Doc Ophthalmol (2016) 132:47–55
DOI 10.1007/s10633-016-9526-1

ORIGINAL RESEARCH ARTICLE

The assessment of multifocal ERG responses in school-age children with history of prematurity

Marta Michalczuk · Beata Urban · Beata Chrzanowska-Grenda · Monika Oziębło-Kupczyk · Alina Bakunowicz-Łazarczyk · Małgorzata Krętowska

Received: 6 May 2015 / Accepted: 12 January 2016 / Published online: 29 January 2016

Abstract
Purpose The authors examined macular function in preterm-born children, using multifocal ERG (mfERG). Possible alterations in P1 amplitudes, P1 amplitudes density and P1 implicit time between school-age children with history of prematurity and their peers were researched. The correlations between parameters of mfERG responses and birth weight, gestational age, macular volume and central macular thickness were verified.
Methods A group of 18 preterm-born school-age children were analyzed (mean age 10.18 ± 1.21 years). The study group was compared to the group of 15 peers born appropriate for gestational age (mean age 10.8 ± 1.52 years). The mfERG was evaluated in all children.
Results There were statistically significant differences for P1 amplitudes from ring 1 ($p = 0.0001$) and P1 amplitudes density from ring 1 ($p = 0.0001$). Calculating the correlation coefficients, we receive significant results for P1 amplitudes from ring 1 versus gestational age ($r = 0.54$; $p = 0.026$), birth weight ($r = 0.54$; $p = 0.026$) and central macular thickness ($r = -0.62$; $p = 0.008$), and for P1 amplitudes density from ring 1 versus central macular thickness ($r = -0.51$; $p = 0.034$).
Conclusions The study suggests that P1 amplitudes and P1 amplitudes density vary in preterm-born children in comparison with their peers born appropriate for gestational age, which might suggest discreet macular dysfunction. The correlation between low birth weight, early gestational age, central macular thickness and mFERG components from ring 1 might evidence that decreased bipolar cells density caused by premature birth is the result of altered development of central retina reflecting in structural anomalies of the fovea.

Keywords mfERG · Children · Prematurity

M. Michalczuk (✉) · B. Urban · B. Chrzanowska-Grenda · M. Oziębło-Kupczyk · A. Bakunowicz-Łazarczyk
Department of Paediatric Ophthalmology and Strabismus, Medical University of Białystok, Waszyngtona 17, 15-274 Białystok, Poland
e-mail: martaa.michalczuk@gmail.com

M. Krętowska
Faculty of Computer Science, Białystok University of Technology, Wiejska 45A, 15-351 Białystok, Poland

Introduction

The survival of premature born infants has increased in highly developed countries during the past 20 years [1]. Increased survival due to advances in neonatal care is accompanied by high immaturity which may result in altered function in the future [1–4]. Retinopathy of prematurity (ROP), bronchopulmonary dysplasia (BPD) and brain injury commonly occur

together or separately in extremely low birth weight (ELBW) infants [5].

Although peripheral retina changes associated with ROP are widely studied, premature birth has influence also on anatomical and functional anomalies of the central retina [1, 6, 7]. Those mentioned anomalies are the result of the altered development of the macula [8–10]. Normal fovea development entails decrease in the rod free zone of the macula (1400 μm for 26 gestational age) to the values present in the mature eye (500 μm) [11–14]. Decrease in the rod photoreceptor free zone is linked to decrease in the distance from central fovea to the cone photoreceptor nuclei and thereby bipolar cells and also closer cone–cone packing [11–15]. Following stages of normal fovea development are dependent on the cooperative formation of both retinal neural cells and retinal vasculature [13, 16]. In preterm infants, changed parafoveal vasculature plays a role in anomalous maturation of the foveal avascular zone and delayed formation of the foveal dimple [7, 15, 16]. The structural changes of the macula caused by the abnormal fovea development reflect in the increased central retinal thickness, which can be examined by means of optical coherent tomography (OCT) [6, 7]. Associations between structural changes of the central retina and gestational age or low birth weight have been observed [7].

Although several studies have described structural central retinal changes in children with history of prematurity, very few of them have examined the impact of those structural changes and birth parameters on multifocal electroretinography (mfERG) responses [17]. The examinations of Fulton et al. [17] exhibited subtle macular dysfunction in 11 children with ROP history. Implicit time increase and deficits in amplitude were found in a group of premature born children.

