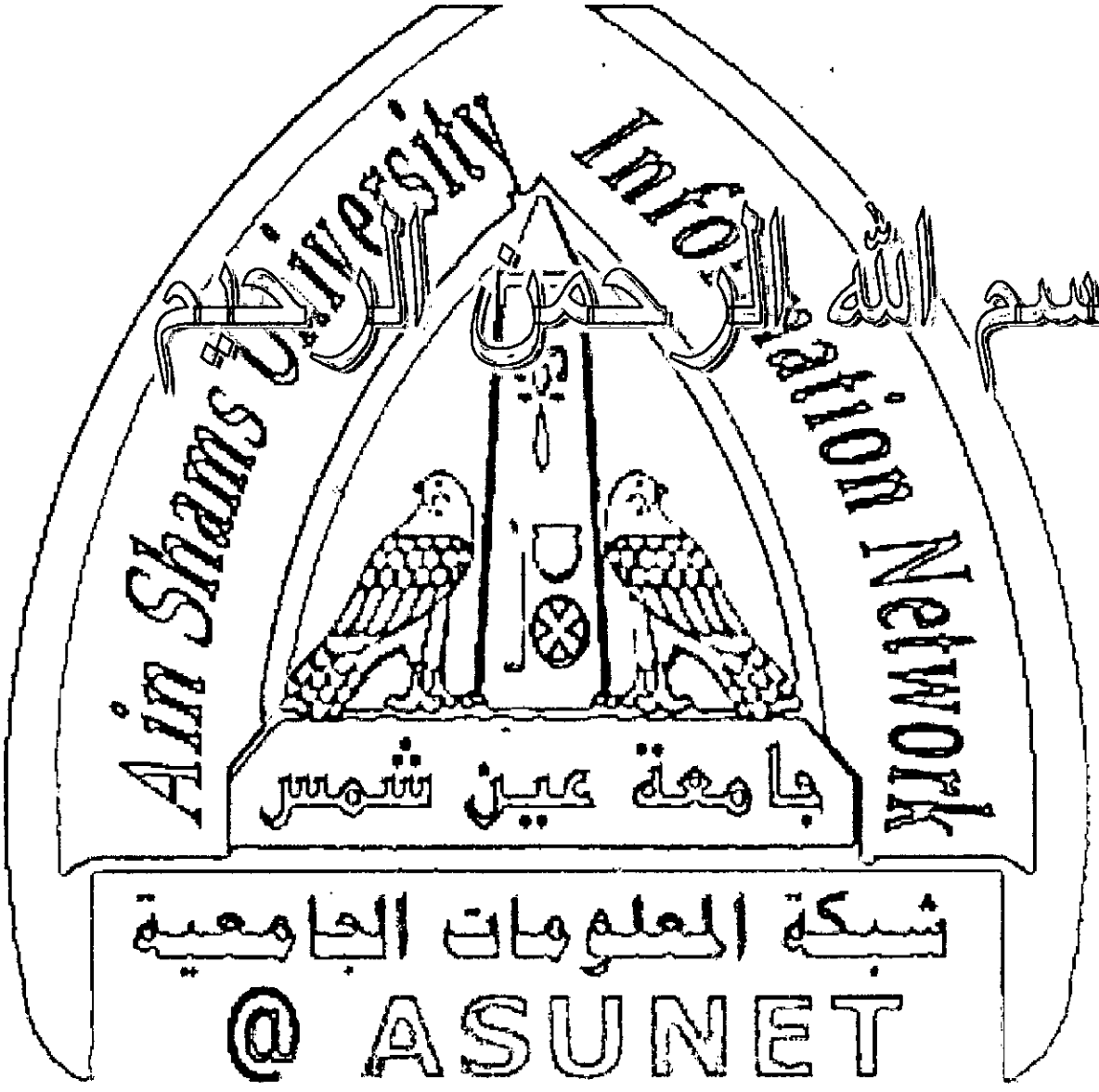
شبكة المعلومات الجامعية



شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

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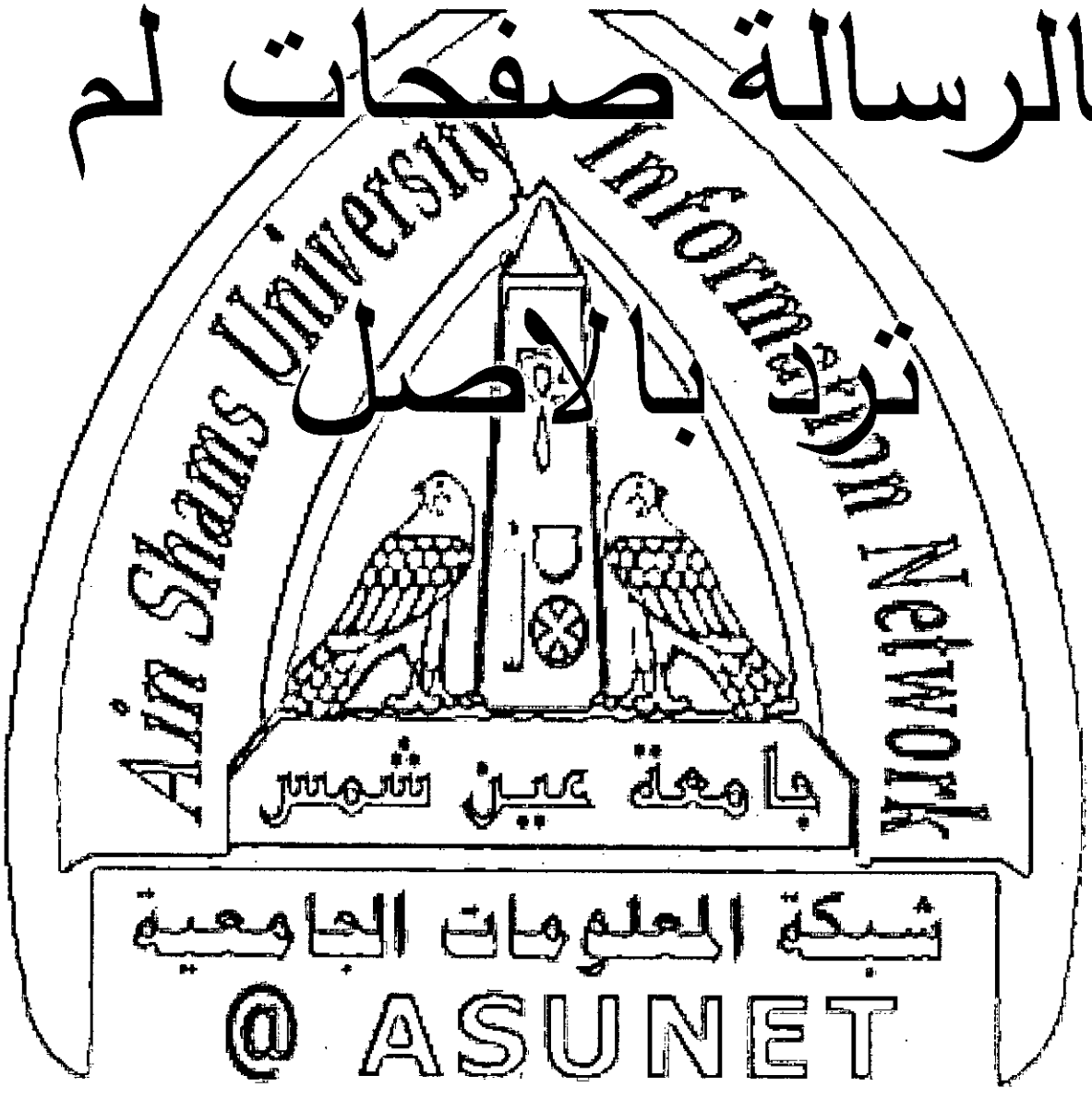

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Phacoemulsification in high axial myopia

Thesis
submitted to the Faculty of Medicine
University of Alexandria

In partial fulfillment of the
Requirements of the degree of

Master Of Ophthalmology

By

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2003

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ACKNOWLEDGMENT

First of all, I thank God for the completion of this work. Next I would like to thank those who generously assisted in the preparation of this thesis.

