
# Anomalie dei circuiti post-retinici nei pazienti glaucomatosi

**Vincenzo Parisi**
IRCCS Fondazione G.B. Bietti, Roma

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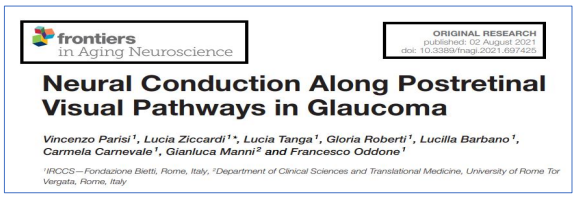
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ORIGINAL RESEARCH
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**Neural Conduction Along Postretinal Visual Pathways in Glaucoma**

*Vincenzo Parisi[1], Lucia Ziccardi[1]*, Lucia Tanga[1], Gloria Roberti[1], Lucilla Barbano[1], Carmela Carnevale[1], Gianluca Manni[2] and Francesco Oddone[1]*

[1]IRCCS—Fondazione Bietti, Rome, Italy, [2]Department of Clinical Sciences and Translational Medicine, University of Rome Tor Vergata, Rome, Italy

## AIM

our study aimed to evaluate the neural conduction along the postretinal large and small axons (assessed by RCT in response to 60′ and 15′ checks) and whether it could be related to RCG function (assessed by PERG recordings) and/or could be dependent or not from the RNFL morphological condition in OAG eyes.

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# Patients

Thirty-seven consecutive patients affected by OAG were recruited and selected from a larger population of 274 patients based on the following inclusion criteria:

(2) (MD) between −2 and −20 dB.

(5) intraocular pressure (IOP) values less than 18 mmHg under topical hypotensive treatment (monotherapy as well as combined therapy) during, at least, 8 months preceding the electrophysiological and morphological evaluation.

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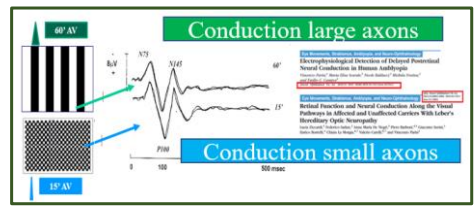
# Methods

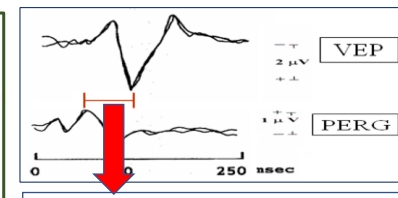
**1) Neural conduction**

VEP

+

PERG

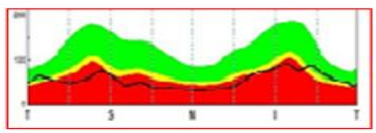



**"Retinocortical Time"**
difference VEP P100- PERG P50 ITs
**electrophysiological index of neural conduction between ganglion cells and the visual cortex)**

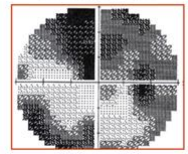
**2) Morphological data**

**RNFL(OCT)**



**3)Psychophysical data**

**HFA 24/2**



**Statistical Analysis**

Differences of PERG, VEP, RCT, and RNFL-T values between OAG and control groups were evaluated by one-way analysis of variance (ANOVA). Pearson's test was applied to compare electrofunctional (PERG IT and A, VEP IT and A, RCT), morphological (RNFL-T), and MD data.

In all analyses, we considered as statistically significant a *p*-value lower than 0.01. Minitab 17 (version 1) software was used for statistics.

