

VISION AND AGING

By

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INTRODUCTION



Structure of the Eye

(Source: *Complete Home Medical Guide* by *British Medical Association(BMA)*)

Man is primarily a visually motivated machine and the organ of the body responsible for this function is the eye. Just like our body, our eyes and vision change every passing day. As we get older, we will begin to observe that the ways our eyes will function when we get to our 60s will be quite different from the ways they were functioning when we were in our 30s. These changes involve the eye itself, the surrounding muscles and even the central nervous system that control the ocular functions.

While some of these changes are considered normal, others are considered pathogenic or disease-related. Consequently, while many normal eye-related



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changes occur in all healthy eyes, many people also suffer from disease-related changes that further impair their vision. Older eyes are more susceptible to common age-related diseases such as macular degeneration, cataracts, glaucoma, dry eye syndrome, etc. than younger eyes.

Systemic health problems such as diabetes and hypertension also make the elderly even more at-risk for eye diseases such as diabetic retinopathy and hypertensive retinopathy respectively. Unfortunately, most of the elderly population cannot distinguish between normal vision loss and disease-related vision loss and as such, fail to seek professional care in time.

AGING

What is aging? Even though we all seem to know when someone is old and when someone is young, many authorities are yet to agree on the proper definition of aging. But we all seem to agree in one concept, i.e., that “Aging” starts right from the day we are born because we all know and agree that a one-week old baby is certainly older than a one-day old baby.

As stated earlier, “Aging” is a term difficult to define, primarily because, its meaning varies from one Individual/Organisation to the other (Schmitt and Castillo, 2007). E.g., Whitebourne (1985), defined **aging** as a “*gradual decline in the efficiency of most bodily systems from our 20s*”. Wood and Britton (1988), referred to **aging** as the “*Progressive loss of one’s adaptive ability,*” i.e, the ability to adapt to various situations. And in 2003, Agulanna and Agulanna, defined **aging** as an “*inevitable process of decline in biological, psychological and social functioning of a person, which culminate in death.*”

In his own right, Abanobi (2010), stated that “*a person can be regarded as aging from the moment of conception or from the moment the individual reaches full maturation,*” while the United Nations defined an “*aging person*” as that “*who has attained the age of 60 years or more.*”

As at today, sociologist and demographers, are yet to agreed just when to begin to consider a person as an older adult. However, as a figure, age 65 years, is commonly found in many publications, but the U.S. Census Bureau sometimes start at age 60years for older adults (Schmitt and Castillo; 2007).

Below is a table showing the stages of life according to Abanobi (2010).

S/No	Stage	Approximate Age (Yrs)
1	Infancy	0-1
2	Preschool	2-5
3	Childhood	6-12
4	Adolescence	13-17
5	Early Maturity	18-24
6	Maturity	25-40
7	Middle Age	41-55
8	Later Maturity	56-75
9	Old Age	Over 75

The stages of life Cycle

(Source: Core Concepts in Epidemiology and Public Health Practice by Abanobi O.C(2010)).

As mentioned earlier, certain developmental changes start to occur in us as we begin to age. They include normal and expected alterations in growth patterns, sexual maturation, the graying of hair colour and loss of skin (dermal) elasticity, i.e, the so-called bagginess, wrinkles and creases. Indeed, wrinkling always occurs at right angles to the pull of the underlying muscles, which accounts for the radial pattern around the mouth and eyes.

Nevertheless, it should be noted that people vary in the rate at which they manifest the indicators of aging. According to Abanobi(2010), these variations may be partly due to genetic make-up and partly due to lifestyle as some people remain youthful well into older adults, while others look far older than their age.

TYPES OF AGING

There are two types of aging - the primary and the secondary. Primary aging refers to the anatomical and physiological changes associated with the aging process (normal aging). This does not depend on any concomitant or co-existing disease mechanism. This type of aging process has been reported to begin at about the age of 30 years and continues steadily, thereafter.

It should be noted that most of our body organs reach their peak of efficiency and reserve at about the age of 20 years and remain relatively stable until about the age of 30 years. Thereafter, a steady and gradual decline is experienced in functional activity and ability. At age 75-80years,

most physiological structures have lost about 50% of their original functional capabilities (Schmitt and Cantillo; 2007).

On the other hand, secondary aging refers to the aging process that has been accelerated by controllable or preventable factors. These factors include social problems, psychological difficulties, economic stress and diseases- such as diabetes, hypertension, cardiovascular, cerebrovascular, cancer and rheumatic disorders.

