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CLINICAL INVESTIGATION

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Visual evoked potentials after photostress in insulin-dependent diabetic patients with or without retinopathy

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Abstract Visual evoked potentials (VEPs) were assessed under basal conditions and after photostress in normal control subjects, in insulin-dependent diabetic patients with retinopathy (IDDPWR) and in insulin-dependent diabetic patients without retinopathy (IDDP). The VEPs recorded under basal conditions showed a P100 latency significantly higher in IDDP and IDDPWR eyes than in control eyes and in IDDPWR than in IDDP eyes ($P<0.01$). N75–P100 amplitude was significantly lower in IDDP and IDDPWR eyes than in control eyes ($P<0.01$). No difference was recorded in the N75-P100 amplitudes between IDDP and IDDPWR eyes. In all eyes, the VEPs recorded after photostress showed an increase in latency and a decrease in amplitude. In both IDDPWR eyes and IDDP eyes VEPs recorded at 20, 40 and 60 s after photostress showed higher mean increments in P100 latency than in C control eyes, and IDDPWR eyes showed higher mean increments in P100 latency than IDDP eyes (IDDP vs control $P<0.01$, IDDPWR vs control $P<0.01$, IDDPWR vs IDDP $P<0.017$). The mean reductions in amplitude observed at 20, 40 and 60 s after photostress in IDDP and IDDPWR eyes were lower than in control eyes (IDDP vs control $P=0.01$, IDDPWR vs control $P<0.01$, IDDPWR vs IDDP $P<0.01$). VEPs were superimposable on the basal VEP (recovery time) at 73.9 s in control eyes, at 88.17 s in IDDP eyes and at 113.3 s in IDDPWR eyes. VEPs after photostress in IDDP patients with normal visual acuity and no fluorangiographic signs of retinopathy may show multiple modifications. This may indicate the presence of an early functional deficiency of the central retinal layers.

Introduction

Diabetes mellitus is a complex disease, associated with systemic as well as ocular complications.

An objective method for evaluating visual function is to record the cortical potentials evoked by patterned stimuli (visual evoked potentials, VEPs) and/or electroretinographic signals (flash or pattern ERG). These methods have been widely used to assess visual function in diabetic patients [1, 2, 6, 9–12, 15, 26, 31, 33, 38].

Macular function can be studied using the macular photostress test (MPST) [4] that determines the period for recovery of visual acuity after dazzling the central retina with an ophthalmoscope [18, 36, 40]. The MPST has been also used to evaluate macular function in diabetic patients [28, 41].

Another method for evaluating macular function is to record VEPs after photostress. This test has been used in normal subjects [22], and in patients suffering for maculopathy [16] and glaucoma [29], but has never been used to evaluate macular function in diabetic patients.

Therefore, the aim of our work was to evaluate the ability of VEPs after photostress to disclose early impairment of macular function in diabetic patients without clinical signs of retinopathy.

Materials and methods

After giving informed consent 15 control subjects (mean age 30.3 ± 4.5 years) and 30 age-matched insulin-dependent diabetic patients (mean age 28.17 ± 7.27 years) were antered into the study. The following criteria were required for the control subjects: normal intraocular pressure (< 21 mmHg), normal visual acuity, normal visual field (Goldmann perimetry), and no ocular and/or neurological problems. The criteria required for the diabetic patients were: normal intraocular pressure (< 21 mmHg), best corrected visual acuity > 7/10, and absence of proliferative retinopathy as evaluated by fluoroangiography. This method allowed us to separate the diabetic patients into two groups according to Klein level [21]: patients without signs of retinopathy (IDDP, $n = 18$) (level 1), and patients with moderate or severe non-proliferative retinopathy (IDDPWR, $n = 12$) (levels 3–5). All the patients of the IDDP group had a 10/10 visual acuity.

The clinical characteristics of the patients are shown in Table 1.

VEP recording

The subjects were seated in a semi-dark acoustically isolated room. Prior to the experiment each subject was adapted to the ambient room light level for 10 min until their pupil diameter was about 3 mm. The display was surrounded by a uniform field of luminance 5 cd/m^2.

