

POTENTIAL ROLE OF VISUALLY EVOKED POTENTIAL AND ELECTRORETINOGRAPHY "PATTERN" IN THE DIAGNOSIS OF PRIMARY OPEN-ANGLE GLAUCOMA

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Glaucoma is a widely distributed progressive optic neuropathy characterized by progressive damage and loss of retinal ganglion cells of the retina and optic nerve, optic disc excavation increasing and usually, but not always, high intraocular pressure, which eventually results in gradual changes in the visual field. In our study, we tried to determine the potential role and place of electrophysiological researches (PERG and VEP) in the early detection of glaucoma optic neuropathy, comparing the electrophysiological parameters (P100 and A) and numeric parameters in ophthalmic patients studied. The results obtained in our study show that the application of PERG methods detects early damage to the retinal ganglion cells, whereas the same changes were not observed by VEP analysis, suggesting a potential role of predictive PERG analysis in the early diagnosis of pre-perimetric glaucoma. *Acta Medica Medianae* 2012;51(4):19-25.

Key words: glaucoma, PERG analysis, VEP analysis

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Introduction

Glaucoma is a widely distributed progressive optic neuropathy characterized by progressive damage and loss of retinal ganglion cells of the retina and optic nerve, increasing of optic disc excavation and usually, but not always, high intraocular pressure, which eventually results in gradual changes in the visual field (1). There are many types of glaucoma, the most common of which is open-angle glaucoma. Patients with elevated intraocular pressure without optic nerve damage and without visual field changes are defined as patients with ocular hypertension and they are at risk of developing glaucoma (2,3). At the same time, glaucoma is the leading cause of blindness worldwide, indicating a potentially significant early detection and treatment of this disease (4).

Glaucoma as a disease cannot be studied in isolation. For a full explanation of glaucoma, its etiology, pathogenesis, clinical presentation, and for the proper diagnosis and appropriate therapeutic approach, it is necessary to associate the studies of glaucoma with the following areas of medicine: neurology, cardiology (local and general circulation), endocrinology (study of diabetes), and geriatrics.

Early detection and prevention of glaucoma progression is vital in order to initiate or modify the medical treatment, and also to prevent permanent and total loss of sight in future (5). Diagnostic tests

which are important in glaucoma must be sensitive enough to detect early glaucoma progression and to identify those patients with ocular hypertension who are at higher risk of developing glaucoma (2,6).

Nowadays, clinical researches which should elucidate the neurophysiological aspects of the pathogenesis of primary open-angle glaucoma are rare. In the recent years, the development of various neurophysiological techniques that provide the insight into the state of the visual system as a whole (7,8) have been intensified. In use are: electrooculogram (EOG), flash electroretinogram (ERG), pattern electroretinogram (PERG), multifocal electroretinogram (mfERG), visual evoked potentials (VEP) and multifocal evoked potentials (mfVEP) (9-11).

These techniques can register early changes in the retinal structure and function of the optic nerve, which is significant both from a diagnostic and a therapeutic point of view (13,14). It has been established that the pattern electroretinogram (PERG) provides information about the function of the ganglion cell layer which is the first to be damaged in glaucoma, and visual evoked potentials (VEP) about the function of the optic nerve (15-18). In practice, we use both neurophysiological techniques together because they provide information about the function of an anatomical entirety, making it possible to monitor the transmission of visual information from the retina to the visual cortex in various diseases including glaucoma (19,20).

Bearing in mind the previous bibliographic data, in our study we tried to determine the potential role and place of electrophysiological studies (PERG and VEP) in the early detection of glaucoma optic neuropathy, comparing the electrophysiological parameters (P100 and A) and numeric parameters in ophthalmic patients studied (21-23).

Patients and methods

Clinical examination was carried out on 25 patients (50 eyes) with primary open-angle glaucoma (simplex form), 27 patients (54 eyes) with ocular hypertension and 25 patients (50 eyes) with no ophthalmic and neurological disease (control group). All study subjects were completely clinically ophthalmologically tested in terms of glaucoma (determination of visual acuity, biomicroscopy, ophthalmoscopy, daily curve of intraocular pressure, gonioscopy, standard automated perimetry), after which they were divided into the above mentioned test groups.