I would like to express my deep gratitude and appreciation to Professor Dr. Abd El-Kader Elsaid Zeidan, professor and chairman of ophthalmology department, Alexandria University for his planning ideas, enlightening guidance, continuous support and encouragement. I was privileged by his participation & supervision.

I express my deepest gratitude and sincere thanks to Professor Dr. Saeed Ahmed Gomaa, Professor of ophthalmology, Alexandria University. For his meticulous supervision, fruitful criticism and sincere advices which were extremely valuable throughout the course of this thesis.

I am deeply obliged to Dr. Amgad Mostafa Dowidar, assistant professor of ophthalmology, Alexandria University. For his continuous support, unlimited assistance and valuable remarks, needless to say that his unlimited help and preservence formed the corner stone to overcome any obstacles that stood in the way of this work.

I am truly indebted to Dr. Gamal Mohamed Khedr, Lecturer of ophthalmology, Alexandria University. For his kind willing help throughout this work, I would like to thank him for his valuable efforts, and great participation in surgery and follow up observation.

CONTENTS

INTRODUCTION

INTRODUCTION

Theoretically, ametropia might result from an anomalous diopteric apparatus or from an abnormal axial length of the globe. A diopteric apparatus that brings parallel rays of light to focus either in front of or behind the retina produces myopia or hypermetropia, respectively. (1)

Obviously the concept of too high or too low a refracting system is relative to a fixed axial length, but ametropia could equally well arise from a fixed diopteric value and a variable axial length. It is also obvious that refractive errors might arise from a combination of these two factors, and there is evidence in support of each of these three views; in fact, ametropia is sometimes classified as being refractive, axial, or combination in type. (1)

Axial length as the determinant factor of refraction

These elementary theoretic possibilities have considerable practical implications. For the past hundred years it has been accepted as almost axiomatic that refractive errors are generally the result of anomalies in axial length and that for all practical purposes the hypermetropic eye is to be regarded as a short eye and the myopic eye as a long eye, with the emmetropic eye having an ideal axial length of 24 mm. These assumptions have led to an enormous amount of work which aimed at disclosing the mechanism whereby the axial length of the eye remained short in some cases and grew abnormally long in others, most attention being given to this latter question. It has become clear in recent years that this preoccupation with axial length, particularly axial elongation in myopia, was based on too hasty a dismissal of the possible role of the diopteric apparatus. (1)

The following developments contributed toward this dogmatism as to the role of axial length:

1. The keratometer showed that on the whole the corneal power did not vary very much, and this variation certainly could not explain the marked range in refraction seen in the population.

2. It was tacitly assumed that the lens was a constant; even after Tscherning invented the phacometer; the prevailing attitude remained unchanged.

3. In contrast, there was striking objective evidence seen daily in operations and at necropsy that eyes differed considerably in size and that highly myopic eyes were long eyes.

4. With the cornea and lens both regarded as of no consequence in the production of refractive errors, there was logically no alternative but to regard the refractive errors as due to anomalies of axial length. This concept gained support from the ophthalmoscopic observation of temporal crescents and central chorioretinal atrophy in myopia, both being regarded as the result of stretching due to axial elongation during growth. (1)

Implications of these views

This preoccupation with the axial length as the cause of ametropia was in essence a reflection of the widely prevalent views in the second half of the 19th century that the major components of refraction consisted of constants-emmetropia itself being a constant value in axial length, corneal power, and lens power. (1)

In this view, the different degrees of ametropia were proportionate deviations in one constant that changes, i.e., the axial length; as only one constant showed change, the anomalies of axial length were to be regarded as pathologic. In this logic all ametropias were refractive errors, i.e., pathologic or

so near pathologic as not to matter, and differences between high errors and low errors were quantitative and not qualitative. That the causation of axial elongation was interpreted in mechanical terms of pressure and traction (e.g., a low orbit, the pull of the recti or the optic nerve) was only natural in an age that held that "what cannot be investigated and understood mechanically cannot be investigated and understood at all". (1)

Degenerative myopia is that form of myopia that is accompanied by degenerative changes occurring particularly in the posterior segment of the globe; it is usually but not invariably associated with lengthening of the antero-posterior axis of the eyeball and is usually, but by no means always, progressive. It is probably that to some extent at any rate the two-the myopia and the degenerative changes- are independent but they are usually closely related. From the medical point of view degenerative myopia is the most important of all refractive errors for it is relatively common, leading frequently to much visual disability and not infrequently to eventual blindness. (2)