Smoking, excessive use of alcohol, poor nutrition and lack of proper exercise also fall into this category of secondary aging. Indeed, preventive health care must be at the forefront of all health care programs for older adults (Schmitt and Cantillo; 2007).

VISION

According to Dorland's Illustrated Medical Dictionary (2007), *Vision*, is the ***"special sense by which objects in the external environment are perceived by means of light they give off or reflect which stimulates the photoreceptors in the retina."*** This phenomenon is what we refer to as the act of seeing or visual acuity, i.e, clarity or clearness. Visual acuity levels show marked changes throughout a person's life. By the 7th decade, a progressive loss in the time required for dark adaptation is experienced (Marial and Onoley, 1988).

These resultant dysfunctional visual abilities have been reported to be predominantly responsible for loss of mobility, freedom, independence and deterioration of the lifestyles of older Adults (Mouse and Freedman; 2007). According to Siegel and Davidson (1984), impaired vision can also lead to isolation, depression, sometimes disorientation and confusion for the older adult.

In addition, there is difficulty in ambulation (walking) leading to injuries from falls, motor accidents and general diminished productivity. Indeed, annual eye examination are strongly recommended for older adults particularly, where there are previous family history of eye diseases such as glaucoma, ARMD, diabetes, etc., (American Optometric Association; 2006).

VISION AND AGING

As mentioned earlier, the visual system changes in many ways as the human body ages. This may result in a number of noticeable differences in how well we see when compared to the previous years. While, not everyone of the same age group will experience the same level of symptoms, the following are some common age-related vision changes associated with the normal aging process. They are both anatomical and neural.

1. Anatomical Changes in the Eye

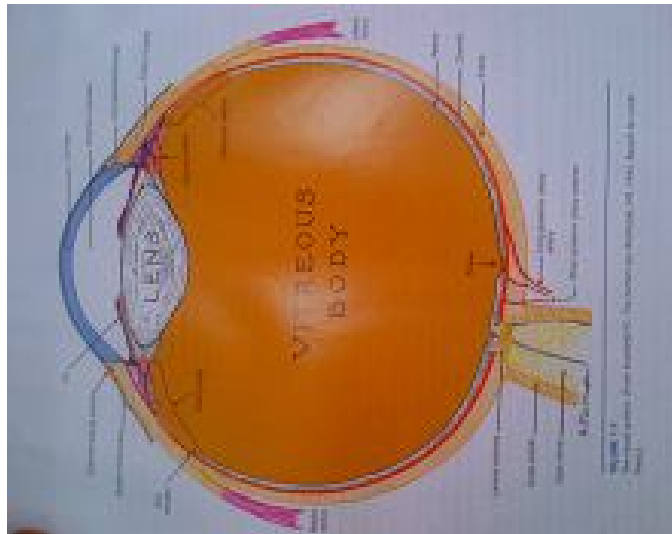


Diagram showing a cross section of the Right Eye

(Source: *Clinical Anatomy of the Visual System* by L.A. Remington)

***Cornea:** The cornea forms the anterior 1/6th of the outer coat of the eyeball. It serves as the window of the eye with both protective and optical functions. It transmits light rays and acts as a strong convex lens by refracting light rays towards the crystalline lens which further refracts them to a focus on the retina.

As we age, the corneal sensitivity to touch decreases. The threshold for touch almost doubles between the ages of 10 and 80 years. The cause for this decrease is not known (Balazsi et al,1984; Millodot,1977). Consequently, considerably less pressure is required to damage the corneal epithelium in adults older than 60years. This reduced sensitivity and corneal fragility increases the risk of corneal abrasion and ulcer.

The cornea is also known to be thicker with age, resulting in extra light scatter inside the aging eye. It also affects the focusing power of the cornea.

***Anterior Chamber:** The depth of the anterior chamber, AC, has been reported to decrease from an average of 3.6mm in the age range of 15-20years to an average of 3.0mm in the 70years and above age group, due to the growth of the crystalline lens (Haegerstrom- Portnoy and Morgan;2007).

Sometimes, this decrease in depth of the AC makes the angle of the AC at the root of the iris more acute, thus increasing the possibility of interference with the aqueous outflow resulting to glaucoma. If this does not happen, the decrease depth increases the refractive power of the eye, making it relatively more myopic.

***Iris:** This is normally brown in Africans. It forms a wall or diaphragm between the anterior and posterior chambers of the eye. At the centre is the circular opening called the pupil. It controls the amount of light entering the eye and the depth of focus.

As we age, there is an increased rigidity of the iris blood vessels and an atrophy of the dilator muscle fibers responsible for the control of our pupil size and reaction to light. This loss in strength causes the pupil to become smaller (senile miosis) and less responsive to changes in ambient lighting (Heagerstrom-Portnoy and Morgan; 2007).

