AGING AND THE EYE

Sumru Onal, Tayfun Bavbek
Department of Ophthalmology, School of Medicine, Marmara University, Istanbul, Turkey

ABSTRACT
Aging is a fundamental biologic phenomenon that occurs even in the absence of disease, each cell having a genetically programmed lifespan. Tissues that do not undergo mitotic division to replace this cell fallout, such as the central nervous system and the retina, have a high incidence of aging manifestations, particularly after 75 years of age. As the lens ages, it increases in weight and thickness and decreases in accommodative power. No method to halt the formation of age-related macular degeneration and/or senile cataract has been shown to be effective. Nevertheless, advances in the treatment of age-related macular degeneration and advances in surgical removal of cataracts have made treatment very effective. Three major topics related to aging and the eye that will be discussed here are presbyopia, age-related macular degeneration and senile cataract.

Keywords: Aging, Eye, Presbyopia, Age-related macular degeneration, Cataract

INTRODUCTION
Changes in the visual function with aging go beyond the need for eyeglass prescription. Frequently the physiological ocular changes that accompany aging interact with environmental or disease factors to cause visual impairment. Especially two eye diseases have high prevalence in the elderly: macular degeneration and cataract. In addition, the effects of systemic disease on the eye can cause visual impairment.

Visual impairment is the most common sensory problem faced by the elderly. Prevalence data accumulated from the National Health Interview study revealed that 12.8% of the elderly reported some visual problems, and this number increased to over 25% for those aged 85 years and older, 12% of whom were legally blind1. Perhaps the most common problem with aging and visual acuity is refraction. Almost 95% of those surveyed over age 65 years either wore glasses or reported needing glasses or some form of corrective lenses to improve visual acuity. However, only 45% of those over age 85 years reported that their glasses corrected all of their visual problems1.

The aging process itself has a number of effects on the eye, some of which have variable effects on vision. Physiological changes in the aging eye are: decreased tear viscosity, increased eyelid laxity, decreased color sensitivity, decreased light sensitivity and impaired lens accommodation2.

The almost universal impairment in the accommodation capability of older persons is caused primarily by an increase in the density and inelasticity of the lens. Decreased contrast sensitivity and increased susceptibility to glare also occur frequently and impair reading, driving, and detailed near vision. Color vision is generally

Corresponding author: Sumru Onal
Altunizade Mah. Okulcikmazi Sok.
No: 11/5 34660 Üsküdar Istanbul
E-mail: sumruo_md@yahoo.com

Marmara Medical Journal 2005;18(1);43-52

43
well preserved, although the progressive yellowing of the lens over time may interfere with blue-green vision. Because of the progressive narrowing of the pupil’s diameter and reduced translucency of the lens with advancing age the quantity of light striking the retina also decreases. Interaction of such normal aging changes with medication effects or with ocular manifestations of systemic illness may also result in a decrease in visual function3.

**Presbyopia**

Presbyopia is a normal part of the aging process and leads to a decrease in accommodation associated with loss of elasticity of the lens and lens capsule. Accommodative ability decreases with age. While a great deal of variability occurs in the normal level of accommodation, a general rule is that 6 diopters (D) of accommodation should be present at 40 years of age, 4D at 44 years of age, and 3D at 48 years of age. For each 4-year period under 40 years of age, 1D should be added; for each 4-year period over 48 years of age, 0.5D should be subtracted. This rule is suggested by Milder and Rubin4.

Treatment of presbyopia involves the use of plus lenses for near work. Several methods can be used to determine the proper add. The correction of emetropia and the use of trial frames to determine the proper add yields the best results. Other options for correction of presbyopia include the use of bifocal contact lenses5.

**Age-related Macular Degeneration**

Age-related macular degeneration (AMD) is not only the leading cause of legal blindness in patients aged 65 or over6, but also is now the overall cause of blindness in the Western world. It is estimated that in the United States 315000 people aged 75 and over will develop AMD over any 5-year period,7 and the incidence continues to rise as a result of the increasing percentage of elderly persons and the improved management of other eye diseases.