VEPs were recorded according to a previously described method [8, 29]. Briefly, the visual stimuli were checkerboard patterns (contrast 70%, mean luminance 110 cd/m^2) generated on a television monitor and reversed in contrast at a rate of two reversals per second. At the viewing distance of 114 cm the individual check size subtended 15' of visual arc and the screen of the monitor subtended 25° degrees. The test was performed on the right eye of all patients, with occlusion of the other eye.

Cup-shaped electrodes of silver–silver chloride were fixed with collodion in the Oz position (active electrode) and the Fpz position (reference electrode) in with the ground on the left arm. The interelectrode resistance was kept below 3 kΩ. The bioelectric signal was amplified (gain 20 000), filtered (bandpass 1–100 Hz) and averaged, with automatic rejection of artifacts, over a number of stimulus periods using a Cadwell 7400 (Polman, Bologna, Italy).

The recording session began with a preliminary experiment in which at least two VEPs were recorded, averaging over 100 stimulus periods and excluding artifacts. The analysis time was 500 ms. The transient response was characterized by several waves with three peaks that in normal subjects appeared after 75–100 and 145 ms. These peaks had negative (N75), positive (P100) and negative (N145) polarity, respectively.

After this preliminary trial, a control VEP was recorded reducing the averages to 40 stimuli per trial (with no more than two sweeps discarded because of artifacts). This VEP record was defined as 'basal' and was kept displayed on the computer monitor.

Photostress was then induced for 30 s by means of a circular diffusing surface (the bulb of a 200 W lamp) that was centrally fixated by the subject from a distance of 20 cm and produced a central scotoma of 6° diameter. The pupil diameter reduced to about 2 mm. Immediately after the end of photostress, fixation was shifted to the pattern stimulus and recording of VEPs started. Recordings were taken for successive 20-s periods (averaging 40 stimuli every 20 s) and displayed successively on the monitor until the VEP obtained was superimposable on the basal recording. The time taken for the VEP to become superimposable was considered as the recovery time after photostress (RT).

For all VEPs the peak latency and the peak amplitude of each of the waves were measured directly from the displayed recordings with a pair of cursors. Our method did not allow us to record in the same averaged run the pattern ERG or the focal ERG as well as the VEP. These two ERG recordings require a longer time to obtain a reliable recording than the preestablished recording time required by our experimental procedure.

Statistics

Results are expressed as mean ± SD. If not otherwise indicated n refers to the number of eyes. Differences between groups were evaluated using a one-way analysis of variance for repeated measures (ANOVA) and considered significant with $P < 0.05$.

Results

Basal VEPs

The mean data for the three groups are shown in Table 2.

In control eyes, the VEP parameters (P100 latency and N75–P100 amplitude) were within our normal limits [35] expressed as mean value ± SD for N75–P100 amplitude (8.98 ± 2.66 μV) and mean value ± 3 × SD for P100 latency (95.15 ± 4.15 ms). P100 latency was significantly higher in IDDP and IDDPWR eyes than in control eyes and was significantly higher in IDDPWR than IDDP eyes. N75–P100 amplitude was significantly lower in IDDP and IDDPWR eyes than in control eyes. No

Table 1 Clinical characteristics of control subjects and patients (*IDDP* insulin-dependent diabetic patients, *IDDPWR* insulin-dependent diabetic patients with retinopathy, *n* number of patients, Hb_{A1c} glycosylated haemoglobin)

Group	No. of eyes	Age (years)	Disease duration	Hb_{A1c}
Controls ($n = 15$)	15	30.3 ± 4.5	–	–
IDDP ($n = 18$)	18	27.8 ± 7.5	11.5 ± 5.2	7.4 ± 1.1
IDDPWR ($n = 12$)	12	28.6 ± 7.1	15.3 ± 5.1	7.6 ± 1.5

Table 2 Mean and standard errors of VEP parameters. Values are mean ± SD (*P100* latency peak P100 in basal recording (ms), *N75–P100* peak-to-peak amplitude N75–P100 (μV) in basal recording, *IDDP* insulin-dependent diabetic patients, *IDDPWR* insulin-dependent diabetic patients with retinopathy)