This linear loss with age varies from 0.43mm per decade for low photopic light levels to 0.15mm per decade for high photopic light levels (Winn et al;1994). These changes are dependent on sex and refractive error.

Consequently, people in their 60s tend to need three times more ambient light for comfortable reading than those in their 20s. In addition, they are more likely to be dazzled by bright light and glare when emerging from a dimly lit room to a bright environment.

On the positive side, miosis reduces retinal illumination and the diameter of retinal blur circles. Consequently, with high levels of illumination, uncorrected visual acuity may appear to improve rather than decrease with age.

However, the miosis usually makes it difficult to examine the function or other internal structures of the eye through undilated pupil. Subjective refraction becomes more difficult because changes in lens power do not change the diameter of the retinal blur circles, as much as, similar changes experienced in eyes with larger pupils.

***Crystalline Lens:** This is a transparent, avascular biconvex structure. It is essentially a refracting structure. The human lens continues to grow throughout life (Haegerstrom- Portnoy and Morgan;2007). The axial thickness of the lens has been reported to increase linearly by about 28% by age 70 years from what it was at the age of about 15-20 years, i.e., from about 3.6mm to 4.6mm at age 70 years. While the nuclear thickness remains constant, the cortical thickness increases.

This results in the lens losing its flexibility, thus becoming rigid. This loss of form, overtime, results in the inability of the eye to focus in detail on objects at normal close range. This change is usually experienced at about the age 37-40 years.

As earlier stated, the miosis arising as a result of the atrophy of the dilator muscle fibers and the growth of the lens, do alter the visual performance of the eye. Thus, amount of light reaching the retina of a normal 60 year old is only about 1/3rd that reaching the retina of a 20 year old.

Implying that an older person must use significantly more light to achieve the same level of retinal illumination as that achieved by a younger person. This explains why, the visual performance of an older person is usually impaired at twilight.

Again, as the lens thickens, it begins the selective absorption of light (Mellorio,1987). Flourogens begin to accumulate, just as proteins of high molecular weight increase towards the lens nucleus, leading to the yellowing of the lens overtime.

This yellowing of lens reduces its transparency and causes lens opacities that serve as scatter points for light. These yellow lens pigments absorb short wavelengths more than the long ones, resulting in older adults having decreased sensitivity at the violet end of the spectrum. Consequently, objects appear yellow, while it becomes difficult for them to differentiate blue from

green, and dark grey from dark brown (Haegerstrom-Portnoy and Morgan; 2007).

***Vitreous:** In a healthy young eye, the vitreous is a clear gel-like substance with the consistency of egg-white. It is essentially protein and hyaluric acid. Its functions are mechanical by maintaining the lens and retina in their normal positions and optical by providing a clear medium through which light rays pass unhindered to the retina.

With age, the vitreous becomes thinner and more water-like (liquefaction) and at the same time undergoes syneresis (Haegerstrom-Portnoy and Morgan; 2007). Thus, pockets of vitreous develop within the eye creating lumps of cellular debris called floaters or muscae volitantes.

Vitreous floaters ordinarily settle at the bottom of the eye in a normal eye, but as the vitreous continues to liquefy, they start moving about with speed and amplitude. Though, they do not impair vision, they are certainly a continuous source of worry and irritation to older adults, particularly, when reading.

Another change observed in the vitreous is what we call posterior vitreous detachment. The thinning or the liquefaction and syneresis of the vitreous make it to pull away from the retina at the back of the eye. While, this detachment does not impair vision, its symptoms which include flashes of light, distorted and blurred images, coupled with increase floaters, are a handful of worries and anxiety for older adults.

2. Neural Changes

Neural refers to the parts of the nervous system which includes the brain, the spinal cord and the nerves. Neurons, which are the specialized cells that make up the nervous system, are the only cells in the body that communicate through chemical and electrical signals.

Our visual properties are totally dependent upon these neurons as they pass the information of whatever we perceive or see to the brain for processing, before we can appreciate whatever object is before us.

***The Retina:** This forms the innermost coat of the eye and is concerned with the reception of images of the fixation objects. The retina is made up of

several layers of neurons and is an extension of the brain. The back layer of the retina also contains photoreceptor cells.

These are specialized cells that transform light energy into neural signals. As we age, these retinal cells thin externally at the periphery leading to the irregular orientation of the surviving cells, thereby causing glare.

It should be noted that other parts of the brain experience cell loss with age as well. Because neurons in the brain do not regenerate, these cell deaths result in reduced abilities of the eye to perceive different aspects of visual stimuli, leading to slow response time, in other words, the older eye respond more slowly to light stimuli than younger eye.