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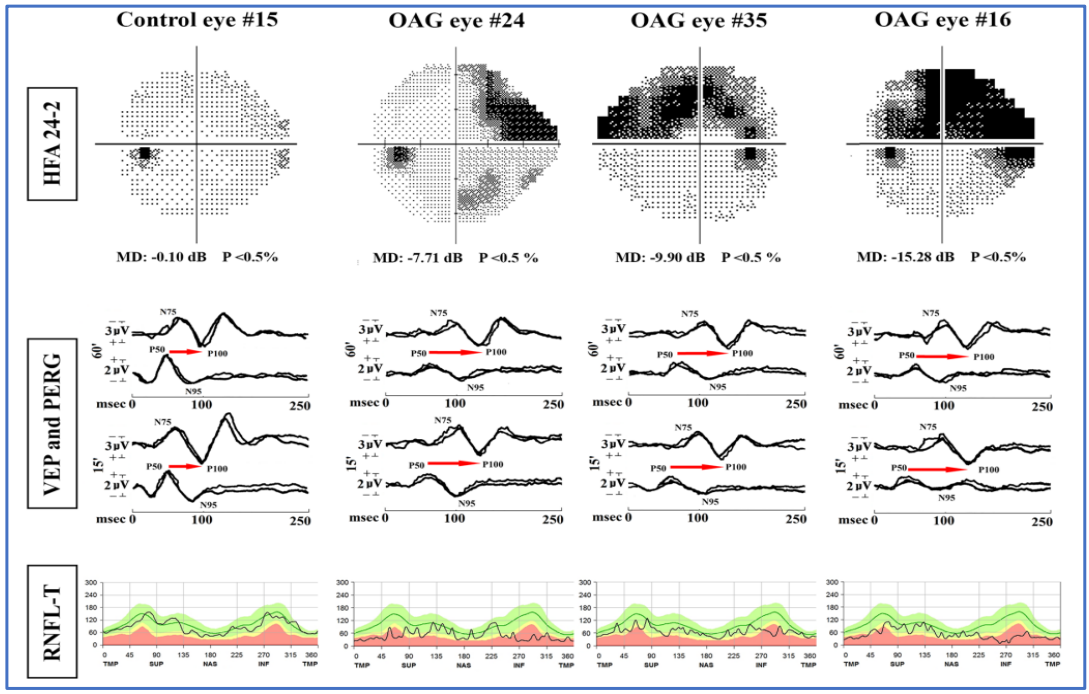
# Results: examples

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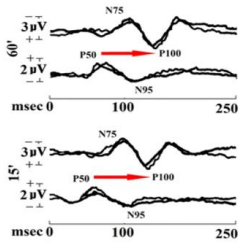
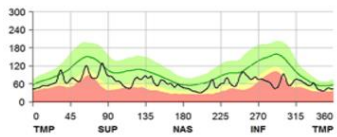
# Results: mean values

## 1) Neural conduction



| | Controls (*N* = 20) | | OAG (*N* = 37) | | ANOVA: | Ab | % |
|---|---|---|---|---|---|---|---|
| | Mean | 1 SD | Mean | 1 SD | *p* | | |
| 60′ PERG IT (ms) | 54.23 | 3.22 | 62.03 | 2.95 | <0.0001 | 32 | 86.4 |
| 60′ PERG A (μV) | 2.02 | 0.16 | 0.90 | 0.19 | <0.0001 | 37 | 100 |
| 60′ VEP IT (ms) | 101.72 | 2.17 | 125.00 | 4.94 | <0.0001 | 37 | 100 |
| 60′ VEP A (μV) | 10.03 | 1.76 | 5.38 | 1.97 | <0.0001 | 35 | 94.5 |
| 60′ RCT (ms) | 47.49 | 2.88 | 63.03 | 4.80 | <0.0001 | 37 | 100 |
| 15′ PERG IT (ms) | 54.56 | 3.62 | 62.89 | 2.79 | <0.0001 | 37 | 100 |
| 15′ PERG A (μV) | 2.23 | 0.19 | 0.78 | 0.17 | <0.0001 | 37 | 100 |
| 15′ VEP IT (ms) | 103.88 | 2.94 | 127.46 | 4.27 | <0.0001 | 37 | 100 |
| 15′ VEP A (μV) | 9.73 | 1.64 | 4.59 | 2.13 | <0.0001 | 37 | 100 |
| 15′ RCT (ms) | 49.32 | 2.07 | 64.57 | 3.68 | <0.0001 | 37 | 100 |

## 2) Morphological data



| | Controls (*N* = 20) | | OAG (*N* = 37) | | ANOVA: | Ab | % |
|---|---|---|---|---|---|---|---|
| | Mean | 1 SD | Mean | 1 SD | *p*= | | |
| RNFL-TT (μ) | 85.96 | 7.62 | 52.16 | 11.87 | <0.0001 | 37 | 100 |
| RNFL-OT (μ) | 110.53 | 4.56 | 63.31 | 17.86 | <0.0001 | 37 | 100 |

