PANMINERVA MED 2013;55(Suppl. 1 to No. 1):1-3

Retinal electrofunctional alterations in patients affected by juvenile diabetes

R. MALAGOLA [1], L. ARRICO [1], G. BELCARO [2], F. LENARDUZZI [1], M. NEBBIOSO [1]

Aim: **The purpose of the study was to evaluate the internal retinal layers functionality and to document the correlations with metabolic control parameters in patients affected by diabetes mellitus type I free.**

Materials and methods: **A group of 40 patients affected by insulin dependent diabetes mellitus type 1 were examined by means of the scotopic threshold response (STR) and oscillatory potentials (OPs). The results were compared with the data obtained from a group of 20 healthy subjects.**

Results: **Statistically significant correlations have been identified between the STR values and the levels of glycosylated haemoglobin and glycemia at the time of the exam.**

Conclusion: **The results show the presence of a retinal functionality alteration located mainly in the internal retinal layers. This level generates the STR response which is produced specially by the amacrine cells that are sensible to the damage of the retinal microcirculation.**

KEY WORDS: Electroretinogram (ERG) - Insulin dependent diabetes mellitus - Juvenile diabetes - Oscillatory potentials (OPs) - Scotopic threshold response (STR).

We evaluated the retinal functionality of patients affected by diabetes mellitus type I free from retinal and general complications caused by the disease.

The aim was to document functional alterations of the internal retinal layers, with particular attention for the amacrine cells, that according to the literature[1-3] are involved in the regulation of the microcirculation. To perform this evaluation we used to electroretinographic examinations: the Scotopic Threshold Response (STR) and the Oscillatory Potentials (OPs).

Corresponding author: Marcella Nebbioso, MD, Department of Sense Organs, Centre Ocular Electrophysiology, Sapienza University of Rome. viale del Policlinico 155, 00161, Rome, Italy.
E-mail: marcella.nebbioso@uniroma1.it.

[1]Department of Sense Organs
Sapienza University of Rome, Italy.
[2]Irvine3 Labs Department of Biomedical Sciences
G. D'Annunzio University of Chieti- Pescara, Italy.

Materials and methods

This research was a supportive care study and was conducted on 40 patients with type I diabetes including 20 males and 20 females, mean age 16.3 ± 2.5 years SD (ranging from 10 to 18 years). In accordance with the Helsinki Declaration, the patient was informed about the use of your data and signed an informed consent. The study protocol was approved by the Ethical Committee, Sapienza University of Rome.

Inclusion criteria were as follows:

— Absence of complications due to diabetes or other systemic diseases;

— Absence of ocular alterations as glaucoma, cataract, maculopathy, diabetic retinopathy, trauma, previous intraocular surgery, and amblyopia;

— Refraction values between ± 3D sphere and ± 1 D cylinder;

— Best-corrected visual acuity (BCVA) for far distance up to 0.1 logMAR (20/25 Snellen) in either eye.

A comprehensive eye examination included BCVA for far and near vision, slit lamp biomicroscopy, intraocular pressure measurement with Goldmann applanation tonometry, and dilated fundus examination.They therefore underwent the testing of STR and OPs.

Electro-functional tests

A *full-field ERG* was performed with the Vision monitor MonPack 120 Metrovision (Pérenchies,

TABLE I.—*Statistical comparison between patients and controls (p < 0.001 and < 0.02).*

Values	Patients Mean SD	Controls Mean SD	P value
STR Amplitude (μV)	10.2 ± 4.35	20.5 ± 2.27	0.001
STR Latency (ms)	152.25 ±12.6	145.3 ± 7.85	0.001
OPs Amplitude/Latency	2.57 ± 0,56	2.02 ± 0.47	0.02

France) according to ISCEV[4] reference procedures. The ERG was registered using: ERG-jet contact lens electrodes to detect signals, skin electrodes on the outer corner of the eye as reference, and an ear clip as neutral electrode. Prior to the exam, patient's pupils were dilated using tropicamide 1% to induce maximal pharmacologic mydriasis in the selected patients and they were adapted to the dark for 30 minutes.

We applied ERG-jet electrodes to the eyes of the patients in a Faraday cage with a low intensity red light, so not to interfere with the dark adaptation.

The parameters we considered were:

— STR: maximum amplitude obtained in the STR and its latency;

— OPs: ratio between the sum of the amplitudes and the sum of the latencies (A/L) of the OPs.

The results were compared with the data obtained from a group of 20 healthy subjects of mean age 18,3 ± 2,15 years and statistically analyzed thanks to the two tailed Students t test for paired samples.

Results

In the examined patients the STR values showed the following pattern:

1) Reduction in amplitude and increase in latency of the STR wave (Table I);

2) Slight reduction of the amplitude/latency ratio in the OPs (Table I).