***Retinal Pigment Epithelium:**

This darkly pigmented tissue behind the retina provides the retina with rich nutrients as well as serving to absorb excess light and prevent scatter within the eye. As we age, the cells of the RPE become irregular making them less able to absorb excess light and control light scatter resulting to increase glare.

It should be noted that both the anatomical and neural changes occur independent of each other. Sometimes however, they occur together resulting in reduced ocular motility - the inability of the eye to move about freely which result in reading difficulty.

EFFECTS OF THE NORMAL AGING PROCESS ON VISION

***Presbyopia:** As we approach the milestone age of 37-40 years, you will begin to observe some difficulty to focus on near objects, particularly reading materials. This difficulty, we involuntarily/unconsciously try to compensate for, by pushing the print further and further away from ourself.

This is due to the hardening of the lens with age, making it to loose some of its ability to relax and contract thereby making it difficult to vary the focal points of the eye. This loss in ability to focus on near objects due to age is referred to as presbyopia (Grosvenor, 2007).

***Cataract:** This is any opacity of the crystalline lens sufficient to cause visual impairment (Kanski, 1997). The word “cataract” actually means a waterfall in greek. The ancient Greeks believed that the grayish appearance

behind the pupil were bad humors falling down from the brain above (Bisley, 1980). Age- related cataract is also referred to as senile cataract.

It can be in the form of nuclear, cortical or posterior subcapsular cataract. All 3 types cause blurred vision and blindness.

The main symptoms are slowly progressive, painless, reduced vision, glare, yellowing of vision due to the modification of light reaching the retina as a result of nuclear sclerosis formation.

Sometime, the elderly may complain of renewed ability to read without glasses despite a decrease in distance visual acuity. This is due to the slow progression of the cataract (nuclear sclerosis) which creates acquired myopia or second sight in the process (Nicholas and Patel, 1994).



(a)Senile cataract-mature (b)Hypermature cataract with wrinkled anterior lens capsule as a result of water leakage out of lens



(a)Posterior subcapsular lens opacities (b)Posterior & anterior subcapsular lens opacities

(Source: *Clinical Diagnostic Ophthalmology* by J.J. Kanski)

Nicholas and Patel (1994), reported that the disorganization of the lens fibre architecture, denaturation of the lens protein in the lens fibre cytoplasm, followed by the aggregation of adjacent protein molecules to form clumps are responsible for cataract formation. These changes, according to WHO (1997), are exacerbated by the increased radiant energy reaching the earth due to Ozone layer depletion.

***Glaucoma:** This refers to a rise in the **IOP** of the eye above normal level. It is a condition of signs (and sometime symptoms) and not a disease sui generis (Miller, 1906). It is an eye blinding disease that operates like a potential thief. There are two main categories of glaucoma, namely, primary-comprising open-angle and closed-angle glaucoma, and secondary glaucoma.



(a)A small, dimple-like central cup (b)Cup-disc ratio indicating diameter of cup expressed as fraction of diameter of disc (c) Only 2% of normal eyes have C/D ratio of about 0.7



(a)Very severe asymmetrical paracentral changes in advanced POAG. Changes more severe in L/E. (b)Mild asymmetrical paracentral changes

(Source: Clinical Diagnostic Ophthalmology by J.J. Kanski)

The most common type of glaucoma is the POAG and the condition is more of signs rather than symptoms. Hence, patients usually do not report for early treatment until they are almost blind. At this stage, very little can be done to restore vision.

The main symptoms include increased IOP, atrophy of the optic disc with characteristic excavation (cupping) and visual defects.

Everybody is at risk of developing glaucoma as we get older. Indeed, statistics show that the risk of getting glaucoma rises from 1% at the age of 40years to 12% at the age of 80years. These risk are as a result of:-

~The shallowing of the depth of the AC due to the growth of the crystalline lens that makes the root of the iris more acute.

~The increased deposition of extracellular materials on the trabecular meshwork with age.

~The degeneration of the endothelial cells lining the canal of the schlemm which has been reported to be at the rate of 430 cells per year after age 40 years.

-All three factors contribute to decreased aqueous outflow at the trabecular meshwork resulting in IOP build-up (Giese and Shelley; 2007).

***Dry Eyes:** As we age, our tear glands can no longer produce enough tears to lubricate the eye. When they do, they are of poor quality. The symptoms include itching, burning sensation, foreign body sensation, intolerance to dust and smoke and occasionally, excessive tearing. The changes tend to be seen more in women (particularly after menopause) than in men (Giese and Shelley; 2007).