The posterior fundus changes of the myopic eye are as striking as they are unique; they are the clinical basis for the diagnosis of pathological myopia and can affect an incapacitating loss of vision in the later stages of the disease. (3)

These fundus changes have generally been assumed to be the consequence of increased axial elongation of the globe with the attendant mechanical tissue strain and vascular changes which occur secondary to a process of stretching. A definite parallel between the degree of myopia and the severity of its fundus changes has never been established, however. Partly because of this, the concept of a biochemical pathogenesis for these changes has been increasingly challenged and the origin subscribed rather to abiotrophy. (3)

These degenerative changes are particularly marked around the optic disc and in the central area of the fundus involving the choroid and retina. These changes are not due to the mechanical effects of stretching but are primary in

nature and in their incidence genetic factors play a prominent part. They have been erroneously described as "myopic choroiditis" but the condition is not inflammatory. They do not run parallel to the degree of myopia and tend to occur after mid-adult life whereas the elongation of the eye is a phenomenon characteristic of the latter part of the first and the second decades. They probably involve both the ectodermal (retinal) and mesodermal (choroidal and scleral) tissues. This includes atrophic changes in the sclera, around the disc, in the choroid and retina in the central area and in the peripheral parts of the retina. (4)

Atrophic changes in the choroid (*myopic choroido-retinal atrophy*) occur mainly in the central area of the fundus. There is a gradual disappearance of the small vessels of the choroid with the development of lacunae making irregular areas of atrophy, which may extend to the region of the disc where they may eventually fuse with each other and with the myopic crescent so as to form an irregular circumpapillary ring. Small hemorrhages are not uncommon in the macular area and occasionally choroidal thrombosis; the latter may give rise to the sudden formation of a circular claret-colored or black spot at the fovea that may persist (*Fuch's fleck*). These changes are associated with an atrophy of the overlying retina and involve a considerable loss of visual acuity; this tends to be progressive and a central scotoma may result. At the same time the retinal pigmentary epithelium becomes depigmented over most of the fundus so that the choroidal vessels are well seen. (4)

Degenerative changes at the periphery of the retina are also common, typically those of cystoid degeneration. These may lead to the formation of retinal holes resulting in a detachment of this tissue. Degenerative changes also occur in the vitreous, which turns fluid with a breakdown of its colloid structure so that dusty opacities or large membrane-like floaters are formed. (4)

Cataract is encountered more often in myopia and in a younger age group. [5] There are many relationships between myopia and cataract. One common one is the development of induced myopia in association with progression of a nuclear sclerotic cataract. However, on an anatomical basis, there are other relationships that help define the uniqueness of cataract in high myopia. For example there is a subset of individuals who demonstrate a relationship among high myopia, cataract, and youth. Kaufman and Sugar documented this relationship in a study in which they evaluated young patients with myopia who presented with a visually disabling cataract. In a group of 12 consecutive patients, the mean age was 44 years, (range 34 to 54 years). [6]

In the majority of cases a nondescript opacification appears throughout the cortex, which usually progresses and matures rapidly. A characteristic opacification usually commences in the posterior part of the cortex in the axial region (*posterior cortical cataract).* Ophthalmoscopically it appears as a vaguely defined, dark area, and with the slit-lamp the opacity is seen to have irregular borders extending diffusely towards the equator and the nucleus; unlike developmental cataract it is not sharply confined to a particular zone. In the beam of the slit-lamp the opacities have an appearance like breadcrumbs and a characteristic rainbow display of colors often replaces the normal achromatic sheen (*polychromatic luster*). Such a cataract may remain stationary in the posterior cortex for a long time or even indefinitely; in other cases the opacification spreads peripherally until all the posterior cortex is affected, and axially until the entire lens is involved. The total cataract formed in this manner is usually soft and uniform in appearance. [4]

Even in the early stages vision is usually much impaired owing to the position of the opacity near the nodal point of the eye. [4] Unlike cataract in otherwise normal eyes, the diagnosis of cataracts in an eye with high myopia