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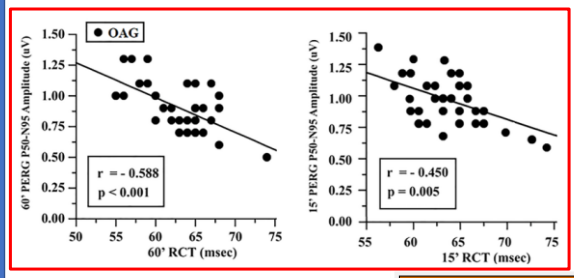
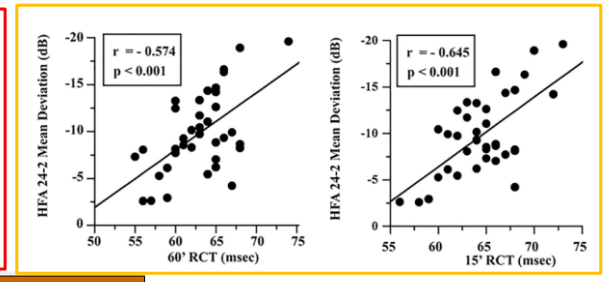
# Results: correlations

## Postretinal neural conduction (RCT) vs

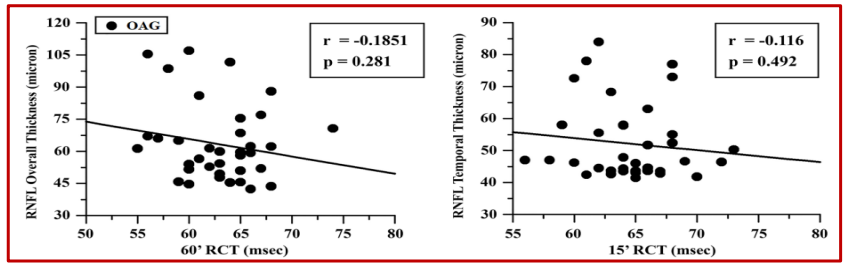
### 1) RGCs Function




### 2) MD




### 3) RNFL

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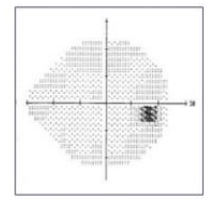
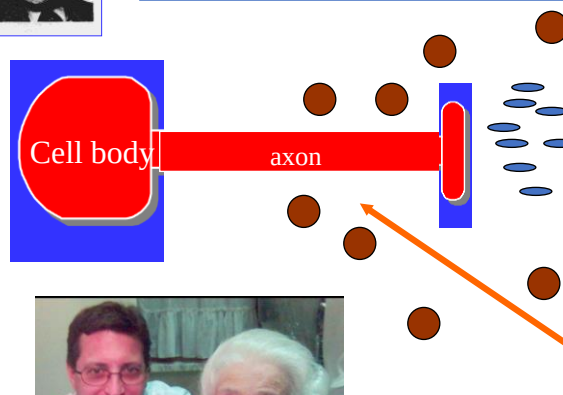
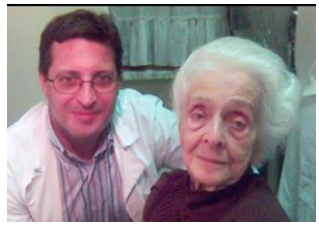
# Discussion 1:Neurophysiological Evidence