Statistically significant correlations have been identified between the STR values and the levels of glycemia and of glycosylated haemoglobin at the time of the exam (Figures 1, 2, and 3). No statistically significant correlations have been detected be-

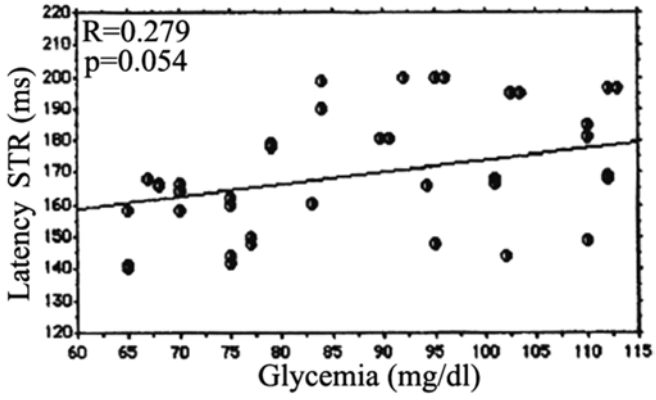

Figure 2.—Graphic representation of the regression lines related to STR latency and glycemia.

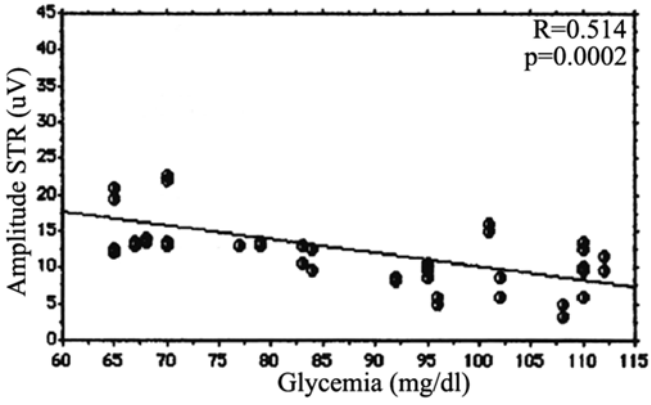

Figure 1.—Graphic representation of the regression lines related to STR amplitude and glycemia.

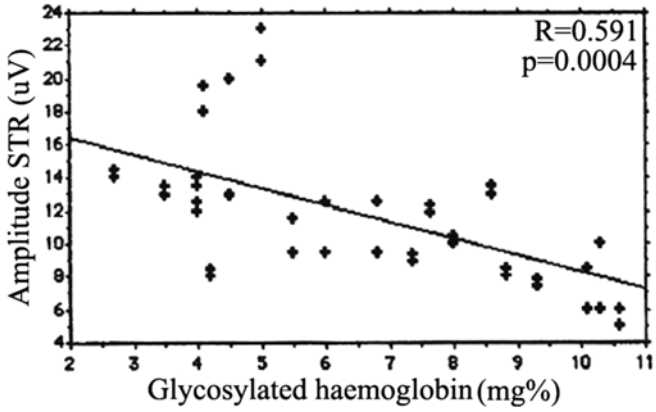

Figure 3.—Graphic representation of the regression lines related to STR amplitude and glycosylate haemoglobin.

tween the latencies of STR and the values of HbA_1, a glycosylated haemoglobin.

Discussion and conclusion

The results clearly show the presence of a retinal functionality alteration located mainly in the internal retinal layers. This level generates the STR response which is produced specially by the amacrine cells. The functional alteration of the amacrine cells is related to the metabolic control parameters and so their activity can be modified by damage of the retinal microcirculation.[1-3, 5] Therefore an alteration of the STR reflects a malfunctioning of the amacrine cells. The altered regulation of the microcirculation would cause initial changes in the retinal trophism, also revealed by the reduction of the OPs amplitude/latency ratio.[5]

The metabolic alteration damages particularly the amacrine cells that, losing the capacity to regulate retinal microcirculation, favors alterations of the cellular trophism. The persistence of this situation can establish a vicious circle that is able to lead to the anatomical alterations typical of the full blown retinopathy.

References

1. Ye X, Laties AM, Stone RA. Peptidergic innervations of the retinal vasculature and optic nerve head. Invest Ophthalmol Vis Sci 1990;31:1731-1737.
2. Aylwas GW. The scotopic threshold response in diabetic retinopathy. Eye 1989;3:626-637.
3. Nelson R, Kolb H. Amacrine cells in scotopic vision. Ophthalmoc Res 1984:16:21-26.
4. Marmor MF, Fulton AB, Holder GE, Miyake Y, Brigell M, Bach M: Standard for clinical electroretinography (2008 update). Doc Ophthalmol 2009;118:69-77.
5. Nebbioso M, Federici M, Rusciano D, Evangelista M, Pescosolido N. Oxidative stress in pre-retinopathic diabetic subjects and antioxidants. Diabetes Technology & Therapeutics. 2012; 14(3):257-63. DOI: 10.1089/dia.2011.0172.

Conflicts of interest.—The Authors certify that there is no conflict of interest with any financial organization regarding the material discussed in the manuscript. All the Authors contributed equally to this work.