Each subject underwent the electrophysiological study (VEP, PERG) as a basic test to confirm the diagnosis. The obtained values of electrophysiological tests are shown as P100 wave latency value (VEP analysis) and the value of the wave amplitude N95 (PERG analysis).

Electrophysiological tests (VEP, PERG) were examined with Nihon Kohden apparatus under the following technical requirements:

VEP: Time base 30 ms, above 100Hz filter, the filters below 1Hz, the amplitude of 20 microvolts, stimulation of "pattern" (chess box), box size 32, frequency of 2 c/sec (cycle per second). Centered are 100 stimuli. Position of electrodes: Active electrode Oz, reference electrode at Fz (by 10-20 international system), the earth electrode on the vertex.

PERG: Time base 30 ms, above 100Hz filter, the filters below 1Hz, 5 microvolts amplitude, stimulation of the "pattern" (chess box), box size 32, frequency of 2 c/sec (cycle per second). Centered are 100 stimuli. Position of electrodes: Active electrode temporal bone, the reference electrode is the lower eyelid, the earth electrode at the vertex.

Tests were carried out at the Department of Ophthalmology (Office for glaucoma) and the Department of Mental Health (Office for neurophysiological tests) at the Clinical Center in Niš.

Statistical analysis

In the analysis, the default error margin of 0.05 or 5% was used for the limits of statistical significance. The comparison of representation of certain categories of attribute characteristics between groups was done with Mantel-Haenszel chi square test. To assess the interdependence between the values of some of the features correlation analysis was used and the Pearson (Pearson) correlation coefficients were calculated.

Results

Demographic and clinical characteristics of the patients in our study are shown in Table 1.

Table 1. Demographic and clinical characteristics of the studied groups

Characteristic	Group			SS
	Glaucoma (n=50)	Ocular hypertension (n=54)	Control (n=50)	
Age	55,60±13,16	52,00±8,67	52,96±9,44	n.s.
Men	18 (36,0%)	18 (33,3%)	22 (44,0%)	n.s.
Women	32 (64,0%)	36 (66,7%)	28 (56,0%)	
DM	6 (12,5%)	2 (3,7%)	6 (12,5%)	n.s.
HTA	10 (20,0%)	6 (11,1%)	16 (32,0%)	n.s.
Thyroid disease	6 (12,0%)	2 (3,8%)	2 (4,0%)	n.s.
Positive family anamnesis	10 (20,0%)	12 (23,1%)	8 (16,0%)	n.s.
Other	8 (17,4%)	20 (37,0%)	8 (17,4%)	n.s.

SS- statistical significance, DM -diabetes mellitus, HTA-hypertensio arterialis, n.s.- no statistical significance, n – total number of subjects in the relevant groups

Table 2. P100 wave latency values in compared groups

Characteristic	Group			Comparison
	Glaucoma (n=50)	Ocular hypertension (n=54)	Control (n=50)	
P100 OD (msec)	114,63±2,79	100,79±5,02	97,94±3,92	A‡, B‡, C*
P100 OD up to 113,4 msec	14 (28,0%)	54 (100,0%)	50 (100,0%)	A‡, B‡
P100 OD over 113,4 msec	36 (72,0%)	-	-	
P100 OS (msec)	114,23±2,55	101,03±4,92	98,27±4,01	A‡, B‡, C*
P100 OS up to 113,4 msec	22 (44,0%)	54 (100,0%)	50 (100,0%)	A‡, B‡
P100 Os over 113,4 msec	28 (56,0%)	-	-	

P100 OD – right eye wave latency value P100, P100 OS – P100 of the left eye,

A – glaucoma vs ocular hypertension; B – glaucoma vs control group, C – ocular hypertension vs control group, * - p<0,05; † - p<0,01; ‡ - p<0,001, n- total number of subjects in the relevant groups

