation between MF-ERG amplitude and implicit time and retinal thickness in emmetropia and physiological myopia (in mild myopia, $R = 0.21$, $P = 0.3$, $R = 0.3$, $P = 0.9$, respectively; in moderate myopia $R = 0.15$, $P = 0.4$, $R = 0.22$, $P = 0.87$, respectively; in high myopia $R = 0.4$, $P = 0.01$, $R = 0.44$, $P = 0.05$, respectively) respectively while there was significant positive correlation between MF-ERG amplitude and retinal thickness and negative correlation between MF-ERG latencies and retinal thickness in pathological myopia ($r = 0.51$, $P = 0.001$, $r = 0.58$, $P = 0.001$ respectively).

Discussion

In high myopic eyes, many studies have reported decline of visual functions such as corrected visual acuity,[16] visual fields,[17] color vision,[18] light sense,[18] and contrast sensitivity.[19] Several papers reporting on conventional ERG changes in myopia have been published since Karpe 's report in 1945,[20] showing reduction of b-wave amplitudes in myopic eyes that related to myopic degree.[21,22] In addition, only few studies have described patients with myopia and good corrected visual acuity associated with tessellated fundus.[23]

In this study, there was a decrease in the amplitude of b-wave and a delay in the latency of standard ERG in high myopia. There was significant difference of ERG values between high myopia and emmetropia while there was no significant difference between mild and medium myopia and emmetropia. There were marked reductions of amplitudes of b-waves of ERG in pathological myopia.

Westall et al.[22] and Papilin,[24] found a reduction of ERG amplitude with the increase of axial length.

Table 7. MF-ERG latencies over rings.

Groups	Ring 1	Ring 2	Ring 3	Ring 4	Ring 5
Emmetropia ($P = 0.001$)	35 ± 0.9	36 ± 1	37 ± 0.5	40 ± 0.9	40 ± 0.8
Physiological myopia($P = 0.000$)					
Low	36 ± 0.7	35 ± 1	37 ± 0.9	39 ± 0.8	40 ± 1
Medium	40 ± 3	43 ± 1	42 ± 1.0	45 ± 1.2	45 ± 10
High	48 ± 4	46 ± 2	44 ± 2.1	48 ± 1	49 ± 10
Pathological myopia ($P = 0.004$)	55 ± 3	52 ± 2	53 ± 1.5	56 ± 3	56 ± 2

Table 8. MF-ERG latencies over quadrants ($P = 0.005$).

Groups	P_1 latencies			
	SN	ST	IT	IN
Emmetropia	30 ± 3.00	29 ± 4.00	31 ± 4.00	30 ± 5.00
Physiological myopia ($P = 0.003$)				
Low	31.3 ± 3.00	30.4 ± 3.00	29 ± 5.00	29.5 ± 5.00
Medium	35 ± 5.00	34 ± 4.00	33 ± 5.00	35 ± 4.00
High	40 ± 3.4	40.4 ± 4.5	41.4 ± 5.00	41.2 ± 3.00
Pathological myopia	50 ± 5.00	51 ± 6.00	50.5 ± 5.6	51 ± 6.6

Table 9. MF-ERG parameters between high physiological myopia and pathological myopia.

ERG	High physiological'	Pathological myopia
Amplitudes over rings (0.005)		
Ring 1	40 ± 6.0	20 ± 5.0
Ring 2	39 ± 5.0	15 ± 7.0
Ring 3	38 ± 4.0	16 ± 6.0
Ring 4	27 ± 3.0	11 ± 5.0
Ring 5	26 ± 5.0	10 ± 6.0
Amplitudes over quadrants (0.008)		
Superonasal	15 ± 2.0	16 ± 5.0
Superotemporal	16 ± 3.0	11 ± 6.0
Inferotemporal	17 ± 3.0	10.5 ± 6.0
Inferonasal	16.6 ± 2.8	9 ± 5.5
Latencies over rings (0.003)		
Ring 1	48 ± 4.0	55 ± 3.0
Ring 2	46 ± 2.0	52 ± 2.0
Ring 3	44 ± 2.0	53 ± 1.9
Ring 4	48 ± 1.5	56 ± 3.0
Ring 5	49 ± 10	57 ± 2.0
Latencies over quadrants (0.007)		
Superonasal	40 ± 3.5	50 ± 5.0
Superotemporal	40 ± 4.5	51 ± 6.0
Inferotemperol	41 ± 5.0	50.5 ± 5.6
Inferonasal	41 ± 4.0	51 ± 6.9

