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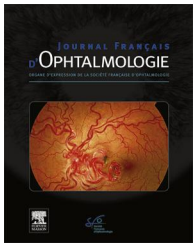

ORIGINAL ARTICLE

Pattern ERG, pattern VEP, and GCL thickness in diabetic patients with no diabetic retinopathy

ERG pattern, potentiels evoqués visuels (PEV) avec pattern et épaisseur de la couche des cellules ganglionnaires (GCL) chez les patients diabétiques sans rétinopathie diabétique

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KEYWORDS
Electrophysiology;
Diabetic retinopathy;
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RNFL

Summary
Purpose. — The main objective of this study was to compare the retinal ganglion cell (RGC) function of diabetic patients without diabetic retinopathy with the RGC function of a control group, using functional tests and anatomical assessments.
Methods. — A cross-sectional prospective pilot study was conducted on two groups. We compared the results of functional tests (Pattern ERG and Pattern VEP - PERG and PVEP) and anatomical tests (macular and RNFL OCT) in a diabetic group without diabetic retinopathy to a control group. The χ^2 test was used to study qualitative data, and the *t* test was used for quantitative data. The significance threshold was a *P* value less than 0.05.
Results. — A total of 37 eyes were included in the study. None of the demographic variables showed any significant association or effect on any of the two groups. GCL thickness was significantly reduced in the diabetic group in the superior, inferior, and nasal outer circles, with a *P* value < 0.001. The amplitude of the P100 wave was significantly reduced in the diabetic group, with a *P* value < 0.05 for the pattern sizes of 60′ and 30′, and the diabetic group had a longer latency for the 15′ VEPs. All of the components of the PERG responses were significantly altered in the diabetic group, with a *P* value < 0.05.

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Conclusion. — Our study indicates that combining different tests may be used as an early means of detection of compromised retinal neuron function in diabetic eyes during the course of early diabetic retinopathy.

MOTS CLÉS
Électrophysiologie ;
Rétinopathie diabétique ;
Potentiels évoqués visuels de motif ;
ERG de motif ;
RNFL

Résumé
Objectif. — L'objectif principal de cette étude est de comparer la fonction des cellules ganglionnaires rétiniennes (GCL) chez les patients diabétiques sans rétinopathie diabétique avec celle d'un groupe témoin, en utilisant des tests fonctionnels et des évaluations anatomiques.
Méthodes. — Une étude pilote prospective transversale a été menée sur deux groupes. Nous avons comparé les résultats des tests fonctionnels (ERG pattern et PEV pattern) et des tests anatomiques (OCT maculaire et RNFL) dans un groupe de diabétiques sans rétinopathie diabétique et dans un groupe témoin. Le test du χ^2 a été utilisé pour étudier les données qualitatives et le test *t* a été utilisé pour les données quantitatives. Le seuil de signification était un niveau de p inférieur à 0,05.
Résultats. — Un total de 37 yeux a été inclus dans l'étude. Aucune des variables démographiques n'a montré d'association significative ou d'effet sur l'un des deux groupes. L'épaisseur de la couche des cellules ganglionnaires était significativement réduite dans le groupe diabétique dans les cercles externes supérieur, inférieur et nasal avec une valeur de $p < 0{,}001$. L'amplitude de l'onde P100 était significativement réduite dans le groupe diabétique avec une valeur de $p < 0{,}05$ pour la taille de motif de 60' et 30', et le groupe diabétique présentait une latence plus longue pour les PEV pattern de 15'. Tous les composants des réponses ERG pattern étaient significativement altérés dans le groupe diabétique avec une valeur de $p < 0{,}05$.
Conclusion. — Notre étude indique que la combinaison de différents tests peut être utilisée comme moyen précoce de détection de la fonction des neurones rétiniens dans les yeux diabétiques au cours des premiers stades de la rétinopathie diabétique.

Introduction

Diabetic retinopathy (DR) is the main factor behind the decrease of visual acuity within the adult population worldwide [1]. Studies have shown that diabetic retinopathy is mainly caused by a vascular disease, and that the modifications of the endothelium layer of the retinal vessels cause a breakdown of the blood—retina barrier but also increase vascular permeability [2]. Several recent studies reported that neuro-retinal dysfunction is also present at an early stage of the development of diabetic retinopathy and therefore the retinal neuronal cells' function may be affected long before the blood—retinal barrier is significantly altered [3—7]. The effect of diabetes on the retinal ganglion cell layer (GCL), the inner plexiform layer and on the retinal nerve fiber layer (RNFL) in diabetic patients has been studied in recent studies [8,9].