The present study used mfERG to examine the function of the central retina in preterm-born school-age children (Fig. 1). We hypothesized that if the actual outcomes are associated with history of prematurity, subtle macular dysfunction should be observed in the study group, in comparison with peers born appropriate for gestational age. Furthermore, we wanted to determine whether mfERG parameters correlate with birth weight, gestational age and central macular thickness.

Subjects and methods

Subjects

We examined 18 school-age children with history of prematurity. The results were compared to controls. Inclusion criterion to the study group was preterm birth. Exclusion criteria were a history of cerebral damage and eccentric fixation. We examined both eyes of each subject, but data from only right eye were included in the analysis. Ultimately, 17 right eyes were involved, while one right eye was excluded because of eccentric fixation. The retina of seven eyes had been treated with argon laser photocoagulation and one eye with cryotherapy due to stage 3 in zone III of retinopathy of prematurity. Nine eyes of prematurely born children had no history of ROP, and they had no treatment.

Fifteen school-age children born at term were enrolled in the control group. MfERG results of those children were served as the norms by the electrophysiology laboratory in the past 5 years.

The current study was performed in the Department of Paediatric Opthalmology and Strabismus, Medical University of Bialystok, Poland. The study and its testing procedures were approved by University Ethic Committee and were in accordance with the Declaration of Helsinki.

mfERG examination

A multifocal electroretinogram was performed according to ISCEV standards [18, 19]. The pupils were dilated with tropicamide 1 % and phenylephrine hydrochloride 2.5 %. Following instillation of Proxymetacaini hydrochloridum 0.5 %, the DTL electrodes were placed on both eyes. A ground electrode was placed on the forehead, and reference electrodes were located on the temporal side of both eyes, near orbital rim of each eye. The best refractive correction was used during the mfERG test. The mfERG responses were obtained using the Espion software—Diagnosys LLC—and a scaled array of 61 hexagons. The 32″ LCD wide-angle monitor with an automatic calibration was employed. Central fixation cross with central dot was used. The stimulus luminance was 800 cd/m^2. The high-pass cutoff was 10 Hz, and the low-pass cutoff was 100 Hz. The P1 amplitudes (nV), P1 amplitudes densities (nV/deg^2)