Group	No. of eyes	P100	N75–P100
Controls	15	93.15 ± 3.55	9.18 ± 2.18
IDDP	18	106.07 ± 8.54*	5.90 ± 2.12*
IDDPWR	12	119.66 ± 11.5*/°	5.08 ± 1.31*

* $P < 0.01$ vs control, */° $P < 0.01$ vs IDDP

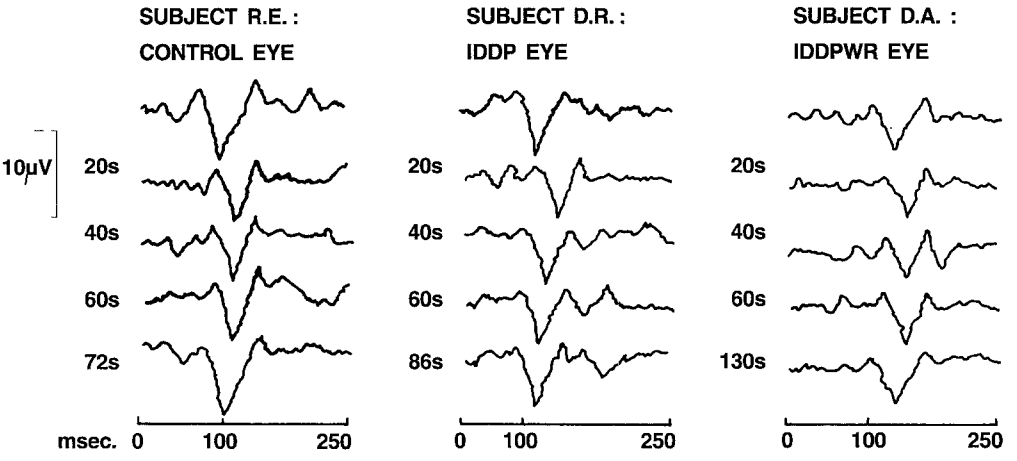

Fig. 1 VEP layout of subject R.E. (control), and patients D.R. (IDDP) and D.A. (IDDPWR) under the normal basal condition and 20, 40 and 60 s after photostress. The recordings from both patients at 20, 40 and 60 s after photostress show a longer P100 latency and a reduced amplitude compared with the recordings from the control subject. The VEPs are superimposable on the basal waveform at 72 s for the control eye, at 86 s for the IDDP eye and at 130 s for the IDDPWR eye

difference was recorded between IDDP and IDDPWR eyes.

VEPs after photostress

Control eyes

Examples of recordings from a normal subject (R.E.) are shown in Fig. 1. The mean P100 latency and N75–P100 amplitude results are presented in Tables 2 and 3 and in Fig. 2 and 3.

At 20 s after photostress we observed an increase in P100 latency and a decrease in N75–P100 amplitude. At 40 and 60 s after photostress the P100 latencies were shorter than the 20-s value, but still longer than the basal P100 latency. The N75–P100 amplitude increased from the value observed at 20 s, but it was still lower than the basal value. The RT was 73.07 + 2.81 s.

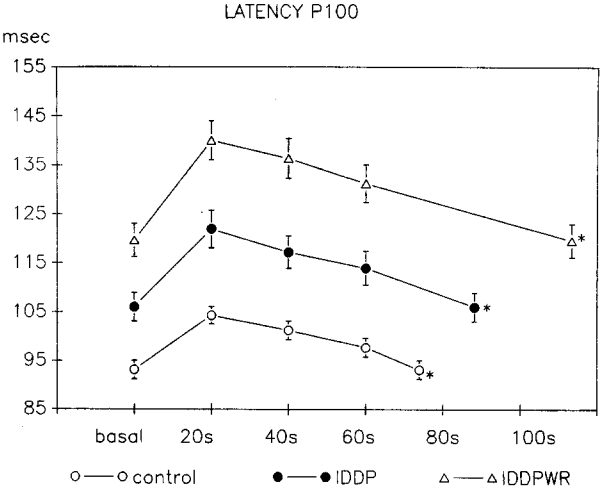

Fig. 2 Graphic representation of mean values of latency P100 under the basal condition and 20, 40, 60, 80, 100 and 120 s after photostress. Error bars represent one standard error of the mean. Recovery time after photostress (*) is 73.92 s for the control eyes, 88.17 s for the IDDP eyes and 113.3 s for the IDDPWR eyes (o control, ● IDDP, △ IDDPWR)

IDDP and IDDPWR eyes

Examples of recordings from an IDDP patient (D.R.) and from an IDDPWR patient (D.A.) are shown in Fig. 1. The mean results are presented in Tables 2 and 3 and in Fig. 2 and 3.

