9

OPTICAL AND RELATED CHANGES IN THE AGING EYE

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Although many of the age-related changes in the lens, ciliary muscle, and other structures concerned with accommodation have been discussed in Chapters 7 and 8, additional optical and related changes also have an effect on the quality of vision of the presbyope. This chapter will consider some of the relevant changes in individual components and their impact on refraction, retinal image quality, and visual performance.

CHANGES IN OPTICAL COMPONENTS

Cornea and Tear Film

Unlike those of the lens, the corneal form and structure do not alter markedly with age. Nevertheless, some optically relevant alterations do occur. Corneal curvature, thickness, and refractive index remain essentially constant, but there is usually a slow drift from predominantly with-the-rule (direct) to against-the-rule (inverse) corneal astigmatism,[1,2] largely as the result of steepening in the horizontal meridian[3] and some change in the asphericity of both the anterior and posterior surfaces.[4] In the absence of any disease process, it appears that, although tear production is reduced and evaporation rates are higher in the older (>45 years) eye,[5] a tear film of satisfactory optical quality is usually maintained.[6] This may not be the case in the months following excimer laser refractive surgery, when dry eye symptoms may be experienced.[7]

Light scattering in the cornea increases to, at most, only a minor extent with age,[8] and there is little change in direct spectral transmittance, which shows a sharp cutoff below approximately 320 nm.[9,10]

Although not strictly optical changes, the observed age-dependent declines in corneal sensitivity[11] and endothelial cell density[12] (Figure 9-1) are of interest, as they have obvious relevance to contact lens and other methods of refractive correction. Due to damage to the corneal nerves, sensitivity is compromised for some months after both photorefractive keratectomy and LASIK, with the latter having greater effect.[13,14] The decline in endothelial cell density may be accelerated by cataract surgeries.[15]

Aqueous

Due to its constant replacement, the aqueous normally retains its clarity and refractive index throughout life, although transparency can be reduced as a result of pathology such as uveitis in response to infection.

Pupil

The pupil is the aperture stop for the optical system of the eye and, as such, is of great importance to

Pallikaris IG, Plainis S, Charman WN.
Presbyopia: Origins, Effects, and Treatment (pp. 77-84).

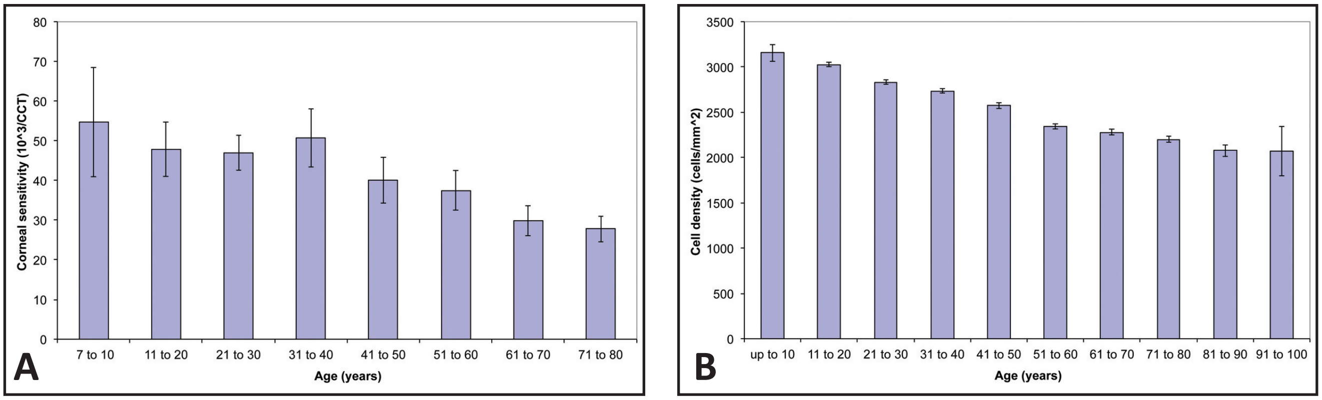

Figure 9-1. Changes in (A) peripheral corneal sensitivity as a function of age, expressed as the reciprocal of the corneal touch threshold (CTT) X 1000, and (B) endothelial cell density. Error bars represent standard errors of the mean. (Based on data compiled from Millodot M. The influence of age on the sensitivity of the cornea. *Invest Ophthalmol Vis Sci.* 1977;16(3):240-242 and Abib FC. Behavior of corneal endothelial density over a lifetime. *J Cataract Refract Surg.* 2001;27(10):1574-1578.)