*** Decreased Color Vision:** The fovea is the most sensitive part of the retina where fine details are resolved. It contains densely packed color-sensitive photoreceptors called cones. As the eye ages, the fovea experiences some cell loss and consequently, loss of important color information.

Hence, colors become less bright and the contrast between different colors become less noticeable, such that blue color may appear faded or washed-out. This is a problem for older artists, seamstresses and electricians. Color discrimination is also affected as the ageing lens starts getting discolored (yellowing) due to cataract formation.

***Visual Field Loss:** The visual field is the projected area of vision with the line of sight in the primary position. It covers the area extending 60 superiorly, 70 inferiorly, 95 temporally and 60 nasally. This gives a total horizontal visual field of 190, a monocular visual field of 155, and a binocular overlap of 120 (Borish, 2006).

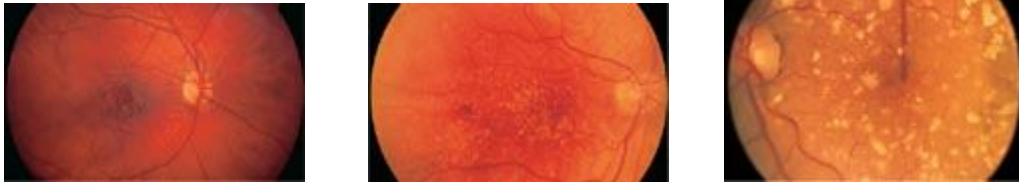
The normal aging eye loses about 20° to 30 peripheral visual field by the age of 70 years. This is an average of 1°-3° per decade. This loss of peripheral field increases the risk of automobile accidents in the elderly. This is where safety is a major concern, particularly, as the elderly adults in this country are made to go through “hell” in pursuit of their pension.

To increase their range of vision, the elderly are advised to turn their head

and look both ways, when approaching an intersection or when crossing the road.

***Glare:** This is a common complaint among the elderly when they are exposed to headlights at night or sun reflection from the windshields or pavements during the day. This makes it more difficult for them to drive. Glare is caused by the changes in the crystalline lens which causes light to scatter rather than being focused, precisely at a point on the retina.

*** Age Related Macular Degeneration (ARMD)**



(a) small, round, discrete, yellow spots at posterior pole (b) more numerous & slightly larger drusen (c) drusen acquiring a glistering appearance.
(Source: *Clinical Diagnostic Ophthalmology* by J.J. Kanski)

ARMD can also be referred to as senile macular degeneration. It is caused by arteriosclerosis or insufficient blood supply through the vessels of the choroids underlying the macular area. Lack of important nutrients in diet may also be a factor in senile macular degeneration. The initial complaints are reading difficulty and inability to see things straight or straight lines appear broken.

There are two types of ARMD, namely:-

~**Dry (Atrophic) ARMD** - which involves the gradual destruction of cones in the macular area.



(a) Focal atrophy of RPE in association with macular drusen
(b) Coalescence of atrophic areas.

(Source: *Clinical Diagnostic Ophthalmology* by J.J. Kanski)

~**Wet (Exudative) ARMD**- when the retina respond to the loss of blood circulation by forming new blood vessels leading to neovascularization.



(a) Crescent-shaped subretinal haemorrhage temporal to macular (b) Blood & fluid at the macula surrounded by ring of hard exudates.

(Source: *Clinical Diagnostic Ophthalmology* by J.J. Kanski)

Statistics show that 10% of all ARMD are of the wet type and it accounts for 90% of visual loss in all ARMD cases.

The symptoms of ARMD include:-

- ~ Blurring or distortion of objects
- ~ Straight lines appear wavy or missing
- ~ Vision is generally poor in low contrast or glare conditions such as in early morning, early evening or night.

EFFECTS OF PATHOGENIC PROCESSES ON AGING VISION

Diabetes Mellitus

This is a disease that is due to numerous complex factors, all of which have in common, elevated level of sugar/glucose in the blood. There are three types, namely:-

- ~ Type 1- is immune-mediated and is referred to as Insulin-dependent diabetes (IDD).

~ Type 2- is Insulin-resistant and is referred to as Non-insulin-dependent diabetes (NIDD).

~ Gestational diabetes- which occurs during pregnancy.

According to Grosvenor (2007), type-1 diabetes used to be described as “Juvenile onset diabetes,” while type-2 diabetes was described as “adult onset diabetes.” But today, “Juvenile onset” and “adult onset” terminologies have been dropped due to the increasing numbers of children currently developing type-2 diabetes, which was formerly thought to occur only in adults.