The Visual evoked potentials (VEPs) are series of bioelectric potential recorded at the surface of the occipital cortex, which are generated after a visual stimulus and assess the function of the visual pathway from the retina to the visual cortex. The pattern VEPs use a pattern visual test (usually a chessboard) and the responses are detected with electrodes placed on the scalp [10]. Multiple studies found an association between retinal neovascularization due to a proliferative diabetic retinopathy and abnormal VEPs, and the dysfunction is explained by a damage at the level of the ganglion cells and the retinal nerve fiber in those diabetic patients [11,12]. Heravian et al. [13] recently evaluated the results of the VEPs and detected a dysfunction of the retinal ganglion cells before the onset of clinical signs of the disease. A dysfunction at the level of the optic nerve or RGCs may alter the amplitude or latency of the VEPs but damage anywhere on the visual pathway can also affect the VEPs (maculopathy, for example). That's why any abnormality of the VEPs should be evaluated with an assessment of the retina, with a pattern electroretinogram (PERG) to localize the lesion and study the dysfunction.

The PERG assesses the function of the inner layers of the macula and the retinal ganglion cell layer function. It can help in differentiating whether the dysfunction is at the level of the macula or the optic nerve [14]. PERG is performed by using a visual stimulus (a structured pattern) and by recording the response with conjunctival or skin electrodes. Recent studies showed that the sensitivity of PERG in diagnosing preclinical abnormalities related to diabetes is fair. Caputo et al. [15] evaluated the results of PERG in patients with type 1 diabetes with early diabetic retinopathy

and they assessed first a reduced amplitude of the N95 wave in diabetics compared to control subjects and second that the amplitude was inversely related to the duration of diabetes. A study performed in 2010 also evaluated the efficacy of PERG after treatment of diabetic macular edema with intravitreal injections of bevacizumab and described an increase of the PERG amplitude after treatment [16].

The main objective of this study is to compare the RGC function of diabetic patients without diabetic retinopathy with the RGC function of a control group, using functional tests (PERG and VEPs) and anatomical assessments (macular and RNFL OCT), and to determine the extent of the dysfunction (if present) of the recorded parameters in diabetic subjects without any diabetic retinopathy. Furthermore, we wanted to evaluate the usefulness of the functional vision assessment with ocular electrophysiology tests in detecting the earliest signs of diabetic retinopathy, even before the vascular retinal changes occur.

Material and methods

We conducted a cross sectional prospective study in the Department of Ophthalmology of a tertiary care center (Eye and Ear Hospital, *Naccash, Lebanon*). We compared the results of two groups: a diabetic group and a control group. The participants of the two groups were recruited between January 2022 and April 2022. The study was approved by the Review Board Committee of the Eye and Ear University Hospital and was performed in accordance with the ethical standards of the Declaration of Helsinki. All patients signed an informed consent and agreed to be included in this study.

Subjects

In this cross-sectional prospective study, we compared the electrophysiological results and the imaging results of diabetic patients without diabetic retinopathy (according to the ETDRS study classification) to the results of a control group admitted to the ophthalmology department of our hospital, without any retinal or corneal or lens disease (e.g., strabismic patients with normal vision).

Inclusion criteria

- Patients were included in the first group if they had diabetes (type 2 diabetus mellitus) with no diabetic retinopathy (as defined by the ETDRS study): ''No evidence of diabetic retinopathy''.
- Patients were included in the control group if they had no diabetes and no other cause of retinopathy or ocular disease which can alter the visual function.

Exclusion criteria

Patients with pre-existing ocular conditions that could affect retinal, or ganglion cell layer measurements were excluded from the study. Specifically, individuals with glaucoma, significant media opacities (e.g., cataracts, corneal opacities, or vitreous hemorrhage), subclinical macular edema, epiretinal membranes, or any form of vitreomacular traction were not included to minimize potential confounding variables. This ensured that retinal and ganglion cell thickness assessments were not influenced by structural changes unrelated to the condition under investigation.

Demographic and clinical data were collected when the patient was admitted to the hospital, including age, sex, duration of diabetes, Hemoglobin A1c level, the presence of associated blood hypertension, lens status, the use of insulin injections, intraocular pressure (IOP) measured by Goldmann tonometry, logMAR Best Corrected Visual Acuity (BCVA).

At the beginning of the study, patients were selected after they underwent a comprehensive eye examination, including refraction, measurement of best-corrected visual acuity using the Snellen test, measurement of IOP, slit lamp examination without and with pupillary dilation, a fundoscopic examination with a Volk pre-corneal lens, and with an indirect ophthalmoscope.

Methods

All patients underwent the subsequent tests: macular OCT, RNFL OCT, Pattern VEP, and Pattern ERG.

Macular and RNFL OCT were performed using the Heidelberg Spectralis to obtain SD-OCT. The macular OCT focuses on the subject's fovea while the operator of the OCT camera monitored the stability of the foveal fixation using an infrared camera. The scan extended from the vitreoretinal interface to the Bruch's membrane. Central macular thickness was generated employing a cross-sectional OCT scan.