Table 3. Values of the wave amplitude N95 in compared groups

Characteristic	Group			Comparison
	Glaucoma (n=50)	Ocular hypertension (n=54)	Control (n=50)	
A OD (microV)	0,26±0,12	1,43±0,63	1,69±0,40	A‡, B‡
A OD over 0,4 microV	-	44 (81,5%)	50 (100,0%)	A‡, B‡, C*
A OD up to 0,4 microV	50 (100,0%)	10 (18,5%)	-	
A OS (microV)	0,29±0,13	1,34±0,62	1,66±0,44	A‡, B‡, C*
A OS over 0,4 microV	-	44 (81,5%)	50 (100,0%)	A‡, B‡, C*
A OS up to 0,4 microV	50 (100,0%)	10 (18,5%)	-	

A OD – left eye amplitude wave value N95, A OS – right eye value, A- glaucoma vs ocular hypertension; B – glaucoma vs control group, C – ocular hypertension vs control group * -p<0,05; †- p<0,01; ‡ - p<0,001, n – total number of subjects in the relevant group

Table 4. Correlation between P100 values and other factors of interest in three groups of subjects

Characteristic	Glaucoma (n=50)		Ocular hypertension (n=54)		Control (n=50)	
	r	p	r	p	r	p
Age	-0,139	0,334	0,026	0,851	-0,009	0,950
Visus	-0,029	0,842	-	-	-	-
IOP (mmHg)	0,262	0,066	-0,037	0,790	0,274	0,054
MD (dB)	-0,671	<0,001	-0,810	<0,001	-0,944	<0,001
CPSD	0,689	<0,001	0,528	<0,001	0,257	0,071
Disease stage	0,777	<0,001	-	-	-	-
Gonioscopy	0,166	0,249	0,265	0,063	0,125	0,409
Excavation	0,647	<0,001	0,432	0,001	-0,214	0,135
A (microV)	-0,653	<0,001	-0,747	<0,001	-0,779	<0,001

In the group of glaucoma subjects, a significant positive correlation was found between the P100 and CPSD values (r=0,689; p<0,001), disease stage (r=0,777; p<0,001) and excavation (r=0,647; p<0,001), while a significant negative correlation was found between the P100 and MD values (r=-0,671; p<0,001), as well as A (r=-0,653; p<0,001).

Table 5. Correlation between A values and other factors of interest in three groups of subjects

Characteristic	Glaucoma (n=50)		Ocular hypertension (n=54)		Control (n=50)	
	r	p	r	p	r	p
Age	-0,009	0,952	0,041	0,768	-0,182	0,205
Visus	0,177	0,219	-	-	-	-
IOP (mmHg)	-0,357	0,011	-0,111	0,426	0,009	0,953
MD (dB)	0,812	<0,001	0,854	<0,001	0,837	<0,001
CPSD	-0,785	<0,001	-0,597	<0,001	-0,368	0,009
Disease stage	-0,781	<0,001	-	-	-	-
Gonioscopy	-0,242	0,091	-0,236	0,099	-0,078	0,606
Excavation	-0,813	<0,001	-0,523	<0,001	0,095	0,514

In the group of glaucoma subjects, a significant positive correlation was found between the A and MD values (r=0,812; p<0,001), while a significant negative correlation was found between the A and CPSD values (r=-0,785; p<0,001), disease stage (r=-0,781; p<0,001), excavation (r=-0,813; p<0,001), as well as P100 (r=-0,653; p<0,001).

As shown in Table 1, the difference in the average age of the respondents, by gender, presence of DM, hypertension, thyroid disease, family history and other major diseases among the samples tested, was not statistically significant, indicating uniformity of the tested groups.

In the further course of the study, we analyzed the latencies of P100 and amplitude N95, as the main parameters of the electrophysiological methods used in our study. The results are shown in Table 2 and Table 3.

Based on the results obtained for P100, the examinees were categorized into two categories (up to 113.4 and over 113.4 msec). The analysis of the average value of P100 on the right eye in patients with glaucoma showed significantly higher values ($p < 0.001$) compared to other groups. The value of P100 was detected in patients with ocular hypertension, in comparison to the control group ($p < 0.05$). Individual representation of P100 values in patients with glaucoma showed a significant difference ($p < 0.001$) compared to the other groups.