Nevertheless, the incidence of type-2 diabetes increases with age resulting in a prevalence of 10% and 20% respectively in the 7th and 8th decade of life (Fried; 2007).

Ocular Symptoms of diabetes mellitus

*** Fluctuation in Refractive Errors (Vision):** One of the early symptoms of diabetes may be blurring of vision due to a temporary increase in the refractive index of the crystalline lens. Increased blood glucose level increases the index of refraction of the crystalline lens, resulting in myopia or more myopia (if already myopic), thus causing blurred distant vision.

On the other hand, decrease blood glucose level results in normal clear visions (if vision was normal before the blood glucose level increased).

Thus, fluctuations in refractive error may very well be the first indication of diabetes.

*** Diabetic Retinopathy (DR):** The prevalence of diabetic retinopathy is higher in IDDs (40%) than in NIDDs (20%) (Kanski, 1997). Indeed, diabetic retinopathy is the most common cause of legal blindness in people between the ages of 20 to 70 years.

Clinically, diabetic retinopathy can be classified into two stages:-

~ The early stage known as background retinopathy and

~ The later and more serious stage known as proliferative retinopathy. Recently however, two additional categories have been added. They are pre-proliferative retinopathy and maculopathy (Grosvenor; 2007).

***Risk factors:-**

~ The most important risk factor is the duration of the diabetes in the individual. While it is rare to develop diabetic retinopathy within 5years of the onset of diabetes, about 5% of the sufferers of NIDD present with background diabetic retinopathy. On the other hand, anybody diagnosed as having diabetes before the age of 30years has a 50% chance of developing diabetic retinopathy within 10years and 90% chance after 30years (Kanski;1997).

~ While good metabolic control of diabetes will not prevent diabetic retinopathy, poorly controlled diabetics develop diabetic retinopathy much sooner than well-controlled ones (Kanski; 1997).

~ Miscellaneous factors: These include pregnancy, systemic hypertension, renal disease and anaemia. While pregnancy may not be controlled, the later three factors must be well controlled in diabetics to avoid their adverse effects on diabetic retinopathy (Kanski, 1997).

Clinical Features

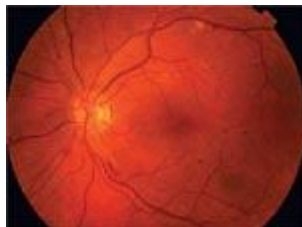
*** Background diabetic Retinopathy**

~ Micro-aneurysm – located in the inner nuclear layer of the retina. They appear as small round dots usually temporal to the macula.

~ Haemorrhages- they have “dot” and “blot” configuration with flame-shaped haemorrhages.

~ Hard exudates- appear as yellow and waxy with distinct margins

~ Retinal Oedema- characterized by retinal thickening which obscures the underlying retinal pigment epithelium.



(a)Early BDR with scattered microaneurysms,dot & small flame-shaped haemorrhages

(Source: Clinical Diagnostic Ophthalmology by J.J. Kanski)

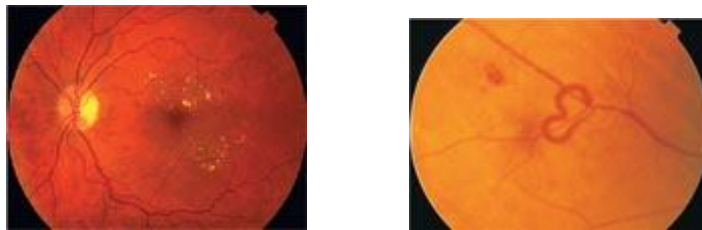
* **Pre-Proliferative DR**

~ Vascular Changes:- Veins begin to appear as “bead”, hop and sausage” – resembling a branch retinal artery occlusion .

~ Dark blot haemorrhages – represents haemorrhagic retinal infarcts.

~ Multiple Cotton-wool spots – white and opaque appearance.

~ Intraretinal microvascular abnormalities- resembles focal areas of flat retinal neovascularization.



(a) More hard exudates but fewer microaneurysms (b) Venous looping
(Source: *Clinical Diagnostic Ophthalmology* by J.J. Kanski)

* **Proliferative DR:**

~ Neovascularization – new vessels proliferate on the optic new head, new vessels at disc (NVD).

~ Vitreous detachment – usually the result of the strong attachments of the cortical vitreous gel to areas of fibrovascular proliferation.

Haemorrhages- bleeding into the vitreous gel.



(a) Severe venous dilation & sausage-like segmentation (b) Cotton-wool spot, severe venous changes & dark blot haemorrhages. (c) Preretinal & early vitreous haemorrhage.

