INTRODUCTION

Ageing is a life long process of progressive physiological changes in biological, psychological and social structure of a person, which no medical or surgical intervention can reverse, or stop. It begins even before we are born and continues throughout life reducing the structural and functional capacity of biological system, which were at a peak in early adult life. The effect of age on the biological systems depends on the individual's life style during his life span and is compounded by genetics, nutritional, traumatic, psychological, metabolic, surgical and disease factor. It differs for individual of the same age. Ageing affects the structure and function of both eyes equally; functions mostly affected include visual acuity, refraction, accommodation, glare, contrast sensitivity and visual field.

AGEING CHANGES IN VISION DEVELOPMENT

Within hours after birth, an infant can move his eyes conjugately and make compensatory eye movements in response to head movements, this is indicative that some vision is present at birth. Infants can distinguish patterns at birth. A few weeks after birth, pursuit movements appear and the child can follow a moving object. At 2 mouths of age, they are able to remember the patterns and compare them to new ones while at 2 years, pattern recognition becomes defined and developed. An infant unable to fixate or hold fixation suggests poor central vision. Visual acuity develops with increasing age in a normal child and attains a maximum between ages 4 and 6, it remains stable until the 40's and then begins to decrease, with rapid decrease occurring after age 60.

Acuity loss may result from functional, organic, optical, retinal and central factors. It may occur as a result of pupil miosis with age which reduces the amount of light entering the eye and stimulating the photoreceptors, media defects and opacities which may increase light scatter, produce halos and induce glare sensitivity, retinal defects and disease which may cause spatial loss, central lesions affecting the brain and visual pathway and a decrease in the number of cortical cells.

AGEING CHANGES IN REFRACTION

There are three possible refractive states namely emmetropia, myopia and hyperopia. Refraction changes with age and occurs as a result of ocular growth after birth. The changes affect the axial length of the eye, the mean refracting power, corneal and lens curvatures, chamber depth, refractive index of the lens cortex and nucleus.

At birth, the antero-posterior diameter of the eyeball is appropriately 17mm, as the eye matures the axial length increases rapidly at first during infantile growth period and then more slowly during juvenile period. By age 20 to 29, it is approximately 24.1mm and by age 50, it decreases to 23.5mm. The mean refracting power of the eye is associated with the refracting power of the lens and cornea, it is approximately 60.7D between age 4 and 9, decreases to about 58.9D between ages 20 to 29 years, and from 50 years and above, it increases to 60.8D.

The cornea and lens are steeply curved at birth compared to adult values, the cornea is flatter than that of the adult cornea and its curvature is greatest at the periphery than in the centre while the reverse is the case for the adult cornea. From birth to age 6, as anteroposterior axis of the eye elongates, the
cornea flattens losing up to 4D of corneal power, with the vertical meridian having the greater curvature. From 40 years and above, the cornea tends to flatten more in the vertical than the horizontal.

At birth, the lens is steeply curved and more nearly spherical in shape than later in life producing a greater refractive power, which helps compensate for the short antero-posterior diameter of the eye. The lens grows throughout life as new fibers are added to the periphery making it flatter with increasing age. From birth to age 6, as the axial length increases, the lens power decreases by about 20D.

**IMPLICATIONS OF THESE AGEING CHANGES.**

For emmetropia to occur, a coordinating mechanism fits the optical parts of the eye together. If there is disproportionate growth of any of the optical parts in relation to the other, ametropia results. If axial length is longer than the focal plane, myopia results, if it is shorter than the focal plane, hyperopia result. If the curvatures of the lens and cornea do not flatten as the axial length is increasing, ametropia results. In the absence of the influence of genetics, disease, poor nutrition, trauma etc, emmetropization occurs as follows: High mean refracting power (contributed by the more spherical lens) with short axial length in children from birth to approximately 9 years of age, a lower mean refracting power from age 8 to adolescence as the axial length increases, a shorter axial length to compensate for the increase in the refractive power of the eye (due to increase in refracting power of the cornea and lens) from age 50 up.