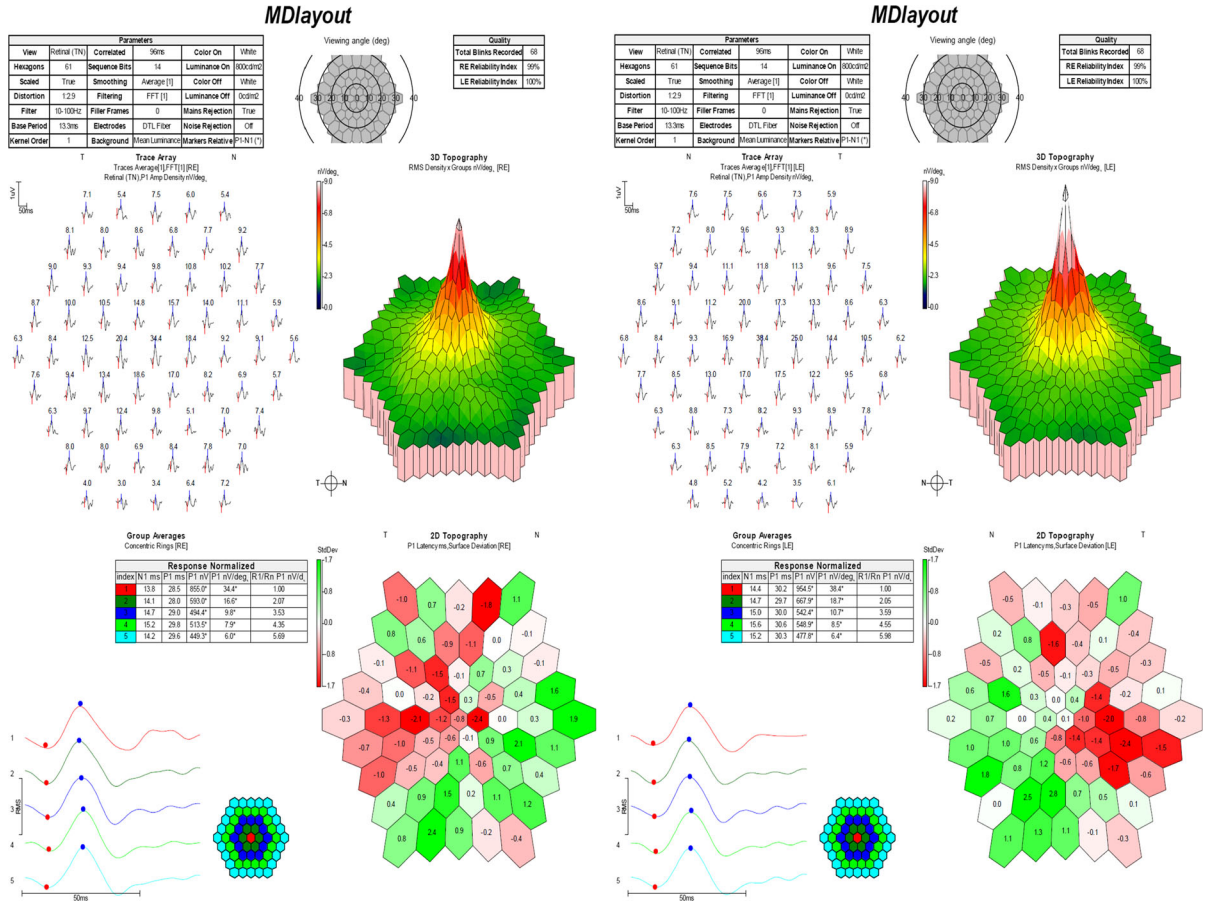

Fig. 1 Representative examples of recorded mfERG responses

and P1 implicit times (ms) of the first-order kernel were examined. The P1 amplitude was measured from the baseline to the peak of deflection. The P1 implicit time was measured from the beginning to the peak.

Optical coherence tomography (OCT)

OCT imaging was performed using a spectral domain device (Spectralis, Heidelberg Engineering, Germany). The average central macular thickness (CMT) in the central ring and total macular volume were calculated using the retinal mapping software.

Statistical analysis

The analysis was performed with the use of STATISTICA 10. Shapiro–Wilk test was applied to test the hypotheses of normal distribution of the variables. The linear relationship was verified with Pearson's correlation coefficient, and for dependent variables, the linear regression with 95 % CI was calculated. *t* test was applied to compare mean values of mfERG parameters, and 0.05 significance level was used in the analysis. To compare the mean values of 15 mfERG parameters, the Bonferroni-adjusted significance level of 0.0033 was applied.

Results

Mean age in the study group was 10.18 ± 1.21 years. Boys ($n = 7$) make up 41 % and girls ($n = 10$) 59 % of the test group. Mean gestational age was 30.4 ± 3.64 weeks. Mean birth weight was 1535.3 ± 589.82 g. The best-corrected visual acuity was equal 20/20 in all subjects.

Mean age in the control group, who had been born at full term, was 10.8 ± 1.52 years. Boys ($n = 5$) make up 33 % and girls ($n = 10$) 67 % of the control group. Visual acuity was equal 20/20 in all subjects. All control subjects were healthy, with no history of ocular disease.

The results of comparison of mean values of mfERG parameters between the study and the control group in right eyes are presented in Table 1. The P1 amplitudes and P1 amplitudes densities were smaller in the test group in comparison with controls in all five-ring retinal regions, but significant differences were observed only for ring 1 (Table 1). There were statistically significant differences for P1 amplitudes from ring 1 ($p = 0.0001$) and P1 amplitudes densities from ring 1 ($p = 0.0001$).

Calculating the correlation coefficients, we receive significant results for P1 amplitudes from ring 1 versus gestational age ($r = 0.54$; $p = 0.026$), weight ($r = 0.54$; $p = 0.026$) and CMT ($r = -0.62$; $p = 0.008$), and for P1 amplitudes density from ring 1 versus CMT ($r = -0.51$; $p = 0.034$) (Table 2). Graphical representation of these dependences together with linear regression and 95 % CI is presented in Fig. 2, 3, 4 and 5. Linear regression coefficients are enunciated in Table 3.

Comparing mean values of mfERG parameters between groups of eyes defined by different previous treatment (laser photocoagulation due to ROP in nine eyes vs no intervention in nine eyes), we observed no statistically significant differences (Table 4). The cryotherapy group was excluded from the analysis because of too small amount of cases (only one eye).