(Source: *Clinical Diagnostic Ophthalmology* by J.J. Kanski)

Symptoms of DR

~ As mentioned earlier, blurred distance vision as a result of the increase in the refractive index of the crystalline lens due to increase in blood glucose level. Usually, the vision returns to normal when the blood glucose level returns to normal.

~ There may be no symptoms in the early stages of background diabetic retinopathy. However, once tiny haemorrhages begin to appear, the individual may begin to complain of blurred vision, which “comes and goes.” If macula edema is present, there will be constant vision degradation.

~ In the later stages, i.e the stage of proliferative DR, widespread haemorrhages cause severe vision disturbance. The individual may observe red objects in front of the eye. This is due to the haemorrhaging blood seeping into the space between the retina and the vitreous humor.

Hypertension

The blood pressure of a resting young adult should not be more than 120/80mmhg. Generally, a person is considered to be hypertensive when his or her blood pressure is persistently higher than 140/90 mmHg, when at rest (British Medical Association, 2010).

Hypertension in older adults is extremely common. High systolic or diastolic pressure is present in more than half of the population older than 65years (Fried, 2007). Hypertension predisposes older adults to vascular end-organ complications such as heart failure, stroke, renal failure, coronary artery disease, peripheral vascular disease and ophthalmic vascular occlusions (CRVO/CRAO).

***Risk factors:**

~ Cigarette smoking, obesity, sedentary lifestyle, high dietary sodium levels, alcohol consumption, poor nutrition and genetic factors.

~ The condition is most common in middle age and elderly people because the arteries become more rigid with age. It is also marginally more common in men than in women. Black people are more susceptible to the condition (BMA; 2010).

~ When hypertension develops during pregnancy, it can lead to the development of the potentially life threatening conditions known as pre-eclampsia and eclampsia. Although the hypertension usually returns to normal after birth, women who have hypertension in pregnancy are at a slightly increased risk of developing it later in life.

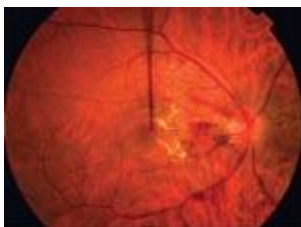
***Symptoms:** Suffice to say here that there are two types of hypertension-primary or essential hypertension and secondary hypertension. The former is most common and affects about 90% of patients. It is believed to be caused by changes in any of the regulatory mechanisms that maintain normal arterial blood pressure, while the later which affects about 10% of the population, is caused mostly by renal artery stenosis and polycystic kidney disease (Fried, 2007).

~ The most common symptoms ascribed to hypertension are headache, epistaxis (bleeding from nose) and tinnitus (ringing, buzzing or roaring sound in the ears). These symptoms are however, just as common among persons without hypertension (Fried, 2007).

***Hypertensive Retinopathy:** The narrowing of the retinal arterioles are usually the primary response of the eye to systemic hypertension. In sustained hypertension, the blood-retinal barrier is disrupted in small areas, resulting in increased vascular permeability.

Clinical features

The fundus picture is characterized by:- (i) Vasoconstriction (ii) Leakage and (iii) arteriosclerosis



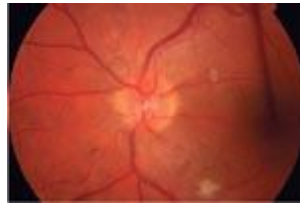
(a) Flame-shaped retinal haemorrhages & small hard exudates at macula (b) Severe disc oedema associated with peripapillary flame-shaped haemorrhages, cotton-wool spots, venous tortuosity with dilation.



(a) Extensive haemorrhages involving the inferior nasal & temporal fundus (b) NVD developed several weeks later.

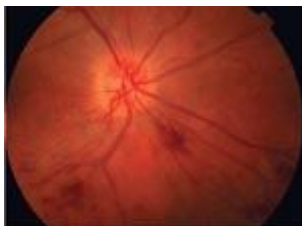
(Source: Clinical Diagnostic Ophthalmology by J.J. Kanski)

(i) Vasoconstriction:- Generalized and focal arteriolar narrowing. In severe hypertension, obstruction of the pericapillary arterioles may occur leading to the development of cotton-wool spots.



(a) Focal & generalized arterial narrowing (b) Cotton-wool spots

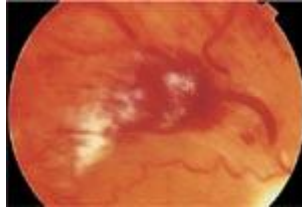
(ii) Leakages: Caused by abnormal vascular permeability which leads to the development of flame-shaped haemorrhages, retinal edema and hard exudates in the Henle's layer of the fovea may lead to a macular star configuration.