Pattern VEPs were recorded using the MonPack One of Metrovision (France) according to the International Society for Clinical Electrophysiology of Vision (ISCEV) standard. The distance between the subjects' eyes and the monitor was 1 meter. The patients were tested with the adapted optical correction for the distance of the test (if needed), under mesopic conditions which were identical for all patients. The test was done without dilation, and each eye was tested independently. The electrodes were positioned in line with the ISCEV guidelines: 2 reference electrodes were positioned at the outer canthus (right and left eye); the ground electrode was placed at the participant vertex; two active subcutaneous electrodes were placed on the occipital scalp over the visual cortex. Responses were recorded using 3 different check sizes: 60′, 30′, 15′ while the participants focused on a fixation point. The mean screen luminance was 100 cd/m^2 with 99% contrast. The recording conditions were those of the ISCEV standards. We recorded two responses for each eye and for each pattern size to ensure substantiate reproducibility. Amplitude and latency of the P100 wave were recorded.

Pattern ERG was performed using the MonPack One of Metrovision (France) with the ISCEV standards. PERG was recorded using alternate black and white reverse checkboards with a central fixation point. The check size was 0.8°. The contrast between black and white squares was close to 100%. The responses were recorded with a total of 5 electrodes: 2 DTL (Dawson—Trick—Litzkow) electrodes (one electrode in each eye; topical anesthesia was used), 2 reference electrodes located at the outer canthus (right and left eye), and 1 ground electrode placed at the participant vertex. The software recorded the amplitude and the latency of the N35, P50, and N95 waves.

Table 1 Demographic variables and baseline characteristics of the patients.

Variable	Control Group	Diabetic Group
Sex		
F	10 (52.6%)	8 (44.4%)
M	9 (47.4%)	10 (55.6%)
Age	53 ± 4	54 ± 3
Associated pathologies	None	None
BCVA (logMar)	0.04 ± 0.07	0.06 ± 0.07
HBA_{1c} (%)	5.5 ± 0.55	6.43 ± 0.4
IOP (mmHg)	14.68 ± 2.47	14.22 ± 2.16
CRT (μm)	263.21 ± 9.94	258.17 ± 13.6
OCT quality of segmentation	29.1 ± 2.13	28.6 ± 1.89

Statistical analysis

We compared the anatomical assessment to the electrophysiological results of the two groups. We used the IBM SPSS 20.0 for statistical analysis. The results were shown as means ± standard deviations (SD), and as percentages with confidence intervals of 95%. We used χ^2 to study qualitative data and the *t* test for quantitative data. Significance threshold was a level of *P* lower than 0.05.

Results

A total of 37 eyes were included in the study: group 1 = 18 diabetic eyes (diabetic group) without any diabetic retinopathy, 10 women and 8 men (44.4% and 55.6%, respectively); group 2 = 19 eyes in the control group, 10 women and 9 men (52.6 and 47.4%, respectively). The mean age in the diabetic group was 53.94 ± 2.84 years (range, 50—58 years). The mean age in the control group was 53.37 ± 4.49 years (range, 47—66 years). No significant statistical difference was found when comparing the age of both groups, *P* value = 0.646. The mean initial BCVA in the diabetic and in the control were 0.04 ± 0.07 logMar (range, 0—0.2) and 0.06 ± 0.07 logMAR (range, 0—0.2) respectively; no significant difference between both groups was assessed, *P* value = 0.418. None of the diabetic group participants were using insulin and the mean duration of diabetes diagnosis was 6.6 years ± 1.04 SD.

The mean baseline spherical equivalence was −0.43 ± 1.68 SD diopters for the diabetic group and −0.3 ± 1.73 SD diopters for the control group with a *P* value of 0.819when comparing the two means. Mean baseline IOP was measured, with a mean of 14.22 ± 2.16 SD mmHg for the diabetic group and 14.68 ± 2.47 SD mmHg for the control group; no significant difference between both groups was assessed (*P* = 0.24). The level of glycated hemoglobin was significantly different when comparing both groups with a mean HBA_{1c} level of 5.5% ± 0.55 for the control group and 6.4% ± 0.42 for the diabetic group (*P* < 0.001). The central retinal thickness (CRT) of all eyes was measured prior to testing with a mean CRT of 258.2 ± 13.6 μm for the diabetic group, and 263 ± 9.95 μm for the control group; no significant difference between both groups was assessed, *P* value = 0.205. To summarize, there was no significant intergroup difference with respect to the demographic variables and baseline characteristics was found, except for the HBA_{1c} level (Table 1).

We ensured the quality of OCT segmentation was thoroughly screened, and no significant artefacts were noted. Additionally, there was no evidence of subclinical macular edema, membranes, or traction in either group, with no statistical differences observed between the two groups in terms of segmentation quality with a *P* value = 0.586 (Table 1).