Senile macular degeneration was first reported as a clinical entity in 1885 by Otto Haab, who described a variety of pigmentary and atrophic changes in the macular region, causing progressive impairment of central vision in patients over the age of 508. Subsequent observers referred to the different fundus manifestations of the disease as separate entities, resulting in a variety of descriptive eponyms. A major step toward a better understanding of the disease was taken when Gass clarified that drusen, senile macular degeneration, and senile disciform macular degeneration represented a single disease9.

In recent years it has been proposed that the disease should be termed age-related maculopathy (ARM), early and late forms, with the term age-related macular degeneration AMD being reserved for the late forms and encompassing dry AMD (geographic atrophy) and wet (exudative, or neovascular ) AMD10,11. Early ARM represents those changes predisposing to AMD. The International Epidemiological Age-related Maculopathy Study Group defined early ARM as a degenerative disorder in persons ≥50 years of age10.

Normal aging results in a spectrum of changes in the macula, many clinically undetected, that effect the outer retina, retinal pigment epithelium (RPE), Bruch’s membrane, and choriocapillaries. Photoreceptors are reduced in density and distribution. Ultrastructural aging changes in the RPE include loss of melanin granules, formation of lipofuscin granules, and accumulation of residual bodies. Basal laminar deposits which are long-spacing collagen collecting between the plasma membrane of the RPE cells and the inner aspect of the basement membrane of the RPE accumulate. Lastly, progressive involutional changes occur in the choriocapillaries12.

Age-related Maculopathy: Nonneovascular Age-Related Macular Degeneration and the evolution of Geographic Atrophy:

Onset of ARM may present clinical features in the absence of drusen often characterized by diffuse mottling of small pigment clumps or as a microreticular pattern of small lines, more obvious on fluorescein angiography.

**Drusen**

Donders described drusen in 185413. Drusen are deposits of extracellular material lying between RPE and the inner collagenous zone of the Bruch’s membrane. Generally, classifications based on features of drusen morphology that are evident clinically are favored (Table I)12. Several population based surveys have found that drusen are present in more than 95% of people, with small hard drusen being most common in all age groups11,14-15. All types of drusen can undergo calcification, thus giving the druse a glistening appearance. Calcification of soft drusen usually precedes drusen regression and the development of RPE atrophy.
**Table I: Clinicopathologic Classification of Drusen**