Discussion

Advances in neonatal medical care including the implementation and continuous improvement of high-tech technology during the last two decades have increased the rate of preterm births and decreased preterm mortality rates. However, preterm-born children exhibit many disabilities on short- and even long-term follow-up (especially those born before 25 weeks) [1]. Visual system in children with history of prematurity differs from visual system in children born in term, and in some of these, cortical visual impairment occurs, which is assumed to be a significant factor in vision loss in very preterm infants [20–22]. Ruberto et al. [21] noted a shorter latency of fVEP and pVEP-ss in the preterm infants at 8 months corrected age compared to those of the full-term infants. In our previous study, we also observed that P100 wave amplitudes and latencies of PVEP significantly differ between preterm-born school-age children and those born at term [22]. In the opinion of Wu

Table 1 Comparison of mean values of mfERG parameters between the study and the control group (right eyes)

mfERG parameters	Study group mean ± std ($n = 17$ eyes)	Control group mean ± std ($n = 15$ eyes)	p[a]
Ring 1: P1 implicit time (ms)	29.01 ± 1.32	29.06 ± 1.18	0.9
P1 amplitudes (nV)	792.18 ± 143.55	1101.09 ± 250.88	0.0001[#]
P1 amplitudes density (nV/deg^2)	31.42 ± 5.95	44.36 ± 10.09	0.0001[#]
Ring 2: P1 implicit time (ms)	28.32 ± 0.82	28.56 ± 1.12	0.49
P1 amplitudes (nV)	621.26 ± 121.84	782.25 ± 187.42	0.0067
P1 amplitudes density (nV/deg^2)	17.22 ± 3.51	21.75 ± 5.21	0.0066
Ring 3: P1 implicit time (ms)	28.33 ± 0.95	28.3 ± 1.18	0.94
P1 amplitudes (nV)	567.41 ± 136.41	731.23 ± 200.15	0.01
P1 amplitudes density (nV/deg^2)	11.3 ± 2.58	14.37 ± 3.94	0.013
Ring 4: P1 implicit time (ms)	28.97 ± 0.99	28.59 ± 1.28	0.35
P1 amplitudes (nV)	537.63 ± 135.73	719.82 ± 251.44	0.015
P1 amplitudes density (nV/deg^2)	8.22 ± 2.16	11 ± 3.81	0.015
Ring 5: P1 implicit time (ms)	29.09 ± 1.27	28.84 ± 1.2	0.57
P1 amplitudes (nV)	534.63 ± 151.6	691.15 ± 238.5	0.032
P1 amplitudes density (nV/deg^2)	7.16 ± 2.06	9.19 ± 3.13	0.036

[a] p value for t test

[#] Statistically significant differences at 0.0033 significance level

Table 2 Pearson's correlation coefficients between mfERG parameters (right eyes) and gestational age, birth weight, total macular volume and central macular thickness (CMT)

mfERG parameters	Variable			
	Gestational age (*r*)	Birth weight (*r*)	Total macular volume (*r*)	CMT (*r*)
Ring 1				
P1 amplitudes (nV)	**0.54 (*p* = 0.026)**	**0.54 (*p* = 0.026)**	−0.30	**−0.62 (*p* = 0.008)**
P1 amplitudes density (nV/deg2)	0.45	0.45	−0.16	**−0.51 (*p* = 0.034)**
Ring 2				
P1 amplitudes (nV)	0.39	0.48	−0.46	−0.47
P1 amplitudes density (nV/deg2)	0.33	0.42	−0.35	−0.39
Ring 3				
P1 amplitudes (nV)	0.11	0.23	−0.21	−0.31
P1 amplitudes density (nV/deg2)	0.15	0.27	−0.29	−0.37
Ring 4				
P1 amplitudes (nV)	0.17	0.36	−0.18	−0.37
P1 amplitudes density (nV/deg2)	0.15	0.33	−0.13	−0.34
Ring 5				
P1 amplitudes (nV)	−0.05	0.10	−0.20	−0.18
P1 amplitudes density (nV/deg2)	−0.06	0.09	−0.18	−0.16