When comparing the GCL thickness between the diabetic group and the control group, a significant difference was found: the GCL thickness was reduced in the diabetic group in the superior, inferior, and nasal outer circles as shown in Table 2 with a *P* value < 0.001. There was a significant reduction of the total GCL average in the diabetic group compared to the control group with a *P* value = 0.003 (Table 2, Fig. 1).

As detailed in Table 3, regarding the pattern VEPs responses between the diabetic group without diabetic retinopathy and the control groups, we found that the amplitude level of the P100 wave was significantly reduced in the diabetic group with a *P* value < 0.05 for the pattern size of 60′ and 30′, but there was no significant intergroup difference regarding the amplitude of the P100 wave for the pattern size of 15′(Fig. 2). When comparing the latency of the P100 wave, we found that the diabetic group had a longer latency for the 15′ VEPs with a *P* value < 0.05 but this difference was not found when studying the latency of the P100 with check size of 60'and 30′(Fig. 3).

Eventually, when comparing the PERG responses (Table 4) between the diabetic group without diabetic retinopathy and the control group for the N35, P50 and N95 components, we found that the amplitude levels were significantly reduced in the diabetic group with a *P* value < 0.05 (Fig. 4). These differences were also significant when comparing the latencies of the PERG, as the diabetic group had longer latencies for the N35, P50 and N95 components with a *P* value < 0.05 (Fig. 5).

No statistically significant difference was observed in the OCT RNFL measurements between the two groups (Table 5).

Discussion

Our results demonstrated an important alteration in the amplitude and in the latency of the P100 wave of the pattern VEP in diabetic patients. We also assessed the alteration of

Table 2 Comparison of GCL thickness results of both groups.

Parameters	Control group	Standard Deviation	Diabetic group	Standard Deviation	*P* value
GCL Center (μm)	10.8421	1.26	11.6667	1.789	0.112
GCL Superior Inner circle (μm)	52.4737	4.325	50.2778	4.92	0.157
GCL Superior Outer circle (μm)	36.1579	3.48	32.0000	3.61	0.001
GCL Inferior Inner circle (μm)	51.3684	7.63	47.9444	6.92	0.162
GCL Inferior Outer circle (μm)	35.8947	5.27	29.8333	5.11	0.001
GCL Temporal Inner circle (μm)	47.5263	5.65	48.0556	5.31	0.771
GCL Temporal Outer circle (μm)	35.7895	5.06	37.5556	5.15	0.300
GCL Nasal Inner circle (μm)	45.6842	2.98	44.7222	3.21	0.351
GCL Nasal Outer circle (μm)	41.4737	5.25	35.2222	5.46	0.001