At 20, 40 and 60 s from photostress we found the same changes in the patients' eyes as in the control eyes. The VEPs recorded at 20 s after photostress showed P100 peaks of longer latency and smaller amplitude than the basal recordings. At 40 s and 60 s from photostress the 100 latency progressively diminished, although it was longer than in the basal VEPs. The amplitude progressively increased, but remained lower than in the basal VEPs.

Photostress induced similar P100 latency and N75–P100 amplitude response curves in control eyes and in eyes of both patient groups. However, the mean increments in P100 latency observed at 20, 40 and 60 s after photostress in IDDP and IDDPWR eyes were higher than in control eyes and higher in IDDPWR eyes than in IDDP eyes (Table 3) (IDDP vs control: $F(1,97) = 8.40$, $P < 0.01$; IDDPWR vs control $F(1,79) = 36.33$, $P < 0.01$; IDDP vs IDDPWR $F(1,88) = 5.97$, $P = 0.017$). Amplitudes observed at 20, 40 and 60 s after photostress were more reduced in IDDP and IDDPWR eyes than in control eyes and in IDDPWR eyes than in IDDP eyes (Table 3) (IDDP vs control $F(1,97) = 19.34$, $P < 0.01$; IDDPWR vs control $F(1,79) = 49.13$, $P < 0.01$; IDDPWR vs IDDP $F(1,88) = 11.08$, $P < 0.01$).

RT was significantly higher in IDDP and IDDPWR eyes than in control eyes (88.17 ± 10.48, 113.33 ± 12.93 and 73.92 ± 2.69 s, respectively; $P < 0.01$ in both groups)

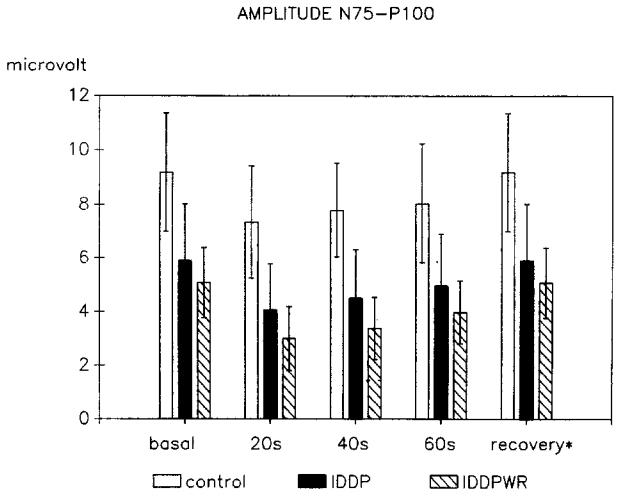

Fig. 3 Histograms of mean values of VEP amplitude under the basal condition and 20, 40 and 60 s after photostress. The vertical lines represent one standard deviation. Recovery time after photostress (*) is 73.92 s for the control eyes, 88.17 s for the IDDP eyes and 113.3 s for the IDDPWR eyes (□ control, ■ IDDP, ▨ IDDPWR)

Table 3 Mean increase in P100 latency and mean percentage decrease in N75–P100 amplitude at 20, 40 and 60 s after photostress. Values are mean ± SD (*IDDP* insulin-dependent diabetic patients, *IDDPWR* insulin-dependent diabetic patients with retinopathy)

	P100 latency increase (ms)	N75–P100 amplitude decrease (%)
20 s		
Controls	11.20 ± 2.89	20.56 ± 9.91
IDDP	15.40 ± 8.55	32.44 ± 8.67
IDPWR	19.54 ± 6.46	41.49 ± 9.16
40 s		
Controls	8.14 ± 2.92	14.81 ± 5.29
IDDP	11.25 ± 6.22	24.62 ± 7.97
IDDPWR	15.88 ± 7.42	33.63 ± 9.68
60 s		
Controls	4.59 ± 2.76	10.10 ± 9.00
IDDP	8.05 ± 5.88	16.87 ± 7.30
IDDPWR	10.80 ± 4.36	21.92 ± 6.31

and significantly higher in IDDPWR eyes than in IDDP eyes ($P < 0.01$).