The statistically important results are highlighted by bold numbers

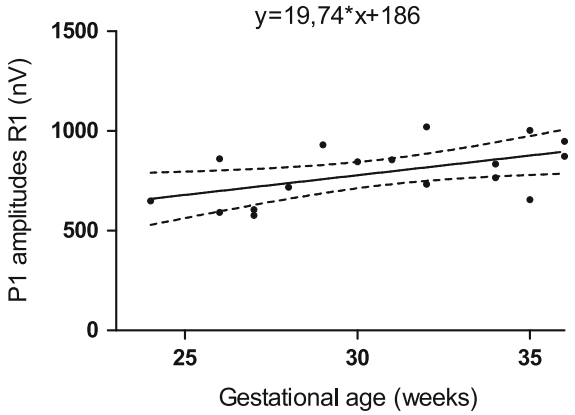

Fig. 2 Linear regression of P1 amplitudes from ring 1 and gestational age

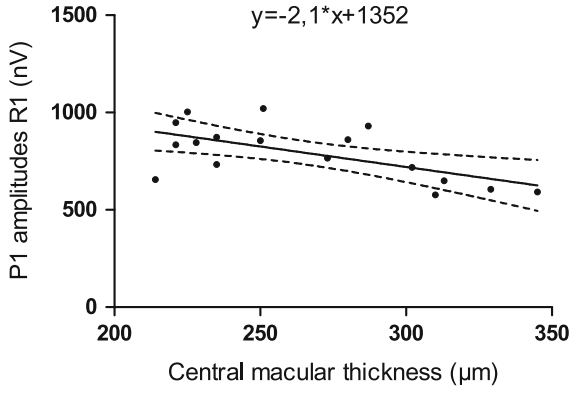

Fig. 3 Linear regression of P1 amplitudes from ring 1 and central macular thickness of the right eye

et al. [20], poorer visual acuity observed in patients with a history of ROP may be result of irregularities in the visual cortex caused by prematurity. Structural and functional changes of the premature retina are also the matter of value [6–10].

Proper development of the macula provides good vision [11–16]. Structurally widening of the pit, elongation of inner and tapering of outer segments, the tight cone packing, after birth followed by proper development of both retinal neural cells and retinal vasculature, defines normal central retina growth and thereby afford good visual acuity [11–14, 16]. In children born preterm, the structural development of the macula is faltered at the moment of the premature birth and afterward the modified sequel of development begins [7]. A consequence of altered macular development is abnormal structure of the fovea [6].

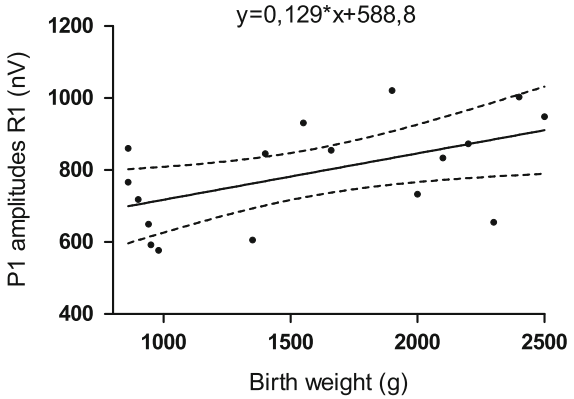

Fig. 4 Linear regression of P1 amplitudes from ring 1 and birth weight

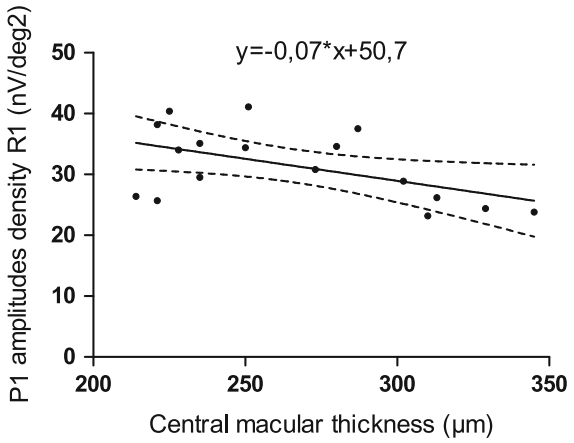

Fig. 5 Linear regression of P1 amplitudes density from ring 1 and central macular thickness of the right eyes

Nowadays a topographic measure of local retinal electrophysiological activity is possible by the use of mfERG [19]. The typical response achieved by the help of mfERG should provide two components: P1–N1 amplitude and latency, which should be measured and compared to normative data. The normal response is largest in the center where the cones and bipolar cells are densest and decrease peripherally. However, the mfERG responses in children and adults are different. One of the reasons is the fact that the distribution of cones in the central retina in infants is distinct. Candy et al. [23] showed that cone density varies from 15,000 cones/mm^2 at the fovea to 12,500 cones/mm^2 at 10° in the infant retina, compared to ~200,000 cones/mm^2 at the fovea and 11,300 cones/mm^2 at 10° in the adult retina. Therefore, P1 amplitudes of the infants' mfERG responses were significantly smaller and implicit times were significantly longer than those of adults [24]. Also the difference in mfERG between subjects with history of prematurity and children born in term has been reported [17].