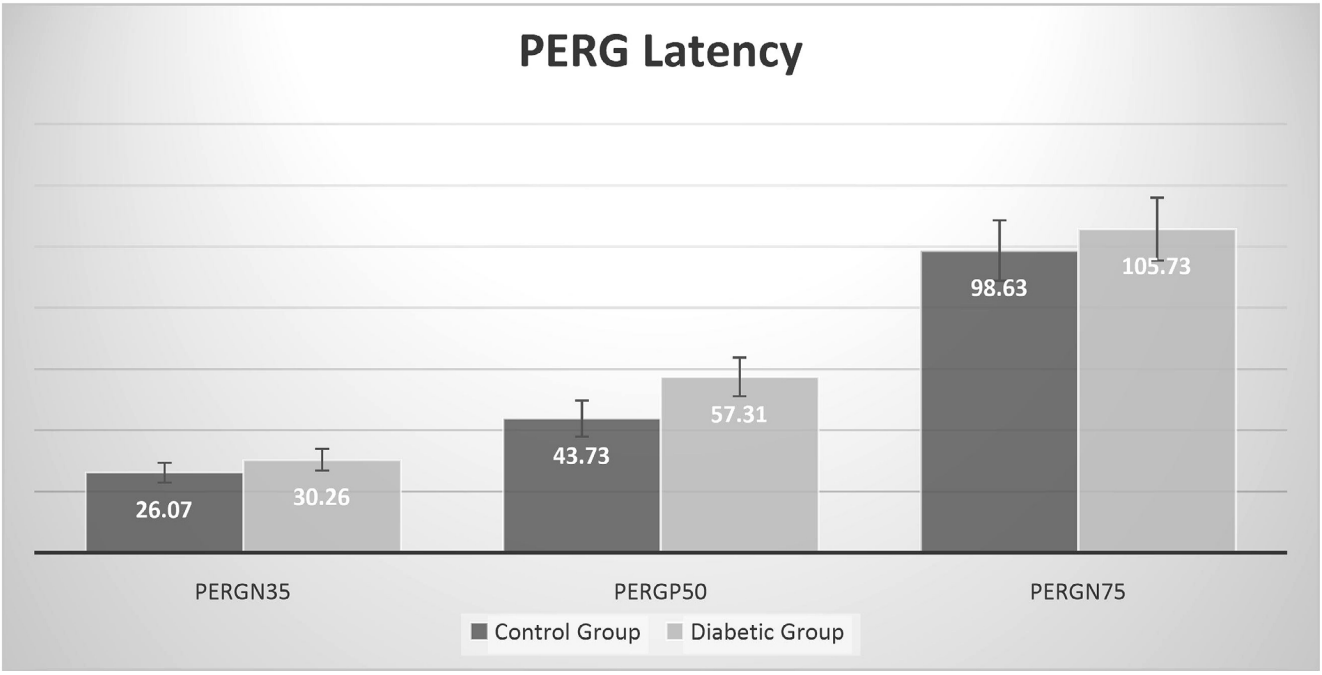

Figure 1. GCL thickness of both groups.

Table 3 Comparison of Pattern VEP results of both groups.

Variables	Control Group		Diabetic Group		*P* value
	Mean	Standard Deviation	Mean	Standard Deviation	
P100 Latency 60° Right Lobe in mS	110.06	6.66	109.79	10.34	0.925
P100 amplitude 60° Right Lobe in μV	13.82	4.49	7.23	3.17	0.0001
P100 Latency 30° Right Lobe in mS	111.32	9.54	114.39	8.27	0.304
P100 amplitude 30° Right Lobe in μV	12.95	3.65	9.45	4.05	0.009
P100 Latency 15° Right Lobe in mS	114.05	9.16	125.00	13.04	0.006
P100 amplitude 15° Right Lobe in μV	11.54	2.66	10.67	4.58	0.487
P100 Latency 60° Left Lobe in mS	109.80	7.31	108.57	10.74	0.684
P100 amplitude 60° Left Lobe in μV	14.42	6.74	7.71	3.54	0.001
P100 Latency 30° Left Lobe in mS	110.38	10.65	113.44	7.98	0.331
P100 amplitude 30° Right Lobe in μV	13.63	4.84	9.42	4.80	0.012
P100 Latency 15° Left Lobe in mS	112.89	9.60	124.18	11.78	0.003
P100 amplitude 15° Left Lobe in μV	11.47	3.04	12.04	6.40	0.729

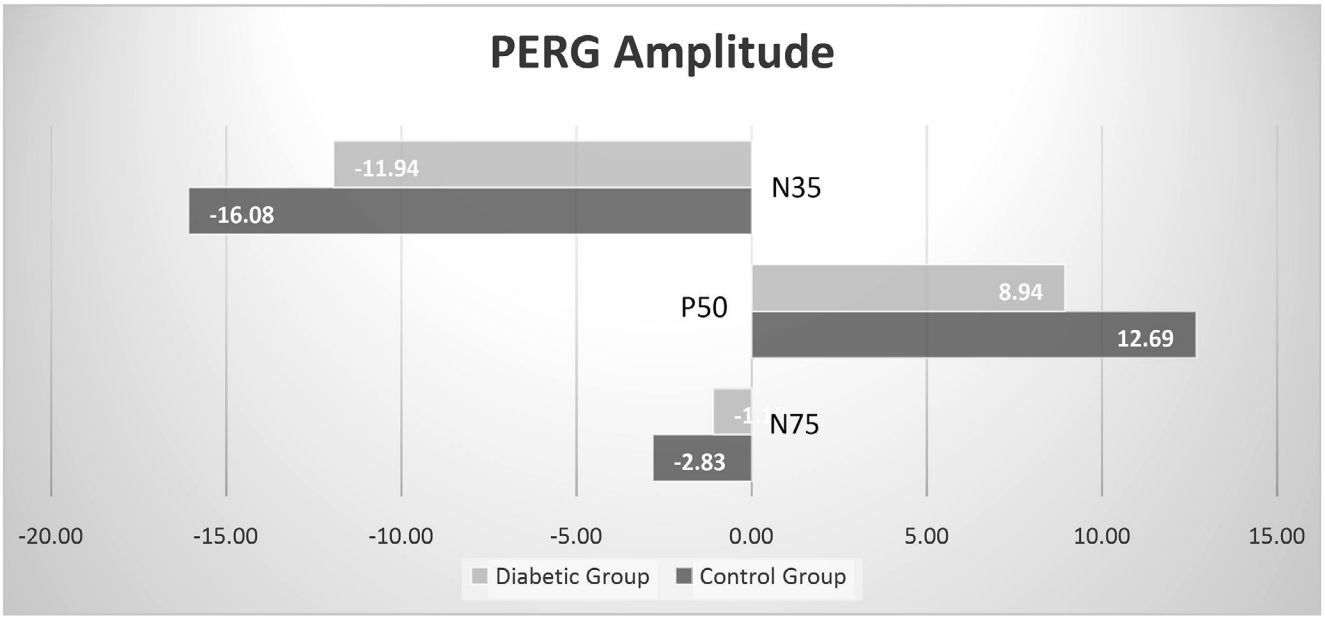

Figure 2. Pattern VEP amplitude of both groups.