Further evaluation of the potential electrophysiological parameters included the detection and analysis of the wave amplitude in the N95 PERG analysis (IIA). Based on the values obtained for A, all the patients were categorized into two groups (up to 0.4 microV and over 0.4 microV). The results presented in Table 3 show that the value of A in patients with glaucoma is significantly lower than in other tested groups ($p < 0.001$). Evident and statistically significant is the difference ($p < 0.001$, $p < 0.05$) in the representation of the value of A among all groups (0.001). Within the group of patients with ocular hypertension 18.5% of persons with a significant decrease in the amplitude of the wave N95 PERG were detected, which was not detected in the same group of subjects, using the P100 value analysis.

Taking into account the results obtained for P100 and A, we have tried to use a correlation in order to determine if there was any correlation between the electrophysiological methods tested in the examined parameters of the subjects. The results are shown in Table 4 and Table 5.

In the group of patients with ocular hypertension, a significant positive correlation was confirmed between P100 and CPSD values ($r = 0.528$, $p < 0.001$), as well as the excavation ($r = 0.432$, $p = 0.001$), whereas a significant negative correlation between the values MD P100 ($r = -0.810$, $p < 0.001$), and A ($r = -0.747$, $p < 0.001$) was confirmed.

In the control group, a significant correlation between P100 and other factors of interest was not confirmed.

In the group of patients with glaucoma, a significant positive correlation was confirmed between the values of A and MD ($r = 0.812$, $p < 0.001$), whereas a significant negative correlation was confirmed between the values of A and IOP ($r = -0.357$, $p < 0.05$), CPSD ($r = -0.785$, $p < 0.001$), disease stage ($r = -0.781$, $p < 0.001$) and excavation ($r = -0.813$, $p < 0.001$).

In the group of patients with ocular hypertension, a significant positive correlation was confirmed between the values of A and MD ($r = 0.854$, $p < 0.001$), whereas a significant negative correlation was confirmed between the values of A and CPSD ($r = -0.597$, $p < 0.001$), and excavation ($r = -0.523$, $p < 0.001$). In the control group, no significant correlations between the certified values and other factors of interest were confirmed.

Discussion

Pathogenetic mechanisms responsible for the damage and loss of retinal ganglion cells (RGC) in glaucoma are still not fully interpreted. Damage to these cells is often attributed to high intraocular pressure (IOP), which exists in most patients – The Mechanical theory of origin of glaucoma (24). However, evidence from studies with low-tension glaucoma and ocular hypertension indicate that elevated IOP is not sufficient to explain the glaucomatous optic neuropathy. Therefore, other risk factors such as vascular and hemorrheological dysfunction and metabolic disorders stand are emphasized – The Vascular theory of glaucoma occurrence (25,26). Recent histological studies of the retina in experimental glaucoma and in patients with glaucoma have indicated that retinal nerve cells suffer damage by apoptosis-like mechanism – The Excitotoxic theory of origin of glaucoma (4,27).

The tests in our study included subjects diagnosed with glaucoma, patients with ocular hypertension and the control group. Based on analysis of demographic and clinical characteristics of the patients included in our study, it is evident that there is no significant difference in the evaluated characteristics between the tested groups, suggesting uniformity and proper selection of patients in our study.

Results of the VEP analysis showed an increase of P100 latency in patients with glaucoma compared to other groups. Positive correlation in patients with glaucoma was found between P100 and CPSD values, stage of disease and excavation, while a negative correlation between the values MD P100 and A was confirmed. In individuals with ocular hypertension, a positive correlation between the value of P100 and CPSP and stage of disease was confirmed, while a significant negative correlation between the values MD and P100 and A values was confirmed. The data point to significant increases in P100 latency in patients with glaucoma, compared to the other study groups, which is consistent with previous studies (28-30), indicating a change in functional status of optic nerve fibers, as well as the overall visual pathway postretinally located, probably as a result of dysfunction of the retinal structure (30,22). Unlike patients with glaucoma, in individuals with ocular hypertension, prolonged P100 above the physiologic range was not detected. Our data are consistent with the findings which have shown that there was no significant difference between the control group and those with ocular hypertension, with the value of VEP (31). However, in earlier studies, a prolonged P100 in patients with ocular hypertension (32,33) was noticed, indicating that functional impairment may precede excavation changes as well as visual field defects (30). Discrepancies in the results are most likely resulting from different patient selection. In fact, previous studies

included both patients with early glaucoma in the group of patients with ocular hypertension (32,33), which may result in conflicting results obtained.