Chen et al. found a delay in MF-ERG P_1 implicit time in myopia, while the amplitudes of P_1 in myopia were the same as in emmetropia. There were no difference in response amplitude. There was no statistically significant correlation between axial length and implicit time, or between axial length and amplitudes measures.[25] The possible explanation for lack of difference in p_1 amplitude is postulate to be the greater degree of inter-subject variability in amplitudes.[26]

The cause of small response delay with approximately normal amplitude may be due to altered synaptic transmission or damage to inner plexiform layers.[15]

Kawabata and Adachi-Usami reported significantly longer response latencies in medium and high myopia than emmetropes with amplitude reduction.[7]

Luu et al. found delay in implicit time and reduction in amplitude in P_1. The amplitudes were significantly correlated with the severity of myopia in adult subjects.[26]

In this study, there was reduction of P_1 amplitude and prolongation of implicit times of P_1 of MF-ERG in medium and high myopia without degeneration. The reduced amplitudes decreased more as the stimulus hexagon were located in the more peripheral areas.

A number of factors for this reduction have been suggested, namely optical, electrical and retinal factors. In relation to optical factors, this reduction may be related to reduced image size and decreased retinal illumination as a result of axial elongation of the eye. Second, in relation to the electrical factors, is the increased distance between the electrical source (the retina) and the electrode. Finally, decreased retinal photoreceptor density,[27] morphological changes in the photoreceptor outer segment,[28] and photoreceptor dysfunction,[29] have been considered as retinal factors.

The causes for prolongation of implicit times may be the differences in the kinetics of synaptic transfer from photoreceptors to ON and OFF pathways of bipolar cells.[30] Other possible causes are modification in dopaminergic system, dopamine levels are reduced in form deprivation myopia,[31] and dopamine agonist have been shown to inhibit myopia.[32]

Dopamine is also involved in the reorganization of receptive field properties that accompany changes in retinal illluminance, it modifies the spatial and dynamic of the ganglion cell response.[33]

In the cases of pathological myopia, in this study, there was more reduction in p_1 amplitude and prolongation in implicit times than physiological myopia. The degree of P_1 amplitudes reduction were proportional with degree of degeneration, the more the retinal degeneration, the more the reduction of amplitude and the more prolongation of implicit limes.

Similarly, Tu et al. found that visual function loss in pathologic myopia is correlated with the increase of diopter of myopia, decrease of corrected visual acuity, increased macular disease and decreased macular function and extensive elongation of ocular axis in pathologic myopia.[34]

Retinal thickness varied greatly from region to region in the retina.[35] So, the site and size of the measured retina area must be constant. Highly myopic eyes hypothetically have thinner retinas than do emmetropic eyes. In fact, increased axial length in myopic eyes has been shown to increase the

Table 10. Standard (full field) ERG among groups ($P = 0.008$).