In the current study on mfERG responses in children with history of prematurity, we also revealed that premature birth reflects in subtle dysfunction of the macula. The P1 amplitudes and P1 amplitudes density were smaller in the test group in comparison with controls in all five-ring retinal regions, but these components of the mfERG for ring 1 were significantly smaller between the test group and control subjects (Table 1). This might prove that developmental redistribution of bipolar cells in the central retina is changed in patients with history of prematurity. The results also suggest that the density of bipolar cells is smaller in children with history of prematurity in comparison with children born in term. However, no statistically important differences between P1 implicit time in children born preterm and children born in term imply that the summing of hyperpolarizing and depolarizing bipolar activity is not necessarily changed in children with history of prematurity. That might explain good visual acuity, whereas structural anomalies of the fovea caused by prematurity are present. These findings are party consistent with the study by Fulton et al. [17]. They observed that the amplitude of mfERG was significantly smaller, the slopes of the functions were significantly shallower,

Table 3 Linear regression coefficients together with 95 % of CI

The right eyes	Slope (95 % CI)	Intercept (95 % CI)
P1 amplitudes from ring 1 versus gestational age	19.74 (2.753; 36.73)	186.0 (−339.6; 711.6)
P1 amplitudes from ring 1 versus birth weight	0.1288 (0.0174; 0.24)	588.8 (401.4; 776.2)
P1 amplitudes from ring 1 versus central macular thickness	−2.104 (−3.57; −0.6391)	1352 (957.5; 1746)
P1 amplitudes density from ring 1 versus central macular thickness	−0.07249 (−0.1389; −0.0061)	50.69 (32.84; 68.55)

Table 4 Comparison of mean values between laser and no intervention group in right eyes

mfERG parameters	Right eyes after laser treatment ($n = 7$) mean ± std	Right eyes with no intervention ($n = 9$) mean ± std	p[a]
P1 implicit times from ring 1 (ms)	29.07 ± 1.62	29.03 ± 1.21	0.958
P1 amplitudes from ring 1 (nV)	691 ± 116.63	845.44 ± 110.96	0.017
P1 amplitudes density from ring 1 (nV/deg2)	26.69 ± 3.97	34.03 ± 4.48	0.004
P1 implicit times from ring 2 (ms)	28.4 ± 0.83	28.34 ± 0.87	0.899
P1 amplitudes from ring 2 (nV)	567.37 ± 102.17	641.04 ± 118	0.211
P1 amplitudes density from ring 2 (nV/deg2)	15.43 ± 2.85	17.97 ± 3.3	0.128
P1 implicit times from ring 3 (ms)	28.23 ± 0.99	28.5 ± 0.98	0.592
P1 amplitudes from ring 3 (nV)	514.96 ± 155.06	596.16 ± 119.9	0.256
P1 amplitudes density from ring 3 (nV/deg2)	10.41 ± 2.9	11.77 ± 2.35	0.320
P1 implicit times from ring 4 (ms)	29.03 ± 0.91	29 ± 1.13	0.957
P1 amplitudes from ring 4 (nV)	487.71 ± 132.95	567.64 ± 140.03	0.266
P1 amplitudes density from ring 4 (nV/deg2)	7.36 ± 2.15	8.74 ± 2.17	0.223
P1 implicit times from ring 5 (ms)	29.16 ± 1.42	29.13 ± 1.27	0.972
P1 amplitudes from ring 5 (nV)	514.33 ± 171.74	543.58 ± 151.4	0.723
P1 amplitudes density from ring 5 (nV/deg2)	6.87 ± 2.34	7.3 ± 2.04	0.702

[a] p value for t test

and the implicit time of each component of mfERG was longer in ROP subjects. The discrepancy between their patients with a history of ROP and control subjects was greatest for central rings (1–3) and smaller for peripheral rings (4–6). In our study, we observed significant changes in mfERG only for ring 1, maybe due to different characteristics of our study group. They concluded that subtle macular dysfunction in children with history of prematurity is the result of altered development of the neural retina [17]. The research exhibits that differences in the densities of the P1 amplitudes between the test and the control group are the result of the inter-group discrepancy of bipolar cells density in the central retina. In the test, no relationships between letter acuity nor refractive error and P1 amplitudes and latencies were reported.