PERG analysis in our study showed a significantly reduced value of the wave amplitude N95 in patients with glaucoma, compared to all groups. However, the analysis of the PERG of patients with ocular hypertension showed that in 18.5% of respondents reduced PERG value analysis was detected, while the other patients (81.5%) had normal findings PERG. At the same time, this group, even though it held the lower amplitude values, showed no signs of damage to the visual field. Positive correlation in patients with glaucoma was found between the values of A and MD, while a significant negative correlation between the value of A and IOP, CPSD, stage of disease and the size of the excavation was confirmed. Similar correlation results were obtained in patients with ocular hypertension (confirmed by a positive correlation between the values of A and MD, while a negative correlation was confirmed between the values of A and the excavation and CPSD). The results obtained by the analysis of PERG in our study are consistent with previous studies (34,11), indicating the importance of damage to the retinal ganglion cells in the pathogenesis of the glaucoma disease. Correlation results in people with glaucoma are consistent with previous studies, which also showed a positive correlation with changes in the visual field (35-37). Similar to these findings, various studies using a similar methodology have shown that patients with glaucoma, in PERG amplitudes, show lower levels and are positively correlated with changes in the optic disc and visual field (28,38,39). Decrease in amplitude detected in some patients with ocular hypertension indicates that this person has a certain degree of retinal dysfunction but still without any detectable changes in the visual field, and that these changes are only detected by analyzing PERG. Bearing in mind that the results show that the number of such patients within the patients with ocular hypertension six months after the beginning of the test doubled, it

can be assumed that the analysis of PERG is one of the reliable methods for early detection of pre-perimetric glaucoma. Our results also confirmed previous studies that have shown the existence of PERG changes with no changes in the visual field in patients with ocular hypertension (34,40-43) as well as in animal studies (17). It should be noted that changes in the PERG analysis in these patients do not necessarily determine the occurrence of glaucoma disease, as it is well known that only about 1% of those with ocular hypertension develop the glaucoma disease annually (44). Therefore, we need proper and precise separation of these patients, which would prevent unwanted effects of antiglaucoma therapy, particularly in patients in whom there is no possibility for the development of glaucoma.

In light of these results, the introduction of electrophysiological testing in everyday clinical practice for the early detection of glaucoma (diagnostic value) and in the monitoring of patients diagnosed with glaucoma (prognostic value) should be considered, with the aim of confirming the success of antiglaucomatous appropriate therapy in conjunction with other clinical diagnostic tests (7,14,19).

The test nowadays increasingly used for the functional state of ganglion cells is the multifocal electroretinogram (9). Simultaneous analyses of the local responses are recorded from different parts of the retina, allowing their fusion and reading the entire retinal responses. Therefore, nowadays, the use of this method allows precise localization of retinal dysfunction, long before any changes are detectable by perimetry allowing timely detection of pre-perimetric glaucoma and its prompt prevention (19,45).

Conclusion

Bearing in mind the results obtained in our study, particularly the values of the amplitude N95 PERG, we can assume that some electrophysiological methods may represent a significant ancillary diagnostic method for prediction and early detection of glaucoma and the monitoring of disease in patients diagnosed with glaucoma.