ERG	Emmetropia	Mild myopia	Moderate myopia	High myopia	Pathological myopia
Scotopic rod response					
b-Wave amplitude	88 ± 9	85 ± 8	89 ± 10	69 ± 5	40 ± 20
b-Wave latency	70 ± 5	72 ± 6	71 ± 7	85 ± 4	92 ± 8
Photopic cone response					
b-Wave amplitude	60 ± 10	59 ± 8	58 ± 10	49 ± 3	30 ± 15
b-Wave latency	20 ± 3	21 ± 4	20.2 ± 4.1	26 ± 2	43 ± 10
Combined response					
b-Wave amplitude	220 ± 20	212 ± 15	216 ± 14	170 ± 10	120 ± 3
b-Wave latency	40 ± 6	41 ± 5	42 ± 4	50 ± 3	60 ± 5
b/a Ratio	1.1 ± 0.1	1.2 ± 0.2	1.22 ± 0.2	1.5 ± 0.4	2.5 ± 0.5
Oscillatory potential					
Latency	20 ± 2	21 ± 2.3	21.2 ± 3	25 ± 2	30 ± 3
Amplitude	35 ± 5	32 ± 4	31 ± 5.1	25 ± 6	12 ± 5
Flicker					
Amplitude	60 ± 10	59 ± 9	58.9 ± 8.1	55 ± 6	40 ± 10
Latency	60 ± 2	60 ± 4	60.5 ± 3	66 ± 3.3	70 ± 7.1

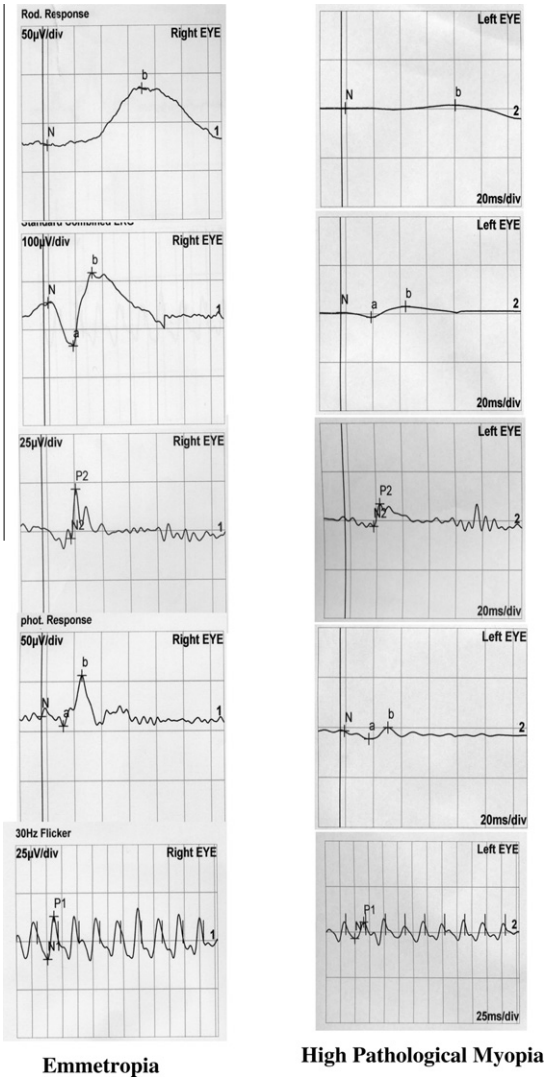

Figure 5. Full field ERG among groups.

incidence of chorioretinal atrophy in the posterior pole and chorioretinal degeneration in the peripheral fundus.[36,37]

As regards retinal thickness measured by OCT, in this study there was no difference in macular thickness between emmetropia and physiological myopia while there was a statistically significant difference between emmetropia and pathological myopia. There was a significant decrease in retinal thickness as the retinal pathology increased. There was no correlation between MF-ERG amplitude and latency and retinal thickness in physiological myopia while in pathological myopia there was correlation between MF-ERG amplitude and latency and retinal thickness.

Table 11. Average retinal thickness among groups in micron (μm) ($P = 0.001$).

