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بالرسالة صفحات
لم ترد بالأصل

VISUAL FUNCTION ASSESSMENT BY PATTERN VISUAL EVOKED POTENTIAL AND PATTERN ELECTRORETINO GRAM IN CHRONIC SIMPLE GLAUCOMA

Protocol of a Thesis
Submitted for partial fulfillment of the M.D.
Degree in Physiology
Suez Canal University

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2000

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ACKNOWLEDGEMENT

*I wish to express my deepest gratitude to **Prof. Dr. Abd El-Aziz Mohamed**, Professor and Head of Physiology Department, Faculty of Medicine, **S**uez **C**anal **U**niversity, **(S.C.U.)** for his highly valuable advice, active supervision, continuous support and encouragement.*

*My sincere thanks to **Dr. Onsy El-Alphy**, Prof. Dr. of Ophthalmology in Research Institute of Ophthalmology for his initiation of this research, constant encouragement, highly valuable advice and facilities that made this work completed.*

*I would like to express my deep thanks to **Dr. Magdi El Barbary**, Assistant Professor of Physiology (S.C.U.), for continuous help, valuable advice, criticism, and sincere effort in the statistical analysis of his work.*

I'm most grateful to members of Research Institute of Ophthalmology for continuous help and provided me with the facilities for conducting visual evoked potential (VEP) assessment.

A special word of thanks and gratitude to my colleagues in the physiology department for their support and encouragement.

Lastly I owe the greatest dept to my sisters and my husband, without their understanding and continual encouragement this work would never have been finished.

INTRODUCTION

Glaucoma is a multifactorial disease in which characteristic damage of the optic nerve accompanied by visual field defect occur usually in the presence of elevated intraocular pressure (I.O.P.) *(Stephen et al., 1981).*

The diagnosis of chronic simple glaucoma (CSG) in routine clinical is made when a raised intraocular pressure of > 21 mmhg is accompanied by characteristic optic nerve head cupping and visual field defects *(Lindsay et al., 1991).*

C. S. G. is a blinding disease in Egypt. Many patients do not become aware of having the disease until it is far advanced to give a satisfactory response to treatment *(Korra et al., 1981)*. Glaucoma is responsible for ten percent of all reported blindness in the united states *(Leske, 1983)* and 5.4% - 6.8% in Alexandria *(Said et al., 1970).*

Pathological work in human has suggested that the (POAG) leads to progressive damage of the retinal ganglion cells and their axons and consequent deterioration in visual field. The damage results either from the direct mechanical effects of high IOP, from compromises to the vascular supply or from a combination of these and other factors *(Fechtnner and Weinreb, 1994).*

Pathological studies of optic nerves and retinae of CSG sufferers, showed that, by the time glaucomatous field loss was detected by Gold man perimetry, up to 40% of optic nerve fibers were lost ***(Quigley et al., 1982)***. Also, histological studies, correlated retinal ganglion cell loss with visual field loss detected by octopus and static perimetries and showed that a 20 – 40 % reduction in ganglion cells corresponded to a loss of sensitivity of 5-10 DB ***(Quigley et al., 1989)***.

Early recognition of glaucomatous damage is important, as once a definitive localized visual field defect is present, the process may progress relentlessly and some patients with increased IOP are recognized to be at greater risk of developing glaucomatous damage ***(Stephen et al., 1986)***.

A number of approaches have been advocated in an attempt to improve both detection and monitoring of CSG. Stereoscopic optic disc and nerve fibber layer photography has been advocated as more sensitive indicators of damage but some limitations have been found. The main problems being the fact that photograph only give a look at the structure but not at the function. Such limitations were soon established by the fact that 50% of the retinal nerve fibers have to be degenerated before the changes can readily detected ***(Hichings and Anderson 1983, Quigley et al., 1982).***

Nevertheless, photographic methods are more sensitive than both the automated perimetry and the cup-to-disc ratio, because they reveal changes 5 years earlier ***(Quigley et al., 1989 & Sommerrr et al., 1991).***

Tests of visual function in glaucoma provide a basis from which the clinician can initially confirm the diagnosis. The wider the range of methods available the more definitive a diagnosis can be in a borderline situation. It is of importance to evaluate different functions and clarify the nature of damage in glaucoma.