**AGEING CHANGES IN HYPEROPIA**

Approximately 60% of full term infants are born with hyperopia. This could be due to the short axial length, and high mean refracting power of eye. The hyperopia increases with increasing age until about age 6; between ages 6 and 8, it stabilizes and then starts decreasing. During adolescence the degree of hyperopia decreases as the axial length of the eye increases until the close of the growth period at which time any hyperopia still prevalent remains relatively constant or appears to increase due to an increase in the index of refraction of the lens cortex. Between ages 20 and 40, refraction remains stable, from age 45-49, hyperopia increases by about 0.75-1.00D due to lenticular growth, between ages 55-59, there is two times more hyperopia than before and from 75 upwards, hyperopia is three times greater due to flatterig of lens, presence of previous latent hyperopia and a reduction in the volume of the globe. There is also a trend towards less hyperopia and more myopia at this age due to an increased relative index of the lens as cataract forms, after age 65, the number of persons developing myopia is great enough to statistically obscure the trend of others towards hyperopia. Children with about 0.50 to 1.25D of hyperopia at birth may become emmetropic at teen age but the higher the initial hyperopia on entering school, the less likely the child will outgrow it. Associated accommodative esotropia of up to 4 to 7D makes it even worse.

**AGEING CHANGES IN MYOPIA**

Approximately 5% of babies are born with myopia. These babies are mostly premature infants and infants born of under aged mothers as congenital myopia is rare. Myopia in these infants could be as a result of a steep cornea, an underdeveloped spherical lens and a long axial length. During the first few months of life in all children born with myopia, the process of emmetropization occurs rapidly such that by end of the first year, few children are found to be myopic. Between ages 5-9, only 2% have myopia, if a child has myopia at age 5-6, the myopia will remain and probably increase. Most myopia generally shows up around 7-8 years of age. It increases in a linear manner with age into the middle or late teens (age 17 or 20 and sometimes up to 25) and then levels off. All myopia is at first progressive but stops before age 20 except for pathologic myopia. Simple myopia rarely exceeds -6.00D. There is usually little change in refractive error during the third and fourth decades. Myopia tends to decline towards emmetropia between ages 45 and 49 and later increases from 50 upwards due nuclear lens changes. If a child is myopic at 5 or 6 years, the myopia will remain or increase. Children who are emmetropic or less hyperopic at age 6 are likely to be myopic before they finish school.

**AGEING CHANGES IN ASTIGMATISM**

The major source of ocular astigmatism is the anterior cornea. Corneal curvature decrease with age because upper lid pressure diminishes, at birth, astigmatism is against-the-rule as the horizontal meridian of the cornea is more curved than the vertical. During preschool age (below 6 years), it changes to with-the-rule as the vertical meridian of cornea has the greatest curvature. In middle life,
the cornea tends to flatten and the flattening is more marked in vertical than the horizontal changing the axis of the astigmatism to against-the-rule astigmatism, astigmatism not present at birth is never acquired. The degree of astigmatism changes very little throughout life as corneal astigmatism changes at a rate of about 0.2D in every 10yrs.

AGEING CHANGES IN ACCOMMODATION
The consistency of the lens material changes throughout life as newer lens fibers are deposited and older ones shifted towards the centre. At birth, the lens may be compared to a soft plastic, in old age; the lens is of a glass like consistency, this account for the greater resistance to shape change in accommodation, as one grows older.

Human accommodative amplitude declines progressively with age beginning in the second decade of life but its symptoms are masked by an increased depth of focus induced by senile and accommodative miosis. Accommodative amplitude is almost gone by the age of 50 to 55 yrs. Near point of accommodation gradually recedes from 7cm at age 10 to 20cm at age 40 and 40cm at age 50. As the accommodative amplitude decreases, the near point recedes towards the far point so that small objects must be held further from the eye to be clearly visualized, presbyopia sets in. Onset of presbyopia occurs when accommodative amplitude has recede to 5.00D or less, this occurs by about age 40 and amplitude continues to decline until 75yrs when is almost 0.00D.