Groups	Average thickness (mean ± SD) (μm)		
	Area A (1 mm)	Area B (3 mm)	Area C (6 mm)
Emmetropia	230 ± 9.50	280 ± 26	245 ± 15
Physiological myopia			
Low	232 ± 8.20	277 ± 22.00	240 ± 12.00
Medium	229 ± 10.10	275 ± 20.00	241 ± 13.00
High	230 ± 10.30	278 ± 21.00	243 ± 10.00
Pathological myopia	170 ± 30.00	240 ± 40.00	200 ± 50.00

Similarly, Wakitani et al. reported that there was no significant difference among the average thicknesses in emmetropia and low myopia, medium myopia and high myopia.[38]

While, Wolsley et al. found that there was retinal thinning in moderate and high myopia.[39]

In summary, there was moderate reduction in amplitude and prolongation of implicit times of MF-ERG in moderate and high myopia with normal retinal thickness while in pathological myopia, there was marked reduction in the amplitude and prolongation of implicit times ERG with the reduction of retinal thickness.

Acknowledgments

Financial support: Mansoura University Ophthalmic Center. This study was approved by the Human Subjects Committee of the University of Mansoura, and adhered to the Declaration of Helsinki. A written informed consent was obtained from all participants.

Reference

1. Lin LL, Shin YF, Tsoi CB. Epidemiologic study of ocular refraction among school children in Taiwan in 1995. *Invest Ophthalmol Vis Sci* 1999;**76**:275–81.
2. Wu HM, Seet B, Yap EP. Does education explain ethnic differences in myopia prevalence? A population based study of young adult males in Singapore. *Invest Ophthalmol Vis Sci* 2001;**78**:234–9.
3. Gozum N, Cakir M, Sezen F. Relationship between retinal lesions and axial length, age and sex in high myopia. *Eur J Ophthalmol* 1997;**7**:277–82.
4. Atchison DA, Jones CE, Schmid O. Eye shape in emmetropia and myopia. *Invest Ophthalmol Vis Sci* 2004;**45**(10):3380–6.
5. Logan US, Gilmartin B, Wildsoet CF. posterior retinal contour in adult human anisomyopia. *Invest Ophthalmol Vis Sci* 2004;**45**(7):2152.
6. Wakitani Y, Sasoh M, Sugimoto M. Macular thickness measurements in healthy subjects with different axial lengths using optical coherence tomography. *Retina* 2003;**23**(2):177–82.
7. Kawabata H, Adachi- Usami E. Multifocal electroretinogram in myopia. *Invest Ophthalmol Vis Sci* 1996;**38**(13):2844–51.
8. Chui TYP, Yap MKH, chan HHL. Retinal stretching limited peripheral visual acuity in myopia. *Vis Res* 2005(5);593–5.
9. Subbaram MV, Bullimore MA. Visual acuity and the accuracy of the accomodative response. *Ophthal Physiol Optics* 2002;**22**(4):312–8.
10. Yanoff M, Fine BS. *Ocular pathology: A text and Atlas*. Philadelphia: Harper Row; 1982, 513-4.
11. Garcia –Valen Zula E, Mori M, Edward DP. Thickness of the perpapillary retina in healthy subjects with different degree of ammetrpia. *Ophthalmology* 2000;**107**:1321–7.
12. Koozekananio D, Roberts C, Katz SE. Intersession repeatability of macular thickness measurement with Humphrey 2000 OCT. *Invest Ophtholmol Vis Sci* 2000;**4**:1486–91.
13. Hee MR, Izatt JA, Swan EA. Optical coherence tomography of the human retina. *Arch Ophthalmol* 1995;**113**:322–32.
14. Hee MR, puliafito A, Wong C. Quantitative assessment of macular oedema with optical coherence tomography. *Arch Ophtholmol* 1992;**113**:1019–29.
15. Hood DC. Assessing retinal function with multifocal technique. *Prog Retin Eye Res* 2000;**19**(5):607–46.