Discussion

Both IDDP and IDDPWR eyes showed alterations in basal VEPs with P100 latencies higher and N75–P100 amplitudes lower than in control eyes.

An influence of retinopathy on the changes from the basal VEPs recorded in IDDPWR eyes may be excluded by the fact that the same changes were present in IDDP eyes. The delay in VEP latency and the reduction in amplitude observed in IDDP and IDDPWR eyes may be attributed to a reduced velocity of nervous conduction in the optic nerve. This hypothesis can be supported by studies that have shown a reduction in the amplitude of the pattern ERG, which is not related to the presence of retinopathy, suggesting that the innermost retinal layers (ganglionar cells and their axons [20, 23, 25]) of the central retina are selectively and at an early stage affected by diabetes [15].

In addition, correlations between VEP abnormalities, peripheral neuropathy and reduced central conduction velocity have been observed [13, 14, 32] and histological studies [34] have indicated the presence of optic neuropathy secondary to axonal degeneration consequent upon dysfunction of the ganglion cell body [27]. However, the influence of retinopathy cannot be excluded. Indeed, IDDPWR eyes showed a higher P100 latency than control and IDDP eyes.

Information on the functional status of the central retina was obtained from VEPs recorded after photostress. In control eyes the dazzling of the macular region induced an increase in latency and a decrease in amplitude of the VEPs, and functional recovery always occurred within 80 s. The changes induced by photostress on the VEP can generally be attributed to the diminished capacity of macular photoreceptors to produce a sufficient electronic potential after dazzling. The VEP recovery to its basal state after photostress depends on the resynthesis of photopigments, a process for which an adequate blood flow seems to be essential [16]. This appears to be confirmed by the fact that in patients with carotid occlusive disease VEP recovery time is increased [5].

However, Bucci et al. [8] and Parisi and Bucci [29] observed in normal subjects in whom the intraocular pressure had been artificially increased and in patients with ocular hypertension or glaucoma that the recovery time after photostress was longer than in normal subjects. They suggest a possible functional role of the inner retinal layers of the central retina in the recovery of VEP after photostress.

In IDDP and IDDPWR eyes photostress caused increases in P100 latency and decreases in N75–P100 amplitude, and the RT was also greater in the patient groups. The pathogenesis of these alterations in VEP parameters after photostress is not clearly understood, but these abnormalities could be attributed to a reduced functionality of the photoreceptor layer and/or the ganglion cell layer of the macular region. The involvement of the sensory layer of the retina has been implicated by several studies [7, 19, 30, 37, 39] that showed a modification of flash ERG in diabetic patients. The flash ERG reflects the activity of the outer layers of the whole retina [3] and the contribution of the macular region to the flash is negligible.

Using the focal ERG, a more sensitive method for studying macular function, Ghirlanda et al. [18] have found that early diabetes causes selective neurosensory deficits of the inner retinal layers, whereas the photoreceptors appear unaffected.

On the basis of our previous observations [8, 21, 29] the present results could be interpreted as a consequence of a reduced function of the inner retinal layers of the central retina, and indeed an influence of the outer retinal layers cannot be excluded.

VEPs after photostress in IDDP with normal visual acuity and no fluorangiographic signs of retinopathy may present multiple modifications. This may suggest the presence of an early functional deficiency of the central retinal layers. The test results are also abnormal in IDDPWR. In these patients the utility of the test is in revealing the presence of macular function impairment in contrast to the fluorangiographic signs which give morphological information.

We do not know the influence of duration of disease and metabolic control on VEPs after photostress. Studies are in progress to evaluate the influence of these parameters.