References

1. Fraser S, Wormald R. Glaucoma. In: Yanoff M, Duker JS, editors. *Ophthalmology*. 2nd ed. St. Louis, Mo: Mosby Elsevier; 2005. p. 1413-615.
2. Kanski JJ. *Glaucoma*. In: Kanski JJ, editor. *Clinical ophthalmology*. 5th ed. Butterworth-Heinemann: Elsevier Science Limited; 2003. p. 192-268.
3. Gordon MO. The Ocular Hypertension Treatment Study: a randomized trial determines that topical ocular hypotensive medication delays or prevents the onset of primary open-angle glaucoma. *Arch Ophthalmol* 2002; 120(6):701-13. [[CrossRef](#)] [[PubMed](#)]
4. Pajić D. *Zelena mrena*. Beograd: Sudio MC; 2007. p. 14-75.
5. *Glaucoma and the brain*. *Insight* 2012; 37(3): 22.
6. European Glaucoma Society: Terminology and guidelines for glaucoma. IIInd edition, DOGMA S.r.l., 2003.
7. Meigen T, Bach M. Electrophysiology in the diagnosis of glaucoma. In: Grehn F, Stamper R, editors. *Essentials in Ophthalmology: glaucoma*. Philadelphia: L J Katz; 2006. p. 73-9.
8. Đurić S. Fiziologija vizuelnog sistema. In: Đurić S, Mihaljev-Martinov J, editors. *Klinička neurofiziologija*. 3rd ed. Niš: Prosveta; 1998. p. 236-54.
9. Chan HH-I, Ng Y-f, Chu PH-w. Applications of the multifocal electroretinogram in the detection of glaucoma. *Clinical and experimental optometry* 2011; 94: 247-58. [[CrossRef](#)] [[PubMed](#)]
10. Fortune B, Zhang X, Hood D, Demirel S, Petteron E, Jamil A et al. Effect of recording duration on the diagnostic performance of multifocal visual-evoked potentials in high-risk ocular hypertension and early glaucoma. *J Glaucoma*. 2008; 17(3): 175-82. [[CrossRef](#)] [[PubMed](#)] [[PubMedCentr](#)]
11. Bach M, Hoffmann MB. The origin of the pattern electroretinogram (PERG). In: Heckenlively J, Arden G, editors. *Principles and Practice of Clinical Electrophysiology of Vision*. Cambridge: MIT Press; 2006. p. 185-96. [[PubMed](#)]
12. Stiefelmeyer S, Neubauer AS, Berninger T, Arden GB, Rudolph G. The multifocal pattern electroretinogram in glaucoma. *Vision Res* 2004; 44: 103-12. [[CrossRef](#)] [[PubMed](#)]
13. Bowd C, Vizzeri G, Tafreshi A, Zangwill LM, Sample PA, Weinreb RN. Diagnostic Accuracy of Pattern Electroretinogram Optimized for Glaucoma Detection. *Ophthalmology* 2009; 116: 437-43. [[CrossRef](#)] [[PubMed](#)] [[PubMedCentr](#)]
14. Leonard K, Hutnik C. Using Electroretinography for Glaucoma Diagnosis. In: Schacknow PN, Samples JR, editors. *The Glaucoma Book*. New York: Springer Science + Business Media; 2010. p. 265-67.
15. Bach M, Hoffmann MB. Update on the pattern electroretinogram in glaucoma. *Optom Vis Sci*. 2008; 85 (6): 386-95. [[CrossRef](#)] [[PubMed](#)]
16. Holder GE. Pattern electroretinography (PERG) and an integrated approach to visual pathway diagnosis. *Prog Retin Eye Res* 2001; 20: 531-61. [[CrossRef](#)]
17. Viswanathan S, Frishman LJ, Robson JG. The uniform field and pattern ERG in macaques with experimental glaucoma: removal of spiking activity. *Invest Ophthalmol Vis Sci*. 2000; 41(9): 2797-810. [[PubMed](#)]
18. Parisi V, Miglior S, Manni G, Centofanti M, Bucci MG. Clinical ability of pattern electroretinograms and visual evoked potentials in detecting visual dysfunction in ocular hypertension and glaucoma. *Ophthalmology* 2006; 113: 216-28. [[CrossRef](#)] [[PubMed](#)]
19. Lam LB. Focal and multifocal electroretinogram. In: Lam LB. *Electrophysiology of Vision: Clinical Testing and Applications*. London: Taylor & Francis; 2005. p. 65-151.
20. Xin D, Greenstein VC, Ritch R, Liebmann JM, De Moraes CG, Hood DC. A comparison of functional and structural measures for identifying progression of glaucoma. *Invest Ophthalmol Vis Sci* 2011; 52(1): 519-26. [[CrossRef](#)] [[PubMed](#)] [[PubMedCentr](#)]
21. Bode SF, Jehle T, Bach M. Pattern electroretinogram in glaucoma suspects: new findings from a longitudinal study. *Invest Ophthalmol Vis Sci* 2011; 52(7): 4300-6. [[CrossRef](#)] [[PubMed](#)]
22. Vaegan, Hollows FC. Visual-evoked response, pattern electroretinogram, and psychophysical magnocellular thresholds in glaucoma, optic atrophy, and dyslexia. *Optom Vis Sci* 2006; 83(7): 486-98. [[CrossRef](#)] [[PubMed](#)]
23. Fortune B, Zhang X, Hood DC, Demirel S, Patterson E, Jamil A et al. Effect of recording duration on the diagnostic performance of multifocal visual-evoked potentials in high-risk ocular hypertension and early glaucoma. *J Glaucoma* 2008; 17(3): 175-82. [[CrossRef](#)] [[PubMed](#)] [[PubMedCentr](#)]
24. Mozaffarieh M, Grieshaber MC, Flammer J. Oxygen and blood flow: players in the pathogenesis of glaucoma. *Mol Vis* 2008; 14: 224-33. [[PubMed](#)] [[PubMedCentr](#)]
25. Delaney Y, Walshe TE, O'Brien C. Vasospasm in glaucoma: clinical and laboratory aspects. *Optom Vis Sci* 2006; 83(7): 406-14. [[CrossRef](#)] [[PubMed](#)]
26. Mackenzie PJ, Cioffi GA. Vascular anatomy of the optic nerve head. *Can J Ophthalmol* 2008; 43: 308-12. [[PubMed](#)]
27. Agarwal R, Gupta SK, Agarwal P, Saxena R, Agrawal SS. Current concepts in the pathophysiology of glaucoma. 2009; 57(4): 257-66.
28. Bach M. Electrophysiologic approaches for early detection of glaucoma. *Eur J Ophthalmol* 2001; 2: S41-9.
29. Weizer JS, Musch DC, Niziol LM, Khan NW. Multifocal visual evoked potentials for early glaucoma detection. *Ophthalmic Surg Lasers Imaging* 2012; 43(4): 335-40. [[CrossRef](#)] [[PubMed](#)]
30. Parisi V. Impaired visual function in glaucoma. *Clin Neurophysiol* 2001; 112(2): 351-8. [[CrossRef](#)]
31. Sampson GP, Badcock DR, Walland MJ, McKendrick AM. Foveal contrast processing of increment and decrement targets is equivalently reduced in glaucoma. *Br J Ophthalmol* 2008; 92(9): 1287-92. [[CrossRef](#)] [[PubMed](#)]
32. Vaegan, Hollows FC. Visual-evoked response, pattern electroretinogram, and psychophysical magnocellular thresholds in glaucoma, optic atrophy, and dyslexia. *Optom Vis Sci* 2006; 83(7): 486-98. [[CrossRef](#)] [[PubMed](#)]
33. Parisi V, Bucci MG. Visual evoked potentials after photostress in patients with primary open-angle glaucoma and ocular hypertension. *Invest Ophthalmol Vis Sci*. 1992; ;33(2):436-42. [[PubMed](#)]
34. Ventura LM, Porciatti V, Ishida K, Feuer WJ, Parrish RK 2nd. Pattern electroretinogram abnormality and glaucoma. *Ophthalmology* 2005; 112(1): 10-9. [[CrossRef](#)] [[PubMed](#)] [[PubMedCentr](#)]