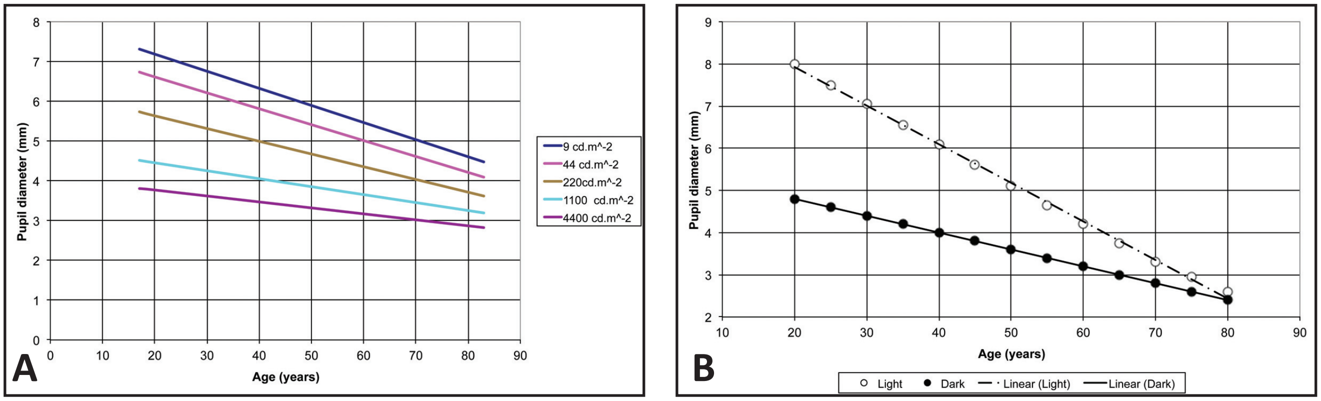

Figure 9-2. (A) Mean entrance pupil size under monocular viewing conditions as a function of age for a 10° diameter circular field illuminated to the luminance levels indicated (after Winn et al[16]) (B). Fully dark- and light-adapted entrance pupil diameters as a function of age. (Based on data compiled from Kornzweig AL. Physiological effects of age on the visual process. *Sight-Saving Rev.* 1954;254:130-138.)

through-focus retinal image quality. This is particularly the case with eyes that have been given bifocal, multifocal, or varifocal simultaneous-vision presbyopic contact lenses or corneal or IOL corrections, where the quality of distance and near vision achieved is heavily dependent upon the interaction of the pupil diameter with the optical characteristics of the correction (see Chapters 14 and 17). Corrections that work well with larger pupils may be ineffective if the pupil diameter is reduced and vice-versa.

As is well known, the pupil diameter under constant lighting conditions reduces progressively with age at all luminance levels (senile miosis). This decrease is essentially linear with age and starts in the adolescent years, well before the onset of presbyopia. Although exact pupil diameters depend on the patient group and measurement conditions, some typical monocular data for the changes with age at a series of photopic luminance levels are shown in Figure 9-2A,[16] and further data for maximal and minimal diameters are shown in Figure 9-2B.[17] For the same field luminance, pupil diameters under binocular conditions are usually smaller than those under monocular conditions, although they become the same at extremely low or extremely high light levels.[18] The position of the center of the pupil may change by up to 0.5 mm as the luminance level varies or when the pupil is dilated pharmacologically.[19]

The smaller pupil of the older eye may be beneficial in reducing the impact of ocular aberrations and light scatter (see next), in increasing DOF, and in protecting the already vulnerable older retina from phototoxic damage. However, the reduction in useful light flux reaching the retina has a modest adverse effect on mesopic and scotopic vision.

TABLE 9-1. *REPRESENTATIVE REGRESSION-LINE FITS TO DIMENSIONAL DATA (MM) FOR THE LENS AS A FUNCTION OF AGE (YEARS)*

PARAMETER	IN VITRO REGRESSION (FULLY ACCOMMODATED)	IN VIVO REGRESSION (UNACCOMMODATED)
Equatorial diameter (mm)	D = 0.0138*age + 8.7	D = 9.2
Sagittal thickness (mm)	T = 0.0123*age + 3.97	T = 0.0238*age + 2.93
Anterior radius of curvature (mm^{-1})	r_a = 0.046*age + 7.5	r_a = 12.9 – 0.057*age
Posterior radius of curvature (mm^{-1})	r_p = –5.5	r_p = –6.2 + 0.012*age

Note. The in vitro data (Rosen et al[20]) refer to isolated lenses, which are thought to take up a fully accommodated form when free of the constraints of the zonule. The unaccommodated in vivo data for diameter and thickness are from Strenk et al[21] (based on magnetic resonance imaging), and the radii are from Dubbelman and van der Heijde[22] (based on Scheimpflug photography).

Lens

As discussed more fully in Chapter 7, the lens undergoes a gradual change in its dimensions, surface curvatures, and refractive index distribution due to the progressive addition of new fibers as it ages. Typical regression data for some of these age changes are provided in Table 9-1.[20-22] In the unaccommodated state, the surface radii decrease with age but there is little change in lens diameter. Although there remains some controversy about the exact changes in the index gradients, Figure 9-3 shows some representative estimates.[23] These suggest the presence of much steeper gradients in the older eyes, with most of the index changes occurring close to the lens surface. These changes are likely to affect the optical power and aberrations of the eye.