<table>
<thead>
<tr>
<th>Type of Druse</th>
<th>Clinical Description</th>
<th>Histologic Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small, Hard (hyalinized)</td>
<td>Yellow, well-d-emarcated boundaries; usually &lt;63 µm diameter; can be evident in the second decade of life; tend to hyperfluoresce on fluorescein angiography</td>
<td>Hyalinized material containing membrane-bound bodies external to the RPE basement membrane; can exist in the absence of basal laminar deposits</td>
</tr>
<tr>
<td>Soft (pseudosoft), cluster-derived</td>
<td>Yellowish with indistinct margins along some portions of the druse’s perimeter; usually occur after age 55 years; variable staining on fluorescein angiography; can regress, leading to atrophy</td>
<td>Fused hard drusen cluster in which the amorphous internal rim of the druse is interrupted, forming globular or finely granular material</td>
</tr>
<tr>
<td>True soft</td>
<td>Yellowish with indistinct margins; smaller than soft cluster-derived drusen; confined to the macula; precursor to choroidal neovascularization</td>
<td>Associated with accumulation of membranous debris external to the RPE basement membrane; consists of focal accentuation of basal laminar deposits, focal accentuation of basal linear deposits, or localized accumulation of basal linear deposits</td>
</tr>
<tr>
<td>Granular</td>
<td>About 250 µm diameter; yellow, solid appearance, their confluence resulting in crescentic sinuous shapes</td>
<td>Coarsely granular structure consisting of membrane-bound globules of amorphous material, small membrane fragments and cellular debris</td>
</tr>
<tr>
<td>Fluid (Serous)</td>
<td>Soft, confluent drusen &gt;500 µm diameter; may have pooled serous fluid in the lipoidal debris; further confluence leads to larger soft fluid drusen that resemble serous pigment epithelial detachments</td>
<td></td>
</tr>
<tr>
<td>Membranous (accumulation of basal linear deposits)</td>
<td>Paler and shallower than the yellow granular drusen; usually &lt;250 µm diameter; on fluorescein angiography they fluoresce later and less brightly than small, hard drusen</td>
<td></td>
</tr>
<tr>
<td>Reticular (pseudodrusen)</td>
<td>Yellowish interlacing network about 250 µm diameter; first appear in the superior outer macula; resembles soft confluent drusen, but is flat and lie deep to drusen; delayed choroidal perfusion on fluorescein angiography; carries a very high risk for choroidal neovascularization</td>
<td>Uncertain; the pattern has been suggested to result from fibrous placement of the middle layer of the choroid</td>
</tr>
<tr>
<td>Regressing (fading)</td>
<td>All drusen types may disappear in time; does not signify a return to normal state; fluorescein angiography generally shows increased transmission of fluorescence where drusen have faded</td>
<td>Both RPE and photoreceptors over regressing drusen disappear, leaving a thick layer of late-type amorphous basal laminar deposit over the apex</td>
</tr>
</tbody>
</table>
On fluorescein angiography soft drusen fill more slowly and are not as brightly fluorescent as hard drusen, but they remain fluorescent for a longer period\(^\text{16}\). On indocyanine green angiography (ICG) hard drusen become hyperfluorescent 2 to 3 minutes after dye administration, and this persists throughout the middle and late phases. Soft drusen are either hypofluorescent throughout the angiogram or remain undetectable\(^\text{17}\).

The cumulative incidence of late ARM in patients with bilateral drusen has been reported in two prospective studies. Among patients attending the ophthalmology clinic in England the 3-year cumulative incidence of late ARM and of exudative AMD alone were, respectively, 23.5% and 18%, one significant risk factor being degree of confluence of drusen within 1600 \(\mu\text{m}\) of the center of the fovea\(^\text{18}\). In the Beaver Dam study of persons with signs of early ARM in both eyes at baseline, the respective figures at 5 years were 11% and 7.1%\(^\text{19}\). In patients who have developed choroidal neovascularization (CNV) in the first eye, the presence of five or more drusen, or one or more large drusen, were two factors associated independently with an increased risk of developing CNV within 5 years in the second eye\(^\text{19}\). Another prospective study followed 101 patients with unilateral exudative AMD and drusen only in the fellow eye for up to 9 years. Yearly incidence rates for the development of CNV or geographic atrophy in the fellow were between 5% and 14%. The risk of CNV peaked at 4 years and decreased thereafter. The risk of CNV in patients with ARM was heralded by an increase in the number, size and confluence of drusen. The risk eventually declines and is followed by later increased risk of geographic atrophy\(^\text{20}\). This risk is also reflected in pathologic specimens, in which active subretinal new vessels are more likely to be associated with soft membranous-type drusen.

**Geographic Atrophy**

Geographic atrophy (GA) is the end result of atrophic AMD and is currently defined as any sharply delineated round or oval area of hypopigmentation or apparent absence of RPE, in which choroidal vessels are more visible than surrounding areas and which must be at least 175 \(\mu\text{m}\) in diameter\(^\text{10}\). However since such small area could result from regression of a single druse, other dimensions proposed have been wider, varying from 200 \(\mu\text{m}\)\(^\text{14}\) to 1 mm\(^\text{23}\). If a larger size is chosen, it has greater prognostic significance, especially if the atrophy has already entered the fovea, because CNV then is less likely to develop or, if it should occur, is more likely to be muted.