Electrophysiological methods are now of wide clinical use in assessing the integrity of the retina and its central connection. When a flash of light strikes the retina, it evokes a volley of the nerve impluses which can be transmitted along the visual pathways to occipital cortex. The response to the light stimulus recognized only after an avenging computer eliminated a synchronous spontaneous activity ***(Vaughan Asburg, 1983)***.

Electrophysiological methods (both pattern electroretinogram (PERG) and pattern evoked potential (PVEP) may be the best approach as they at least partially, reflect the function of the ganglion cells and therefore area of value for revealing minor functional changes ***(Berardi et al., 1990, Bach 1992)***.

The ERG represents the summed activity of the distal retina in response to light flashes, and is widely used to assess retinal function. Because it is dominated by extramacular rods and cones, it is most useful in-patients suspected of having widespread retinal disease ***(David & Jane 1992)***. The pattern ERG (PERG) was developed to record responses from localized areas of the retina. Clinical interest in the PERG developed following observation that it was extinguished following section of the optic nerve, whereas the flash ERG was unaffected. The investigators of these studies concluded that the PERG originates from retinal ganglion

cell activity, could be able to detect the changes of retinal ganglion cells with glaucoma *(Tobimatsu et al., 1989)*.

The VEP which is the averaged summated electrical response of the visual cortex evoked by repetitive visual stimulation, essentially reflects the condition through visual pathway from retina to the visual cortex *(Gallowy, 1981)*. The VEP is sensitive to pathological changes in diseases affecting the visual pathway and shows a greater degree of alternation in latency or amplitude for the same degree of damage *(Gallwoy, 1982)*. The VEP is almost entirely derived from the fovea, and illumination of peripheral retina produce little response *(Vaugham Asbory, 1983)*.

Using only the pattern reversal ERG, ***Hawline et al., (1989)*** found that subnormal amplitude of the major positive component of the ERG, NI-PI, was detected in 6 of all 11 eyes (54%) and the amplitude of the major negative ERG component PI-N2 was found subnormal in 5 eyes (45%) in glaucoma group.

Using only the pattern reversal VEP in glaucoma, ***Emad and Tarek (1992)*** found that the mean absolute latencies of P 100 wave statistically significantly delayed in all patients compared to controls, but the mean amplitude of P100 showed no significant changes between patients and controls.

Using both the pattern reversal VEP and ERG, ***Fernandez et al., (1994)*** found that the mean amplitude of (N95) of ERG was highly significant between glaucoma and control group. The mean of P 100 latency of VEP was lightly significant between glaucoma and controls.

Using only the pattern reversal VEP, ***El Bassiouny and El-Nahrawy (1993)*** found a proportionate relation between the degree of optic damage as evidenced by C/D ratio and the latency of P 100.

Aim of the Work

Visual function assessment (objectively) by VEP and ERG to verify the site and degree of insult (retinal and post retinal levels) caused by primary open angle glaucoma.

BASIC CONCEPTS OF GLAUCOMA

Intraocular pressure

The vertebrate eye is a fluid- filled spheroid having a flexible and partially elastic wall. Maintenance of a stable shape is necessary for the importance of optical properties of the eye. The tissue pressure of the intraocular contents is called the intraocular pressure, commonly abbreviated IOP. The IOP is maintained within a fairly narrow range by a complex and dynamic equilibrium in which a nearly constant rate of aqueous humor production is matched by a nearly constant rate of aqueous humor escape from the eye through drainage pathways. Small variations in either the rate of production or in the rate of outflow from the eye can result in large changes in IOP. Thus any variation in a host of parameters affecting aqueous humor production or filtration (including body position, blood pressure, external forces applied to the surface of the globe, and central venous pressure) may result in dramatic changes in IOP *(Brubaker, 1984 and Carel et al., 1984).*

The inflow of aqueous from the ciliary processes is primarily the result of active secretion, which is pressure-independent. However, because the ciliary processes are not a totally fluid tight barrier there is a component of aqueous production due to leakage which is pressure-dependent and generally referred to as ultrafiltration *(Cole, 1977).*

On the other hand, the outflow of aqueous humor appears to be entirely pressure-dependent. The most important route for aqueous outflow is through the trabecular meshwork into Schlemms canal and from there to the episcleral venous system via a complex arrangement of collector channels and aqueous veins. This conventional outflow route accounts for approximately 90% of all drainage from the normotensive