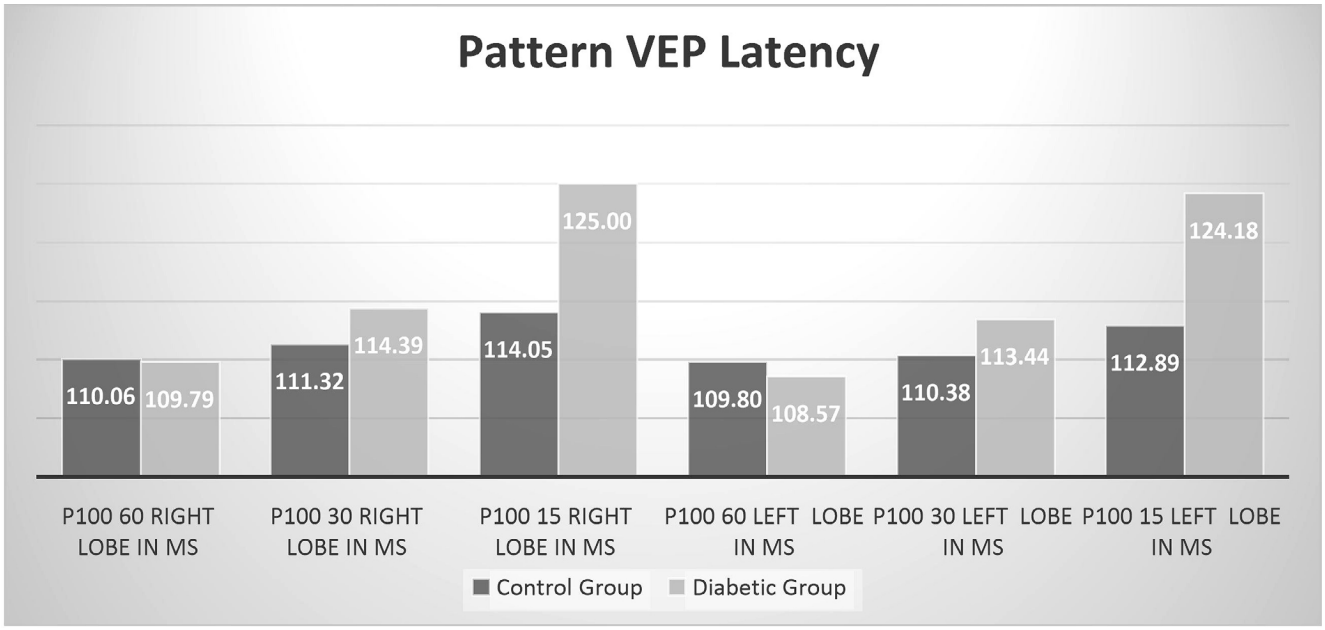

Figure 3. Pattern VEP P100 Latency of both groups.

Table 4 Comparison of PERG results of both groups.

Variables	Control Group		Diabetic Group		*P* value
	Mean	Standard Deviation	Mean	Standard Deviation	
PERGN35amplitude	−2.83	.51	−1.10	.83	0.0001
PERGP50amplitude	12.69	2.15	8.94	2.16	0.0001
PERGN75amplitude	−16.08	2.14	−11.94	2.56	0.0001
PERGN35	26.07	3.35	30.26	3.57	0.0001
PERGP50	43.73	5.88	57.31	6.35	0.0001
PERGN75	98.63	9.87	105.73	10.28	0.0048