(a) Flame-shaped retinal haemorrhages (b) Severe disc oedema & macular star.

(Source: Clinical Diagnostic Ophthalmology by J.J. Kanski)

(iii) Arteriosclerosis: Results in the thickening of the retinal vessel wall which presents as “arterio-venous crossings.” Though, the presence of this arterio-venous crossings alone do not indicate the severity of the hypertension, it however tells us that the hypertension has been present in the individual for many years.



(a) Flame-shaped, dot-blot haemorrhages, retinal oedema & cotton spots.
(Source: *Clinical Diagnostic Ophthalmology* by J.J. Kanski)

The retinal changes that occur in hypertensive retinopathy can be grouped into four categories (Miller, 1996).

Group 1: Mild to moderate generalized arteriolar attenuation or sclerosis



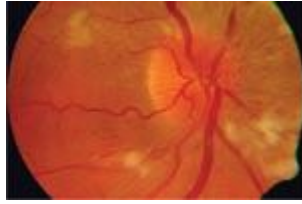
Focal & generalized arterial narrowing

Group 2: Moderate to marked generalized sclerosis of the retinal arterioles, exaggeration of the light reflex, arteriolar constriction associated with deflection of veins at the arterio-venous crossings (Salus’ sign).



Mild disc oedema-hallmark of malignant hypertension

Group 3: Copper-wiring appearance of the arterioles, banking of veins distal to the arterio-venous crossings (Bonnet's sign), tapering of veins on either side of the crossings (Gunn's sign).



Flame-shaped haemorrhages, cotton wool spots and hard exudates.

Group 4: Consists of all the characteristics in group 3 plus the silver-wiring of arterioles and disc swelling (papilloedema).

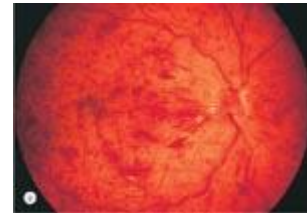


Severe disc oedema & macular star.

Apart from all the above features, there are other ocular manifestations associated with systemic hypertension. They include- branch retinal vein occlusion, branch retinal artery occlusion, retinal arterial macro-aneurysm, ischaemic optic neuropathy, ocular motor nerve palsies and exudative retinal detachment. Uncontrolled systemic hypertension also has adverse effects on diabetic retinopathy.



(a)BRVO-showing residual haemorrhages, macular hard exudates



(b)CRVO-showing severe disc oedema associated with peripapillary flame-shaped haemorrhages, cotton-wool spots, venous tortuosity with diltation.

Conclusion/Recommendation

Kudos to our Dean for initiating a public interest/enlightenment lecture of this nature. Atleast, it has given all of us here present the opportunity to assess the burden of eye problems confronting the elderly individual. These problems are real. The elderly in the society needs support, whether by their children, relatives, friends, governmental/non-governmental Organizations, etc., because we shall all get there, if we are lucky.

Research Reports have suggested that nutrition has a role to play in all age-related vision problems of the elderly except in glaucoma (Caffery, 2007). Inadequate antioxidant intake can lead to excess free radical formation that has direct impact on cataracts and macular degeneration. Free radicals also play some roles in the formation of arterial plaques resulting in vascular causes of visual loss such as retinal vein and arterial occlusions. It also influences the course of macular degeneration.

Excess fat intake can cause increased blockage of arteries leading to retinal and vein occlusion. Obesity is a risk factor for diabetes and diabetic retinopathy. Indeed, the overall health and nutritional status of the older adult must be of concern to the maintenance of good vision.

But we all know that older adults are particularly prone to nutritional problems due to their condition of living which include poverty, loneliness, neglect and sometime, mental disabilities. In fact, Ryan et al (2007), established that poor nutrition was a major problem to older adults in the US. He said that about 40% of those above 65years had inadequate nutritional intake.

Older adults should also be advised against smoking, excessive intake of alcohol and diets rich in cholesterol. Prevention is better than cure.

Finally, VISION is an essential part of everyday life. We all depend on it. Vision affects development, learning, communication, working, health, and quality of life.

The elderly adults are people too, for Sirach 3:12 says “My son, take care of your father when he grows old: give him no cause for worry as long as he lives. Be sympathetic even if his mind fails him; don’t look down on him just because you are STRONG and HEALTHY. ” And Colssians 3:14 says “And to all these qualities add LOVE.....” So, lets us all support our

elderly adults and show them with love, for indeed they have vision challenges.

THANK YOU

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