The evolution of GA can be drusen-unrelated, drusen-related, or following pigment epithelial detachments\(^\text{24}\). Among patients with early ARM the 5-year incidence of pure GA atrophy has been found to be 4.6%\(^\text{7}\). Once GA has commenced, the factors that influence the rate and direction of further spread are the number, distribution, and regression of drusen and the extent of incipient atrophy (RPE thinning). When this extends to the central fovea, visual acuity can be expected to drop more rapidly. In general, the percentage of foveal involvement increases rapidly at first, slowing once all the area of incipient atrophy has become involved. Histological studies have shown that eyes with geographic atrophy may also contain small inactive CNV and that CNV is more frequently bilateral than clinical impressions suggest\(^\text{25,26}\).

Neovascular (Exudative) Age-related Macular Degeneration:

AMD is the major cause of severe visual loss in older adults\(^\text{11,15,27-31}\). Most AMD patients have macular drusen or retinal pigment epithelial abnormalities or both\(^\text{32}\). However, approximately 10% of AMD patients manifest the neovascular form of the disease\(^\text{33}\). Neovascular AMD includes CNV and associated manifestations such as retinal pigment epithelial detachment, retinal pigment epithelial tears, fibrovascular disciform scarring, and vitreous hemorrhage\(^\text{32}\).

The prevalence of AMD-associated vision loss in at least one eye increases with age\(^\text{11,14,15,27,28,30,31,34,35}\). AMD was the leading cause of blindness in white (prevalence, 2.7 per 1000) but not black subjects randomly selected in the Baltimore Eye survey. In this study, AMD resulting in blindness affected 3% of all white subjects 80 years of age or older\(^\text{31}\). Risk factors implicated in clinical and laboratory studies include drusen, visible (but not ultraviolet) injury, micronutrient deficiency, cigarette smoking, family history (genetic predisposition), and cardiovascular risk factors (including systemic hypertension)\(^\text{36,37}\).
Choroidal neovascularization

CNV appears as a neovascular sprout growing under or through the RPE through breaks in Bruch’s membrane. Blurred vision and distortion, especially distorted near vision, are the symptoms most patients with CNV notice. Patients may also complain of decreased vision, micropsia, metamorphopsia, or a scotoma. Visual acuity, although frequently decreased, may not always be affected. Functional vision generally declines in accordance with Snellen visual acuity.

In some patients with AMD, CNV may appear as a gray-green elevation of tissue deep to the retina with overlying detachment of the neurosensory retina. The gray-green color may arise from hyperplastic RPE in response to the CNV. However, it is not always present in older individuals with AMD. Often, the presence of blood or lipid or a sensory retinal detachment in an elderly patient with vision loss indicates the presence of CNV.

Retinal Pigment Epithelial Detachments (PED)

Retinal PED appear clinically as sharply demarcated, dome shaped elevations of RPE. They usually transilluminate if they are filled with serous fluid only. Although an overlying sensory retinal detachment may be a clue to the presence of CNV beneath a PED, sometimes a shallow neurosensory detachment may occur as a result of breakdown of the physiologic RPE pump or from disruption of the tight junctions between adjacent RPE cells in the absence of CNV. The presence of a PED may or may not be a feature of CNV. The fluorescein angiographic pattern can differentiate a drusenoid PED, which does not have CNV, from a fibrovascular PED, which is a form of occult CNV, as well as from serous PED, which may or may not overlie an area with CNV. Several clinical signs suggest the presence of CNV underlying an PED, including overlying sensory retinal detachment and lipid, blood, and chorioretinal folds radiating from the PED. When confined to the sub-RPE space, the blood may appear as a discretely elevated, green or dark red mound. The hemorrhage can dissect through the RPE into the subretinal sensory retinal space or into the retina. Rarely, blood may pass through the retina into the vitreous cavity, causing extensive vitreous hemorrhage.

Disciform Scars

Histologically, CNV usually is accompanied by fibrous tissue, even when no fibrous tissue is readily apparent on initial presentation. This fibrous tissue may be accompanied by CNV (fibrovascular tissue) or not (fibroglial tissue). The fibrous tissue complex may be beneath the PRE (usually proliferating within the inner aspect of an abnormally thickened Bruch’s membrane) or between the RPE and the photoreceptors. Often, over time, the plane of the RPE is destroyed by the fibrovascular or fibroglial tissue, so the location of the CNV with respect to RPE can no longer be identified readily. When the fibrous tissue becomes apparent clinically, the CNV and fibrous tissue complex may be termed a “disciform scar.”