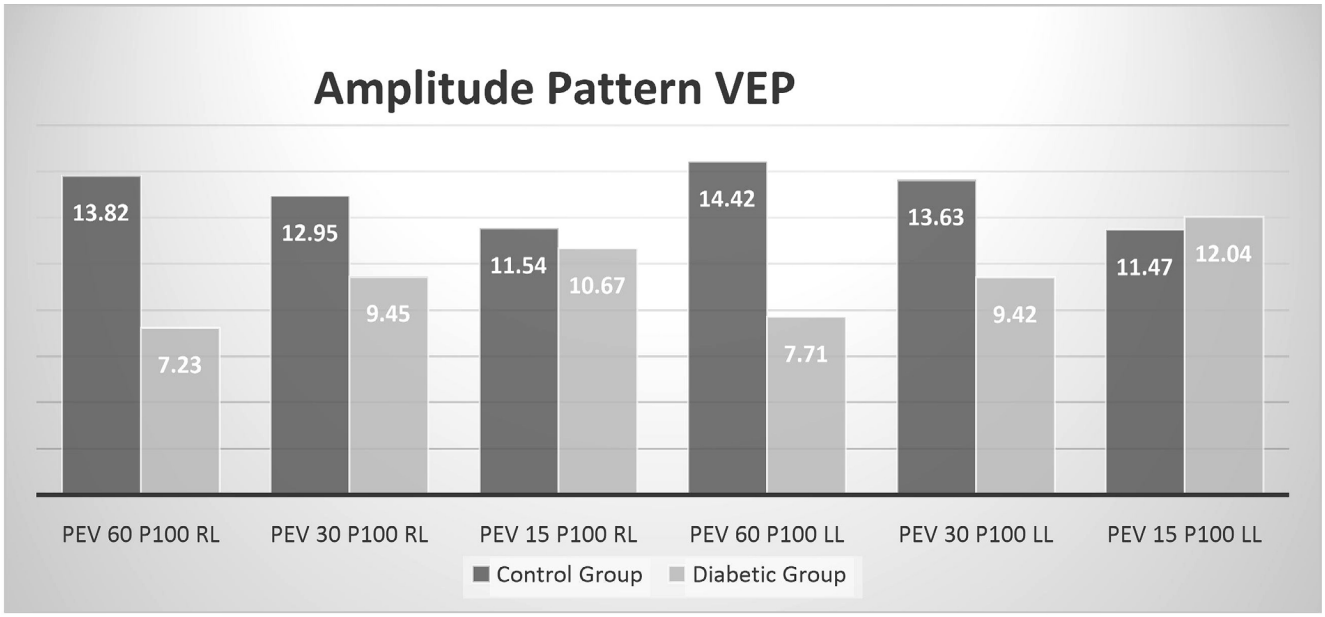

Figure 4. PERG amplitude of both groups.

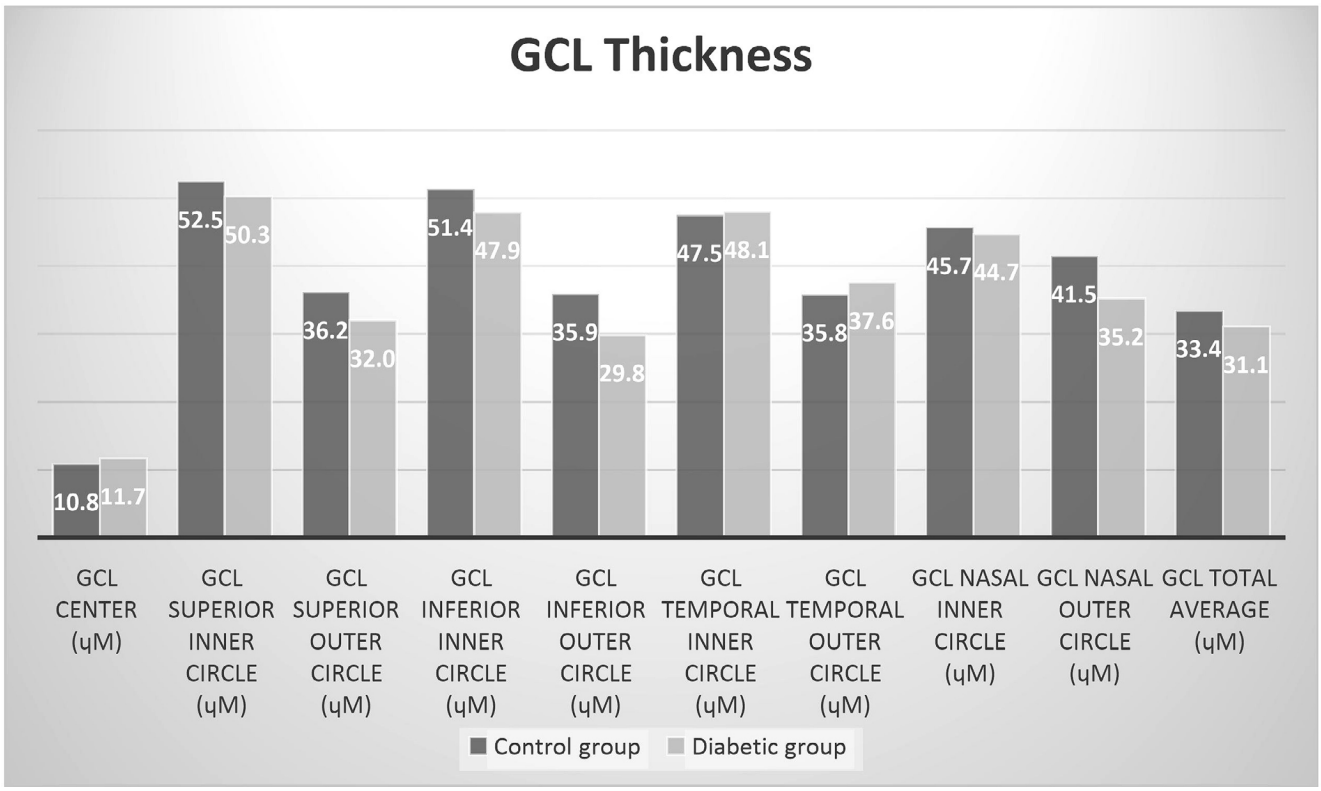

Figure 5. PERG Latency of both groups.

the function of the GCL using the Pattern ERG: all components of the PERG were altered in the diabetic group without a diabetic retinopathy.