References

1. Algan M, Ziegler O, Gehin P, Got I, Raspiller A, Weber M, Genton P, Saudax E, Drouin P (1989) Visual evoked potentials in diabetic patients. Diabetes Care 12:227–229
2. Arden GB, Hamilton AMP, Wilson-Holt J, Ryan S, Yudkin JS, Kurtz A (1986) Pattern electroretinograms become abnormal in the preproliferative stage: possible use as a screening test. Br J Ophthalmol 70:330–335
3. Armington JC (1974) The electroretinogram. Academic Press, New York
4. Baillart JP (1954) L'examen functionel de la macula. Rapport à la Societè d'Ophthalmologie de Paris. Bull Soc Ophtalmol Fr [Suppl] 4:I–LXVII
5. Bianchini E, Franchi A, Manni R, Villani LG, Cordella M, Botta GC (1987) Carotid occlusive disease: an electrophysiological macular investigation. J Cardiovasc Surg 28:524–527
6. Boschi MC, Frosini R, Menicucci R, Sodi A (1989) The influence of early diabetes on pattern electroretinogram. Doc Ophthalmol 71:369–374
7. Bresnik GH, Palta M (1987) Temporal aspects of the electroretinogram in diabetic retinopathy. Arch Ophthalmol 105:660–664
8. Bucci MG, Parisi V, Giannini R, Rossini PM (1991) Recordings of visual evoked potentials after photostress in artificially increased intraocular pressure. Clin Vis Sci 6:431–436
9. Cirillo D, Gonfiantini E, De Grandis D, Bongiovanni L, Robert JJ, Pinelli L (1984) Visual evoked potentials in diabetic children and adolescents. Diabetes Care 7:273–275
10. Collier A, Mitchell JD (1985) Visual evoked potentials and contrast sensitivity function in diabetic retinopathy. BMJ 291:248
11. Comi G, Martinelli V, Galardi G, Medaglini S, Poggi A, Beccaria L, Meschi F, Flores D'Arcais A (1986) Visual evoked potentials in diabetic teenagers: influence of metabolic control and relationship with peripheral neuropathy. Methods Pediatr Systems Ophthal 9:85–87
12. Comi G, Martinelli V, Galardi G, Medaglini S, Beccaria L, Meschi F, Rosti L, Bressani N, Chiumello G (1987) Evaluation of central nervous conduction by visual evoked potentials in insulin-dependent diabetic children. Metabolic and clinical correlation. Acta Diabetol Lat 24:157–162
13. Cracco J, Castells S, Mark E (1985) Spinal somatosensory evoked potentials in juvenile diabetes. Ann Neurol 15:55–58
14. Donald MW, Williams-Erdahl DL, Surridge DHC, Monga TN, Lawson JS, Bird CE, Latemendia FJJ (1984) Functional correlation of reduced central conduction velocity in diabetic subjects. Diabetes 33:627–633
15. Falsini B, Porciatti V, Scalia G, Caputo S, Minnella A, Di Leo MAS, Ghirlanda G (1989) Steady-state electroretinogram in insulin-dependent diabetics with no or minimal retinopathy. Doc Ophthalmol 73:193–200
16. Franchi A, Magni R, Lodigiani R, Cordella M (1987) Vep pattern after photostress: an index of macular function. Graefe's Arch Clin Exp Ophthalmol 225:291–194
17. Franzone M, Brunetti GM, Coggi G, Peronzini S (1985) Test del tempo di recupero maculare dopo abbagliamento: attendibilità dell'esame. Boll Oculistica 64 [Suppl 11/12]:141–151
18. Ghirlanda G, Di Leo MAS, Caputo S, Falsini B, Porciatti V, Marietti G, Greco AV (1991) Detection of inner retina dysfunction by steady-state focal electroretinogram pattern and flicker in early IDDM. Diabetes 9:1122–1127
19. Gjotteberg M (1974) The electroretinogram in diabetic retinopathy. A clinical study and critical survey. Acta Ophthalmol 52:521–533
20. Hollander H, Bisti S, Maffei L, Hebel R (1984) Electroretinographic responses and retrograde changes of retinal morphology after intracranial optic nerve section. Exp Brain Res 55:483–494
21. Klein BEK, Davis MD, Segal P (1984) Diabetic retinopathy: assessment of severity and progression. Ophthalmology 91:10–17
22. Lovasik JV (1983) An electrophysiological investigation of the macular photostress test. Invest Ophthalmol Vis Sci 24:437–441
23. Maffei L, Fiorentini A (1981) Electroretinographic responses to alternating gratings before and after section of the optic nerve. Science 211:953–955
24. Maffei L, Fiorentini A (1982) Electroretinographic responses to alternating gratings in the cats. Exp Brain Res 48:327–334
25. Maffei L, Fiorentini A, Bisti S, Hollander H (1985) Pattern ERG in the monkey after section of the optic nerve. Exp Brain Res 59:423–425
26. Martinelli V, Merenda M, Natali-Sora MG, Meschi F, Beccaria L, Comi G (1987) Correlation between pattern electroretinopathy and visual evoked potentials in diabetes. Electroenceph Clin Neurophysiol 66:S64
27. Martinelli V, Filippi M, Meschi F, Pozza G, Canal N, Comi GC (1991) Electrophysiological study of optic pathways in insulin dependent diabetes mellitus. Clin Vis Sci 6:437–443
28. Mosci C, Polizzi A, Grillo N, Capris P, Zingirian M (1986) Ottimizzazione del test del recupero maculare nello studio dei soggetti diabetici. Boll Oculistica 65:347–356