35. Tsaousis KT, Plainis S, Parry NR, Pallikaris IG, Tsilimbaris MK, Detorakis ET. Visual Electrodiagnosis in glaucoma screening: A clinical study. *J Glaucoma* 2013, Epub ahead of print. [[CrossRef](#)] [[PubMed](#)]
36. Cellini M, Toschi PG, Strobbe E, Balducci N, Campos EC. Frequency doubling technology, optical coherence technology and pattern electroretinogram in ocular hypertension. *BMC Ophthalmol* 2012; 12: 33. [[CrossRef](#)] [[PubMed](#)] [[PubMedCentr](#)]
37. Kiszkielis M, Lubinski W, Penkala K. The photopic negative response as a promising diagnostic tool in glaucoma. A review. *Klin Oczna* 2012; 114(2): 138-42. [[PubMed](#)]
38. Tafreshi A, Racette L, Weinreb RN, Sample PA, Zangwill LM, Medeiros FA, Bowd C. Pattern electroretinogram and psychophysical tests of visual function for discriminating between healthy and glaucoma eyes. *Am J Ophthalmol* 2010; 149(3): 488-95. [[CrossRef](#)] [[PubMed](#)] [[PubMedCentr](#)]
39. Salgarello T, Colotto A, Falsini B, Buzzonetti L, Cesari L, Iarossi G et al. Correlation of pattern electroretinogram with optic disc cup shape in ocular hypertension. *Invest Ophthalmol Vis Sci*. 1999; 40:1989-97. [[PubMed](#)]
40. Riva CE, Salgarello T, Logean E, Colotto A, Galan EM, Falsini B. Flicker-evoked response measured at the optic disc rim is measured in ocular hypertension and early glaucoma. *Invest Ophthalmol Vis Sci* 2004; 45(10): 3662-8. [[CrossRef](#)] [[PubMed](#)]
41. Arai M, Yoshimura N, Sakaue H, et al. A 3-year follow-up study of ocular hypertension by pattern electroretinogram. *Ophthalmologica* 1993; 207: 187-95. [[CrossRef](#)] [[PubMed](#)]
42. Bayer AU, Maag KP, Erb C. Detection of optic neuropathy in glaucomatous eyes with normal standard visual fields using a test battery of short-wavelength automated perimetry and pattern electroretinography. *Ophthalmology* 2002; 109(7): 1350-61. [[CrossRef](#)]
43. Aldebasi YH, Drasdo N, Morgan JE, North RV. S-cone, L + M-cone, and pattern, electroretinograms in ocular hypertension and glaucoma. *Vision Res* 2004; 44: 2749-56. [[CrossRef](#)] [[PubMed](#)]
44. Kass MA, Heuer DK, Higginbotham EJ, Johnson CA, Keltner JL, Miller JP et al. The Ocular Hypertension Treatment Study: a randomized trial determines that topical ocular hypotensive medication delays or prevents the onset of primary open-angle glaucoma. *Arch Ophthalmol* 2002; 120(6): 701-13. [[CrossRef](#)] [[PubMed](#)]
45. Miguel-Jiménez JM, Ortega S, Boquete L, Rodríguez-Ascariz JM, Blanco R. Multifocal ERG wavelet packet decomposition applied to glaucoma diagnosis. *Biomed Eng Online* 2011; 17: 10-37.