The electrophysiological results of our study correlate with the previously published studies concerning these tests.

First, Pattern VEP as described earlier is a simple and an objective technique to study the visual pathway. Pattern VEP is interpreted by comparing the amplitude and the latency of the P100 wave to a control. A dysfunction of the optic nerve or of the GCL can alter the results of Pattern VEP, which emphasizes the importance of studying the results of Pattern VEP in diabetic patients to assess any neuro-retinal alteration prior to any retinal vascular changes [17]. The result of our study agrees with the results of Mariani et al.

Table 5 Comparison of RNFL results of both groups.

Parameters	Control group	Standard Deviation	Diabetic group	Standard Deviation	*P* value
RNFL Center (μm)	110.7500	15.32699	99.2000	14.43687	0.186
RNFL Supero nasal (μm)	141.0000	33.23653	127.2000	24.64161	0.365
RNFL Supero temporal (μm)	144.5000	30.16068	109.4000	31.74590	0.058
RNFL Temporal (μm)	85.5000	10.08299	76.0000	13.73613	0.258
RNFL Infero temporal (μm)	137.5000	35.93049	131.4000	21.77104	0.654
RNFL Infero nasal (μm)	147.0000	32.17660	136.5000	35.59332	0.637
RNFL Nasal (μm)	85.7500	6.89807	81.2000	14.82072	0.624

[18], Ponte et al. [19] and Corduneanu [20], as these studies reported an increase of the P100 latency in diabetic patients without retinopathy or in the diabetic patients with mild to moderate retinopathy sub-group in the case of Corduneanu et al. study. Heravian et al. [13] also found that the amplitude of P100 was reduced in the diabetic group without diabetic retinopathy compared to the control group. In a study performed by Collier et al. [21], it was found that VEPs were abnormal in diabetic patients with early peripheral neuropathy (P100 latency slightly greater) but were normal in the control group, the main limitation of this study was the small sample size. The VEP findings and the results found in the literature shows that early alteration of the diabetic retinopathy could be an alteration of GCL. In fact, this prolongation of P100 latencies could be the result of an early changes of the myelinated optic nerve fibers.

However, the results of the Pattern VEPs alone should be studied with caution because of the limitations of this test and the variability of this test. Indeed, the amplitude and the latency of Pattern VEPs can be altered by any abnormality on the visual pathway, and when testing Pattern VEPs a high rate of differences in the results between patients and on the same eye can be due to the recording conditions. That is why we emphasize the importance of using a Pattern ERG in association with a Pattern VEP to analyze properly the results of the tests.

In fact, the functionality of the retinal ganglion cells is well known to be studied using the pattern ERG. It is well established that the amplitude and the latency of the P50 and N95 of the PERG reflects the activity of the GCL [22]. The study of these parameters allows an evaluation of the activity of the neuro-retinal pathway and an alteration of this pathway in diabetic patients may be affected prior to and/or in the absence of any vascular retinal damage. The result of our study correlates with the results found in the literature: Caputo et al. studied 42 patients and they assessed a reduced amplitude of N95 in diabetic patients compared to control patients; moreover, the amplitude was inversely associated with the duration of diabetes [15]. Mermeklieva et al. [23] found a decrease of all the components of the Pattern ERG when studying a group of 84 patients with diabetes. These results confirm the GCL localization of the alteration found at an early stage in diabetic patients.

Furthermore, the GCL thickness results found in our study using the OCT confirm this hypothesis and are correlated with the result found in the literature. In fact, Carpineto et al. [6] found in their study a significant decrease of the GC-IPL thickness values in patients without diabetic retinopathy and in patients with minimal diabetic retinopathy, compared to control patients without diabetes. Similar data was found in a study by Ezhilvendhan et al. [24] and in a study by Rodrigues et al. [25].

In conclusion, the outcome of our study indicates that combining the study of Pattern VEP, PERG, and GCL thickness can be used as an early mean for the detection of retinal neuron function in diabetic eyes, as they can detect alterations occurring during the early course of diabetic retinopathy. The result of our study correlates with the results found in the literature about a potential functional neurodegenerative alteration which occurs very early in diabetic eyes before any anatomical documented diabetic retinopathy is assessed, though larger clinical trials are of necessity to confirm our findings.

Disclosure of interest

The authors declare that they have no competing interest.

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