Moreover, we observed significant correlations between: birth weight versus P1 amplitudes for ring 1 ($r = 0.54$; $p = 0.026$); gestational age and P1 amplitudes for ring 1 ($r = 0.54$; $p = 0.026$); and central macular thickness versus P1 amplitudes for ring 1 ($r = -0.62$; $p = 0.008$) and P1 amplitudes density for ring 1 ($r = -0.51$; $p = 0.034$) (Table 2). These statistically important correlations between birth weight, gestational age, central macular thickness and mfERG parameters for ring 1 might prove that the decreased density of bipolar cells can be the result of altered development of the central retina caused by premature birth. It is well known that macular structure is changed in preterm children. Wu et al. [20] noticed abnormal foveal contours and retention of the inner retinal layers in OCT of children with a history of ROP. Ecsedy et al. [6] examined macular structure by optical coherence tomography imaging in 40 premature children. OCT images showed that the central retinal region became larger, and the foveal depression was decreased, due to the continuity of the inner retinal layers observed under the foveal pit. In their opinion, the mechanism of these changes may be impairment of the normal centrifugal movement of foveal cone nuclei and inner retinal cells during development. In the present study, we observed negative significant correlation between P1 amplitudes and P1 amplitudes density for ring 1 and central macular thickness. At the same time, there was no correlation between mfERG parameters and total macular volume. Because bipolar cells make the main contribution to the multifocal ERG responses, these

results suggest that the difference in bipolar cell density is greatest in the central retina in preterm children. However, anomalies in fovea anatomy can be associated with excellent visual abilities, including very good visual acuity [25]. In the current study, all examined patients had visual acuity equal 20/20, even though we observed significant changes in mfERG responses in children with history of prematurity. Interestingly, Dale et al. revealed considerable disagreement between mfERG in detection of retinal abnormalities. mfERG tends to miss small local abnormalities that are detectable on OCT. On the other hand, OCT can appear normal in the face of clearly abnormal mfERG results [26].

In the present study, seven eyes of the study group had been treated with argon laser photocoagulation and one eye with cryotherapy due to stage 3 of retinopathy of prematurity in infancy. Nine eyes of prematurely born children had no history of ROP, so they had no treatment. We found no statistically significant differences in mfERG parameters from all five-ring retinal regions among these two groups of eyes (Table 4). Nevertheless, none of our patients had threshold ROP. It is known that patients with a history of threshold ROP are more likely to show abnormal foveal development, besides in the treated ROP patients SD-OCT revealed retention of the layer of retinal ganglion cells, inner plexiform layer, and inner nuclear layer in the macula [20].

Still, we acknowledge that our study has some limitations. The number of patients in the control and the study group was small. The small number of patients after cryotherapy and the same stage of ROP in all premature children make impossible to analyze whether mfERG depends on kind of therapy or stage of retinopathy. However, mfERG could be susceptible to outer retinal damage in ROP treated children.

In conclusion, our study suggests that P1 amplitudes density and P1 amplitudes vary in preterm-born children in comparison with their peers born appropriate for gestational age, which might suggest discreet macular dysfunction. The correlation between low birth weight, early gestational age, central macular thickness and mFERG components in ring 1 might evidence that decreased bipolar cells density caused by premature birth is the result of altered development of central retina reflecting in structural anomalies of the fovea.

Compliance with ethical standards

Conflict of interest All authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge or beliefs) in the subject matter or materials discussed in this manuscript.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from the parents of all children included in the study.