POTENCIJALNA ULOGA VIZUELNO EVOCIRANIH POTENCIJALA I "PATTERN" ELEKTRORETINOGRAFIJE U DIJAGNOZI PRIMARNOG GLAUKOMA OTVORENOG UGLA

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Glaukom predstavlja široko distribuiranu progresivnu optičku neuropatiju koja se karakteriše postepenim oštećenjem i gubitkom ganglijskih ćelija retine i očnog živca, povećanjem ekskavacije optičkog diska i obično, mada ne uvek, visokim intra-okularnim pritiskom, što na kraju rezultuje postepenim promenama u vidnom polju. U našoj studiji smo pokušali da utvrdimo potencijalnu ulogu i mesto elektrofizioloških ispitivanja (PERG i VEP) u ranom otkrivanju glaukomne optičke neuropatije, upoređivanjem elektrofizioloških parametara (P100 i A) i numeričkih oftalmoloških parametara kod ispitivanih bolesnika. Rezultati dobijeni u našoj studiji pokazuju da primena PERG metode otkriva rana oštećenja ganglijskih ćelija retine, dok iste promene nisu ustanovljene VEP analizom, ukazujući na potencijalnu prediktivnu ulogu PERG analize u ranoj dijagnostici preperimetrijskog glaukoma. *Acta Medica Medianae* 2012;51(4):19-25.

Ključne reči: glaukom, PERG analiza, VEP analiza