Classification of CNV using Fluorescein Angiography

Whenever one suspects CNV for which treatment might be indicated, stereoscopic fluorescein angiography should be performed promptly. Fluorescein angiography frequently allows one to determine the pattern (classic or occult), boundaries (well defined or poorly defined), and location of the neovascular lesion with respect to the foveal avascular zone (FAZ). Classic CNV appears as a well-demarcated area of uniform hyperfluorescence surrounded by a hypofluorescent margin in the early phase frames of the angiogram, with fluorescein leakage that obscures the boundaries of the lesion through the mid- and late-phase frames. Occult CNV is divided into two types. Type I is characterized by a fibrovascular RPE detachment that appears as stippled hyperfluorescence with irregular elevation of this fluorescence at the level of RPE, usually within 1-2 minutes of fluorescein injection. The boundaries are often poorly defined or difficult to demarcate and there is fluorescein leakage in the late phase frames of the angiogram. Type II has poorly demarcated boundaries with fluorescein leakage from an undetermined source at the level of RPE in the late phase frames of the angiogram, which do not correspond to classic CNV or fibrovascular PED in the early- or mid-phases. Lesion composition is assessed using the fluorescein angiography and the classification is defined by the proportion of the lesion that is classic CNV. Predominantly classic lesions are defined as those in which the classic component comprises 50% or more of the entire lesion. Lesions in which the classic component comprises less than 50% of the entire lesion area are defined as minimally classic and those with no evidence of classic CNV are defined no classic. The terms well defined (well demarcated) and poorly defined (poorly or ill demarcated) refer to a description of
the boundaries of the lesion. In a well-defined lesion, the entire boundary for 360 degrees is well demarcated. If the entire boundary is not well demarcated for 360 degrees, then the lesion is poorly defined. These terms describe lesion boundaries for a lesion that may be composed of classic CNV, or occult CNV, or both. When lesions have well-defined boundaries, the CNV lesion can be classified according to the location of the most posterior boundary with respect to the center of the FAZ on the fluorescein angiogram. CNV lesions located more than 200 µm from the FAZ center are termed extrafoveal; those between 1 and 199 µm from the center are juxtafoveal; CNV lesions extending under the center of the FAZ are termed subfoveal. In contrast to other pathologic conditions predisposing to CNV in eyes with AMD, CNV presents more commonly under the FAZ center46.

Treatment

Laser photocoagulation has been shown to be beneficial only for well-defined lesions. If the entire boundary of the lesion is not well defined, then the treating ophthalmologist can not determine where to apply laser photocoagulation with certainty in order to cover the lesion its entirety; undertreatment or overtreatment will occur likely. Failure to cover the entire lesion increases the likelihood of recurrent CNV47-49 and, for extrafoveal and juxtafoveal lesions, additional visual acuity loss47,49,50. Overtreatment likely will destroy retinal tissue and corresponding function that was not overlying CNV unnecessarily49.

The decision for laser photocoagulation should be made considering the size of the CNV and the initial visual acuity. Specifically, the smaller the lesion and the better the visual acuity at the time of treatment, the greater will be the benefit. Laser treatment may not be indicated in three common presentations: if only occult CNV is noted in a subfoveal lesion; if the boundaries of the entire neovascular lesion are not well demarcated; and if a subfoveal lesion is large (>3.5 MPS disc diameter). A patient undergoing treatment for an extrafoveal or juxtafoveal lesion should understand that this therapy will not improve existing vision and will induce a permanent scotoma46.

The choice of wavelength for photocoagulation (green or red) appears to have no effect on the treatment benefit. The risk of recurrence appears greatest in the following situations: when the fellow eye has evidence of active CNV or scarring; when treatment fails to cover the neovascular lesion in its entirety; and when photocoagulation is not as intense as a moderately white treatment intensity standard46.