References

1. Cavallaro G, Filippi L, Bagnoli P, La Marca G, Cristofori G, Raffaeli G, Padrini L, Araimo G, Fumagalli M, Groppo M, Dal Monte M, Osnaghi S, Fiorini P, Mosca F (2014) The pathophysiology of retinopathy of prematurity: an update of previous and recent knowledge. Acta Ophthalmol 92:2–20
2. Fanaroff AA, Stoll BJ, Wright LL et al (2007) Trends in neonatal morbidity and mortality for very low birth weight infants. Am J Obstet Gynecol 196:147
3. Wilson-Costello D, Friedman H, Minich N et al (2007) Improved neurodevelopmental outcomes for extremely low birth weight infants in 2000–2002. Pediatrics 119:37–45
4. Wood NS, Marlow N, Costeloe K et al (2000) Neurologic and developmental disability after extremely preterm birth. N Engl J Med 343:378–384
5. Koo KY, Kim JE, Lee SM et al (2010) Effect of severe neonatal morbidities on long term outcome in extremely low birthweight infants. Korean J Pediatr 53:694–700
6. Ecsedy M, Szamosi A, Karkó C, Zubovics L, Varsányi B, Németh J, Récsán Z (2007) A comparison of macular structure imaged by optical coherence tomography in preterm and full-term children. Investig Ophthalmol Vis Sci 48:5207–5211
7. Hammer DX, Iftimia NV, Ferguson RD, Bigelow CE, Ustun TE, Barnaby AM, Fulton AB (2008) Foveal fine structure in retinopathy of prematurity: an adaptive optics Fourier domain optical coherence tomography study. Investig Ophthalmol Vis Sci 49(5):2061–2070
8. Grun G (1982) The development of the vertebrate retina: a comparative survey. Adv Anat Embryol Cell Biol 78:1–85
9. Dorn EM, Hendrickson L, Hendrickson AE (1995) The appearance of rod opsin during monkey retina development. Investig Ophthalmol Vis Sci 36:2634–2651
10. Drucker DN, Hendrickson AE (1989) The morphological development of extrafoveal human retina. Investig Ophthalmol Vis Sci 30:226

11. Yuodelis C, Hendrickson A (1986) A qualitative and quantitative analysis of the human fovea during development. Vis Res 26:847–855
12. Hendrickson AE, Yuodelis C (1984) The morphological development of the human fovea. Ophthalmology 91:603–612
13. Hendrickson AE (1994) Primate foveal development: a microcosm of current questions in neurobiology. Investig Ophthalmol Vis Sci 35:3129–3133
14. Hendrickson AE (1994) The morphologic development of human and monkey retina. In: Albert DM, Jakobiec FA (eds) Principles and practice of ophthalmology: basic sciences. WB Saunders Co, Philadelphia, pp 561–577
15. Springer AD (1999) New role for primate fovea: a retinal excavation determines photoreceptor deployment and shape. Vis Neurosci 16:629–636
16. Provis JM, Sendercoe T, Hendrickson AE (2000) Astrocytes and blood vessels define foveal rim during primate retinal development. Investig Ophthalomol Vis Sci 41:2827–2836
17. Fulton AB, Hansen RM, Moskowitz A, Barnaby AM (2005) Multifocal ERG in subjects with history of retinopathy of prematurity. Doc Ophthalmol 111:7–13
18. Hood DC, Bach M, Brigell M, Keating D, Kondo M, Lyons JS, Marmor MF, McCulloch DL, Palmowski-Wolfe AM (2012) ISCEV Standard for clinical multifocal electroretinography (2011 edition). Doc Ophthalmol 124:1–13
19. Marmor MF, Fulton AB, Holder GE, Miyake Y, Brigell M, Bach M (2009) Standard for clinical electroretinography (2008 update). Doc Ophthalmol 118:69–77
20. Wu WC, Lin RI, Shih CP et al (2012) Visual acuity, optical components, and macular abnormalities in patients with a history of retinopathy of prematurity. Ophthalmology 119(9):1907–1916
21. Ruberto G, Redaelli C, Cataldo S et al (2004) Compared progression of visual evoked potentials in preterm and term newborns. J Fr Ophtalmol 27:1031–1038
22. Michalczuk M, Urban B, Chrzanowska-Grenda B et al (2015) An influence of birth weight, gestational age, and apgar score on pattern visual evoked potentials in children with history of prematurity. Neural Plast 2015, Article ID 754864
23. Candy TR, Crowell JA, Banks MS (1998) Optical, receptoral, and retinal constraints on foveal and peripheral vision in the human neonate. Vis Res 38:3857–3870
24. Hansen RM, Moskowitz A, Fulton AB (2009) Multifocal ERG responses in infants. Investig Ophthalmol Vis Sci 50:470–475
25. Recchia FM, Recchia CC (2007) Foveal dysplasia evident by optical coherence tomography in patients with a history of retinopathy of prematurity. Retina 27:1221–1226
26. Dale EA, Hood DC, Greenstein VC, Odel JG (2010) A comparison of multifocal ERG and frequency domain OCT changes in patients with abnormalities of the retina. Doc Ophthalmol 120:175–186