16. Tokoro T. Corrected visual acuity in high myopia. *JPnJ Clin Ophthalmol* 1973;**27**:885–90.
17. Yamaji R, Yamasowa R, Yamaji S. The quantitative perimetry of high myopia. *Folia Ophthalmol Jpn* 1976;**27**:633–44.
18. Curtin BJ. *The myopia*. Philadelphia: Harper & Raw; 1985, 353-7.
19. Collins JW, Carney LC. Visual performance in high myopia. *Curr Eye Res* 1990;**9**:217–23.
20. Karpe A. Basis of clinical electro retinography. *Acta Ophthalmol* 1945;24–73.
21. Kawabata H, Adachi-Usami E. Electrophysiological changes of photopic function in myopia. In: Tokora T, editor. Proceeding of the sixth international Conference on myopia, Hakone, 1996; Myopia update. Springer-verlag: Tokyo, 1997: 388–93.
22. Westall CA, Dhaliwal HS, Panton CM. Values of electroretingram responses according to axial length. *Doc Ophthalmol* 2001;**102**:112–30.
23. Ishikawa M, Miyake Y, Shiroyama T. Focal macular electro retinogram in high myopia. *J Jpn Ophthalmol Soc* 1993;**97**:881–7.
24. Papilin O. Influence of axial length of eye on the size of the b-potential in the clinical single- flash electroretinogram. *Acta Ophthalmol* 1999;199.
25. Chen JC, Brown B, Schmid KL. Delayed MF-ERC responses in Myopia. *Vis Res* 2006;**46**:1221–9.
26. Luu CD, Lau AM, Lee SY. Multifocal electroretinogram in adults and children with myopia. *Arch Ophtholmol* 2006;**124**:328–34.
27. Beresford JA, Crewther SG, Crewther DP. Anatomical Correlates of experimentally induced myopia. *Aust NZJ Ophthalmol* 1998;**26**(supp):584–7.
28. Crewther DP. The role of photoreceptors in the Control of refractive state. *Prog Retina Eye Res* 2000;**19**:421–57.
29. Liang H, Crewther DP, Crewther SG. A role for photoreceptor outer segments in the induction of deprivation myopia. *Vis Res* 1995;**35**:1217–25.
30. Copenhagen DR. Excitation in retina: the flow, filtering and molecules of visual signaling in the glutamatergic pathways from photoreceptors to ganglion cells. In: chalupa LM, Werner JS, editors. *The visual Neurosciences I*. London: The MIT press; 2004. p. 320–33.
31. Wallman J, Winawer J. Homeostasis of eye growth and the questions of myopia. *Neuron* 2004;**43**:447–68.
32. Schmid KL, Wildsoet CF. Inhibitory effects of apo morphine and atropine and their Combination on myopia in chicks. *Optom Vis Sci* 2004;**81**(2):137–47.
33. Witkouskg P. Dopamine and retinal function. *Doc Ophthalmol* 2004;**108**:17–40.
34. Tu Y, Wei WB, Zhou D. Observation on morphology and function of macula in pathologic myopia. *Z honghua Yan Ke za zhi* 2008;**44**:794–9.
35. Mc Donnel JM. Ocular embryology and anatomy. In: Ogden TE, editor. *Retina*, vol. 1. CV Mosby: St. Louse; 1989. p. 5–16.
36. Curtin BJ, Karlin DB. Axial length measurements and fundus changes of the myopic eye. *Am J Ophthalmol* 1971;**71**:42–53.
37. Karlin DB, Curtin BJ. Peripheral chorieoretinal lesions and axial length of the myopic eye. *Am J Ophthalmol* 1976;**81**:625–35.
38. Wakitani Y, Sasoh M, Sagimoto M. Macular thickness measurements in healthy subjects with different axial lengths using optical coherence tomography. *Retina* 2003;**23**:177–82.
39. Wolsley CL, Saunders KJ, Silyetri G. Investigation of changes in myopic retina using multifocal electroretingrams, optical coherence tomography and peripheral resolution acuity. *Vis Res* 2008;**48**(14):1554–61.