Photodynamic therapy (PDT) involves the use of an intravenously injected photosensitizing drug combined with a low-intensity laser light to cause damage of choroidal neovascular tissue selectively through a photochemical reaction by the light-activated drug that appears to result in direct cellular injury, including damage to vascular endothelial cells and vessel thrombosis51,52. It has been shown that PDT with verteporfin (Visudyne) can safely reduce the risk of moderate and severe vision loss in patients with subfoveal lesions that are predominantly classic CNV secondary to AMD. While this benefit seemed to be even greater in the absence of occult CNV, the effect may be related to the smaller lesions and worse visual acuity associated with predominantly classic lesions without occult CNV and not solely to the lesion composition itself53. Additionally, lesion size in the TAP Investigation and VIP Trial was also an important predictor of the magnitude of treatment benefit with verteporfin therapy in occult with no classic and minimally classic lesion compositions54.

Potential future treatments for CNV in AMD are submacular surgery, radiation therapy and pharmacologic therapy with angiogenesis inhibitors46.

Senile Cataract

Cataract, an opacity of the lens that impairs vision, is the most common cause of visual loss in humans. Any opacity in the lens or its capsule whether congenital or acquired is known as cataract55.

Throughout the world the elderly population is increasing. For the period 1980-2020 the projected increase in the elderly population for the developed world is 186%, while in developing countries the projected increase is 356%. On this basis, the World Health Organization estimates that 54 million blind people aged 60 years or more will occur by the year 202056.

Based on morphology cataract can be classified as capsular, subcapsular, cortical, supranuclear, nuclear, lamellar and sutural cataract. Cataracts have also been classified according to developmental stage. These are: immature, intumescent, mature, hypermature and Morgagnian cataract. Etiological classification is ideally considered as the most widely accepted classification of cataract. Various types of
cataracts under this classification are congenital, developmental, senile, metabolic, traumatic, toxic and secondary cataract and cataracts associated with systemic diseases\textsuperscript{57}.

The most common type of cataract encountered is senile or age-related cataract. It is an affection of advanced life and is essentially an aging process. Sometimes there appears to be a familial tendency for cataract in which case the condition may develop at an earlier age in successive generations and phenomenon is known as anticipation and as a rule is usually bilateral but develops earlier in one eye than the other. Usually some degree of cataract is present after the age of 50 years and it equally affects both sexes. Although the precise etiopathogenesis is not clear, yet the various factors involved in senile cataractogenesis are hereditary factors, cigarette smoking, alcohol use, ultraviolet irradiation, dietary factors, and severe dehydration\textsuperscript{58-61}.

Senile cataract is of various types and occurs in subcapsular, cortical and nuclear regions of the lens. The locations of the predominant senile cataract have been shown to be cortical in 70\%, nuclear in 25\%, and subcapsular in 5\% of cases\textsuperscript{57}.

Subcapsular Senile Cataract (Cupuliform Cataract)

These cataracts may be anterior or posterior and are seen as brown granules and cysts in the shape of a shallow cup in the subcapsular region. The anterior subcapsular cataract lies directly under the lens capsule and is associated with fibrous metaplasia of the anterior epithelium of the lens. The posterior subcapsular cataract lies just in front of the posterior capsule and is associated with posterior migration of epithelial cells of the lens. These cataracts usually develop in 60 to 80 year-old age group, but may be present in an inherited form at an earlier age. Patients with posterior subcapsular cataract specifically get troubled by bright sun light and headlights of incoming vehicles\textsuperscript{57,62}.