**1) Neurotransmission particles release**

**Normal visual perception**





**2) Target cell-electrical activity dependent**

**3) Neurotrophic factor release (NGF, BDNF…)**

**3) Bioelectrical input to visual cortex**

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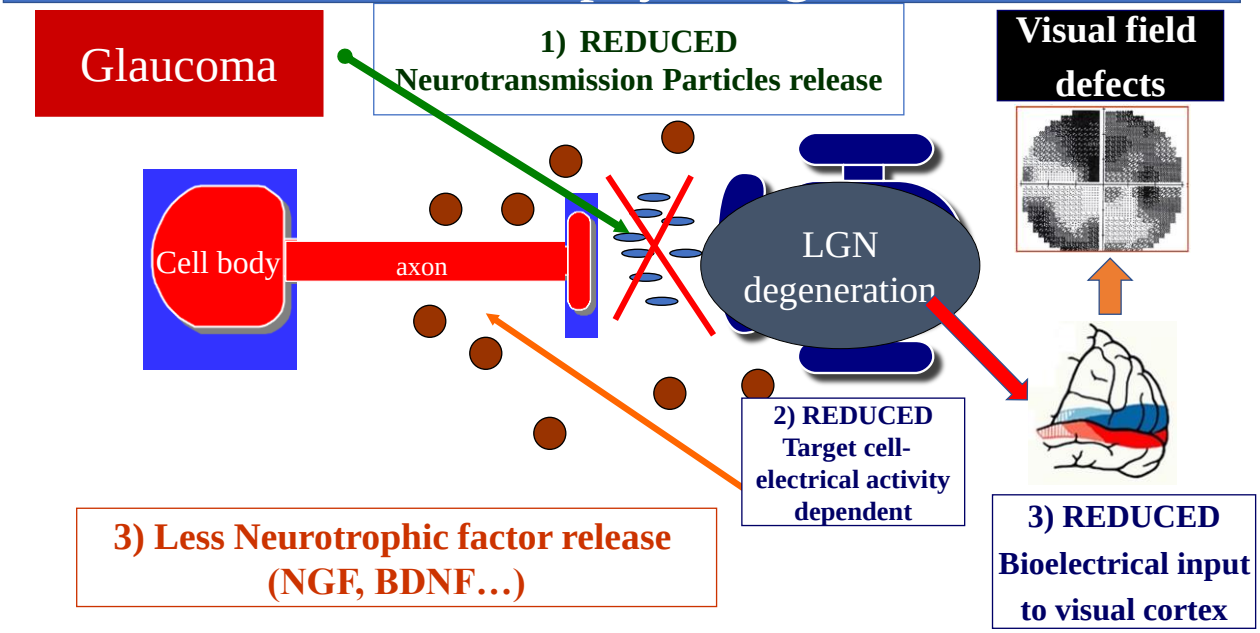
# Discussion 1:Neurophysiological Evidence

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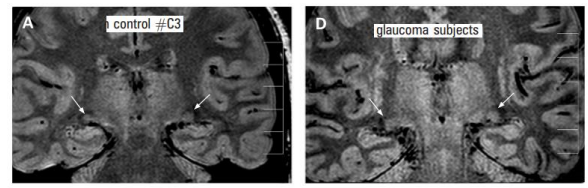
# Discussion 2: Neuroradiological Evidences

**LGN neural degeneration in OAG patients**

Gupta et al. BJO 2009, Dai et al. AJN 2011
Furlanetto et al, PlOS one 2018




**impairment of the afferent synapsis from RGC to LGN neural elements with a consequent trans-synaptic degeneration of LGN**

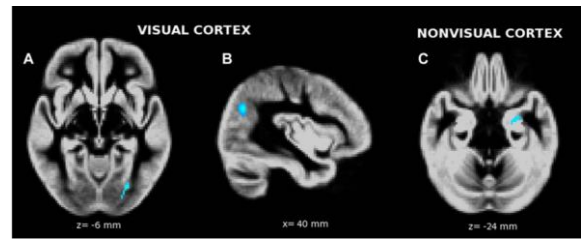
Lawor et al, Surv. Ophthalmol 2018

**axonal disruption of the optic nerves and optic radiations in OAG patients**

Garaci et al, Radiology 2009

**related brain atrophy on both sides of the visual cortex, and in other CNS structures**

Nucci et al, COP 2013; Frezzotti et al, HBM 2013;
Giorgio et al, HBM 2018

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# Conclusions

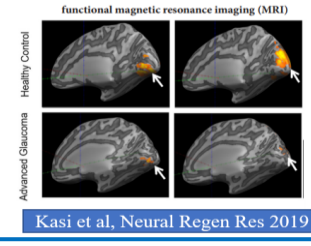
In conclusion, in OAG, by appropriate electrophysiological approaches (simultaneous PERG and VEP recordings with the assessment of RCT), it is possible to detect a functional damage both postretinal large and small axons.

- related to the RGC dysfunction
- contribute to the visual field defect

- independent from the morphological condition of RGC fibers forming the optic nerve head.

functional and structural changes on the postretinal elements, leading to abnormal synaptic connectivity between RGC fibers and LGN and a consequent reduced bioelectrical activity to the visual cortex.

Our findings are in agreement with all recent opinions (see, as review, Kasi et al., 2019) that consider OAG as a neurodegenerative process, in which the involvement is not exclusively located at the level of the neural ocular elements (i.e., RGC) but also an impairment of all visual pathway structures responsible for conveying visual information from the eye to the brain.



Kasi et al, Neural Regen Res 2019

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Thank to co-authors

Thank you for your attention!!!!!

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