EFFECT OF AGE ON CONTRAST SENSITIVITY (CS)
Contrast Sensitivity develops with age in the newborn. At birth, CS is very poor; any low contrast object is difficult for an infant to see. As the visual system matures, CS improves. Between 20 and 30yrs of age, CS is at its maximum. The ability to detect objects decreases as a function of age, the ability to see higher spatial frequencies (smaller objects) gets poorer with increasing age. CS is reduced for subjects in the 50 to 60 age range. It is further reduced for subject in age range 80 to 90. Ageing changes in CS are caused by two main factors namely; changes in the optical properties of the eye (i.e., media opacities and pupil size) and reduced neural factors (e.g. patient with open-angle glaucoma and moderate field losses may exhibit reduced CS).

GLARE
Glare is a loss in visual performance or visibility, or the annoyance or discomfort produced by a luminance in the visual field greater than the illuminance to which the eyes are adapted. With age, there is increased heterogeneity in size and molecular weight of the alpha crystalline protein component in the lens. Insoluble high molecular weight species are formed which decrease lens transparency and increase glare effects through light scatter.

Colour vision changes with age as the ability to discriminate between colours diminishes in older individuals. With age, the lens becomes more yellow acting as a yellow filter and decreasing the amount of short wavelength light that reaches the retina. The yellow or brownish lens absorbs short-wavelength light and produces a blue-yellow color vision deficit. The effect of the progressively yellowing of the lens on color vision is made worse by senile miosis that occurs with age. Color vision can be improved in the elderly by the use of higher luminance levels.

VISUAL FIELDS
The visual field is the portion of space in which objects are visible simultaneously during steady fixation. Visual field decreases linearly with advancing age and continues through life. The lateral field starts decreasing asymmetrically as from age 35 while the superior field decreases more from problems affecting the upper lid. There is more rapid field loss after age 60.

AS OPTOMETRISTS, WHAT DO WE DO?
The effect of ageing on the structure and function of individuals of the same age differ as a result of life style and are further compounded by genetic, nutritional, traumatic, psychological, metabolic, surgical and disease factor. This means that the effects of age on any individual can be influenced by proper and balanced nutrition, proper and early management of debilitating diseases and surgical procedures, avoidance of trauma and the psychological management of patients with ageing problems. Since ageing cannot be stopped, we as optometrists can check and manage its consequences to give better vision to our patient.

Babies should be screened at birth so that rare problems like cataract can be detected and treated within the first few weeks of life to enable normal vision develop. Vision should be tested after birth by checking for brisk papillary responses to light to
rule out complete dysfunction of the eyes. For an infant, check for the presence of central and steady fixation of the eyes to light or a coloured object. This indicates acuity of at least 6/60 in the eye investigated. Visual acuity of a preschool child should be tested with the illiterate E or picture charts. If visual acuity is normal between ages 4 and 5, it should be tested every 2 years until age 16. Significant refractive errors are uncommon for children below age 6. Refraction can be performed once in this age range to rule out the presence of any errors of refraction but must be carried out at least every 2 years between ages 6-11 to correct any progression of myopia for mental development. Retinoscopy is recommended before subjective refraction since hyperopia may be undercorrected and myopia overcorrected if only subjective refraction is used as children always accommodate.

From 40 years up, presbyopia sets in, visual acuity reduced, hyperopia increases, myopia decreases, astigmatism changes and reading skill decline. Correct refractive error and recheck refraction every 2 years, adequate illumination and contrast should be provided during tests. Larger lens changes may be used during subjective refraction. Correction of refractive error should always be attempted despite the presence of associated ocular diseases or the ophthalmoscopic picture as hazy media may be compounded by a poor optical image. Refraction should be done at a more leisurely pace with more tolerance for indecision and a trial frame is more suitable than a refractor. The object of treatment in the elderly is to restore the vision to the state normal for that age group and not to the state it was before senility. With old age come physical and psychological alterations due to stagnation and actual retrogression of development, accompanied diseases, loss of income, spouse, friends and a feeling of self worth, slowing movements and easy fatigue, the fear of becoming blind. Give any possible device that will aid vision and educate and redirect the elderly to other hobbies if poor vision does not permit continuation of regular vocation. In patients 65 years and above, do not try to change their habitual visual pattern if they are inconsistent with the patterns in the young example, introducing cylinders or prisms, the simpler the lens correction, the easier it is to adapt to. The working distance, segment height should be accurately measured and changed as little as possible.