Cortical Senile Cataract (Cuneiform or Soft Cataract)

Cortical senile cataract is the most common form of senile cataract. This type of cataract is characterized by opacities in cortical fibers and appears to be due to an accumulation of globules and vacuoles between adjacent fibers. There is hydration due to accumulation of water droplets between the fibers followed by changes in the colloid system within the fibers. The lens proteins are first denaturated and then are coagulated forming opacity. Ultimately the whole lens becomes opaque and assumes a pearly white appearance. The appearance of cuneiform cataract with its vacuoles, radial spoke–like separation of lens fibers and wedge-shaped water clefts and shield–like configuration is characteristic. Cuneiform opacities represent areas in which lens fiber membranes get damaged allowing sodium influx and osmotic inhibition of water. Increased membrane permeability and inactivation of active transport process in these areas leads to loss of potassium, glutathione, soluble protein and inositol. These biochemical changes eventually lead to precipitation, opacification and aggregation of lens proteins. If the whole cortex is opacified such a cataract is known as mature cataract\textsuperscript{57,62}.

Nuclear Senile Cataract

In this type of senile cataract the nucleus gradually becomes opaque and cortex being clear. Increased optical density of the nucleus occurs normally with aging but it may be stimulated to excess with the formation of brown nucleus (brunescent cataract) or even a black nucleus (cataracta nigra) and is usually bilateral. Dehydration and compaction of nucleus are associated with the process of nuclear sclerosis. The sclerotic process renders the lens inelastic and hard and decreases its ability to accommodate. At first a certain degree of myopia is induced. These changes start centrally and speed towards periphery. This type of cataract does not develop into hypermature stage. The progress of cataract is slow and myopic eyes are more prone to develop this type of cataract. Biochemical changes in nuclear senile cataract include an increase in the concentrations of protein, corresponding decrease in the degree of hydration, marked increase in sodium level along with decrease in potassium concentration. These changes are associated with accumulation of yellow-brown pigment urochrome which may represent an oxidation product of amino acids or lipids. Nuclear sclerosis begins between 50 to 60 years of age and progresses very slowly unless accelerated by the superimposition of subcapsular cataract\textsuperscript{57,62}.

Treatment

Treatment of cataract essentially consists of its surgical removal. The indications for a particular lens surgery technique to be used may be determined by several factors. Different conditions or pathological states of the eye and the lens may dictate the use of one technique over another. In different countries, the availability of
equipment and the level of training of the surgeon may be factors that dictate the technique.

**Intracapsular Cataract Extraction**

Intracapsular Cataract Extraction (ICCE) is an old traditional method of cataract removal. In ICCE the entire lens is removed with cryoprobe or capsule forceps. This method has not been the procedure of choice in industrialized nations since the development of modern extracapsular techniques in the late 1970’s, primarily because of lower rates of postoperative posterior segment complications as hemorrhage, vitreous loss, retinal detachment, and cystoid macular edema. Posterior chamber intraocular lens (IOL) implantation is impossible as the posterior capsule is absent due to total removal of the lens and its capsule

**Extracapsular Cataract Extraction**

Extracapsular Cataract Extraction (ECCE) became popular in the 1980’s. The lens is removed without disturbing the integrity of the posterior capsule and anterior vitreous face. ECCE involves the excision of a portion of anterior capsule and nucleus expression through incision and aspiration of the residual equatorial cortex. The posterior capsule is left undisturbed and serves as resting site for the posterior chamber IOL implants. Postoperative posterior capsule opacification (PCO) may occur in a significant number of cases. PCO can be managed using neodymium-yttrium-aluminum-garnet (Nd:YAG) laser capsulotomy.

**Phacoemulsification**

Kelman devised this technique about 25 years ago. It differs from ECCE in that corneoscleral incisions required are very small (3.0 mm), central continuous curvilinear capsulorrhesis of about 4-5 mm is preferred over other methods of anterior capsulotomy, the use of foldable IOLs and in that the nucleus is emulsified and aspirated in the posterior chamber by the phacoemulsifier which acts through a titanium needle in a longitudinal axis at an ultrasonic speed of 400,000 times a second. Smaller incisions, more rapid wound healing, short convalescence and early stabilization of refractive error with less astigmatism are major advantages.

New and future technologies are phaconit, laser cataract surgery using Nd:YAG or Er:YAG laser systems, laser phacoit and focused electromagnetic field technology.

**REFERENCES**