29. Parisi V, Bucci MG (1992) Visual evoked potentials after photostress in patients with primary open-angle glaucoma and ocular hypertension. Invest Ophthalmol Vis Sci 33:436–442
30. Parisi V, Uccioli L, Monticone G, Carboni A, Falleni C, Ignagni T, Del Giudice R, Bucci MG (1994) Registrazioni di potenziali evocati visivi e dei potenziali oscillatori dell'elettroretinogramma fotopico in pazienti diabetici insulino-dipendenti con e senza retinopatia. Riv Med Militare (in press)
31. Porciatti V, von Berger GP (1983) Pattern electroretinogram and visual evoked potentials in optic nerve disease: early diagnosis and prognosis. Doc Ophthalmol Proc Ser 40:117–126
32. Pozzessere G, Rizzo PA, Valle E, Mollica MA, Sanarelli L, Morano S, Pietravalle P, Di Mario U, Morocutti C (1989) A longitudinal study of multimodal evoked potentials in diabetes mellitus. Diabetes Res 10:17–20
33. Puvanendran K, Davethasan G, Wong PK (1983) Visual evoked responses in diabetes. J Neurol Neurosurg Psychiatry 46:543–547
34. Reske-Nielsen E, Lundbaek K, Rafaelsen OJ (1965) Pathological changes in the central and peripheral nervous system of young long-term diabetics. I. Diabetic encephalopathy. Diabetologica 1:223–241
35. Rizzo P, Carboni M, Passaro R, Parisi V, Del Giudice R, Rizzo A, Terrana P (1988) Potenziali evocati visivi da checkerboard pattern reversal: dati normativi. Riv Med Aeronautica Spaziale 2:99–105
36. Severin SL, Tour R, Kershaw H (1967) Macular function and the photostress test. Arch Ophthalmol 77:163–167
37. Simonsen SE (1975) Prognostic value of ERG (oscillatory potentials) in juvenile diabetics. Acta Ophthalmol [Suppl] 123:223–224
38. Trick GL, Burde RM, Gordon MO, Kilo C, Santiago JV (1988) Retinocortical conduction time in diabetics with abnormal pattern reversal electroretinogram and visual evoked potentials. Doc Ophthalmol 70:19–28
39. Van Der Torren K, Van Lith G (1989) Oscillatory potentials in early diabetic retinopathy. Doc Ophthalmol 71:375–379
40. Zingirian M, Castellazzo R, Trillo T (1968) Test del recupero maculare in soggetti normali. Standardizzazione del metodo. Boll Oculistica 47:883–848
41. Zingirian M, Polizzi A, Grillo N (1985) The macular recovery test after photostress in normal and diabetic subjects. Acta Diabetol Lat 22:169–172