The noncataractous lens also suffers from a gradual loss in transmittance (ie, an increase in optical density), particularly at the blue end of the spectrum,[24] caused by increases in light absorption and scattering. The increase in density, which is found at all wavelengths, starts from birth and continues throughout life.[24,25] Figure 9-4 shows representative data for young and old lenses. Exact values of transmittance depend on the ray path and measurement conditions used, as the absorption and scattering effects differ in the nucleus and cortex.[26]

Vitreous

The main change with age is that the vitreous gel is gradually replaced by unbound water so that more movement of the vitreous becomes possible. Optically, the chief effect is that increased number and movement of "floaters" may occur, these being the shadows on the retina of clusters of fibers and other debris in the vitreous chamber. However, the overall transmittance is changed little.

Axial Length

Although there have been numerous transverse and longitudinal studies on the changes in axial length through infancy and childhood to young adulthood, the possibility of further changes through life has received little attention. One exception to this is the work of Grosvenor,[27] who hypothesized, based on the transverse studies of Sorsby et al[28] and other related work, that axial length decreased by approximately 0.6 mm (equivalent to a hyperopic shift of roughly 2.00 D) through adulthood and that this played a role in compensating for changes in the refractive power of the eye and maintaining approximate emmetropia. No longitudinal studies appear to have been conducted to follow up this hypothesis, which has obvious relevance to the long-term stability of refractive correction with IOLs.

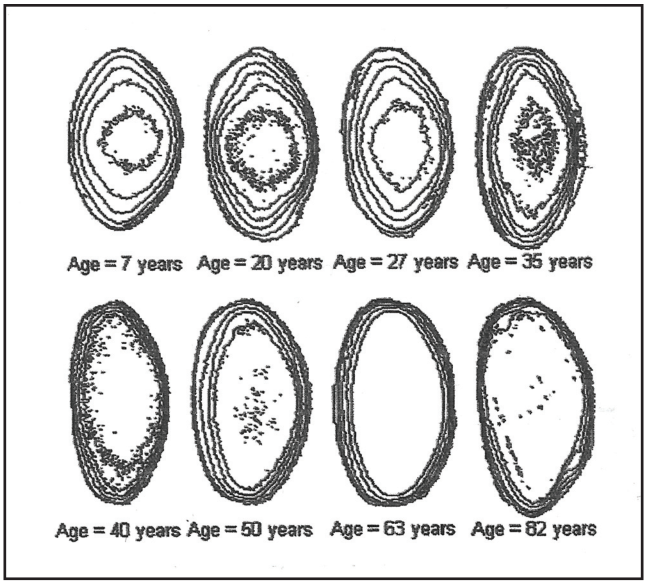

Figure 9-3. Changes in the iso-index contours for in vitro lenses of different ages (sagittal sections with the anterior vertex to the left). The contours are at intervals of 0.01 in refractive index, ranging from 1.36 to 1.43. The lenses are probably in a maximally accommodated form. (Reprinted from *Vision Res,* vol 45(18), Jones CE, Atchison DA, Meder R, Pope JM, Refractive index distribution and optical properties of the isolated human lens measured using magnetic resonance imaging, pp 2352-2366, Copyright 2005, with permission from Elsevier.)

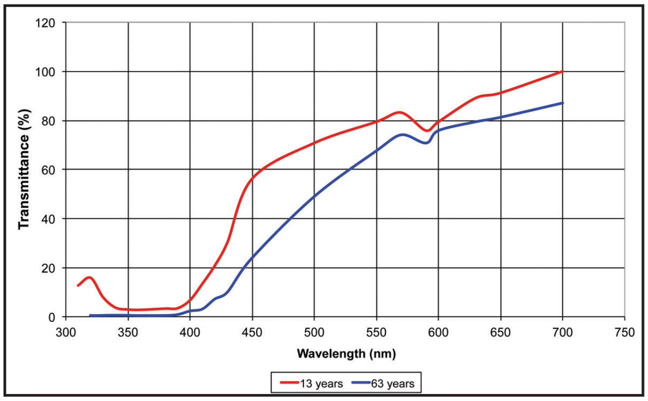

Figure 9-4. Spectral transmittance of the lenses of the eyes of a 13- and a 63-year-old patient. (Based on data compiled from Weale RA. Age and the transmittance of the human crystalline lens. *J Physiol.* 1988;395(1):577-587.)

EFFECTS IN COMPLETE EYE

Changes in Refraction With Age

Although refraction in individual eyes may follow a variety of courses, with myopic shifts in childhood being common, it appears that slow drifts in mean spherical refraction typically occur in all eyes. The broad trends are shown in Figure 9-5[29] where continuous curves are transverse data based on self-selected phakic clinical patients, whereas isolated symbols refer to randomly selected patients. As can be seen, there is general agreement that a progressive reduction in levels of hyperopia in childhood and early adulthood is followed at approximately age 40 by a gradual drift back toward low hyperopia, with the strong suggestion of a reversal of this trend at approximately age 70 years. Data of the type shown in Figure 9-5 can be criticized on the basis of possible patient selection effects, but the observed trend of a hyperopic refractive shift of approximately 1.00 D between ages 40 and 70 years appears real and poses problems for the long-term effectiveness of some types of surgical presbyopic correction.

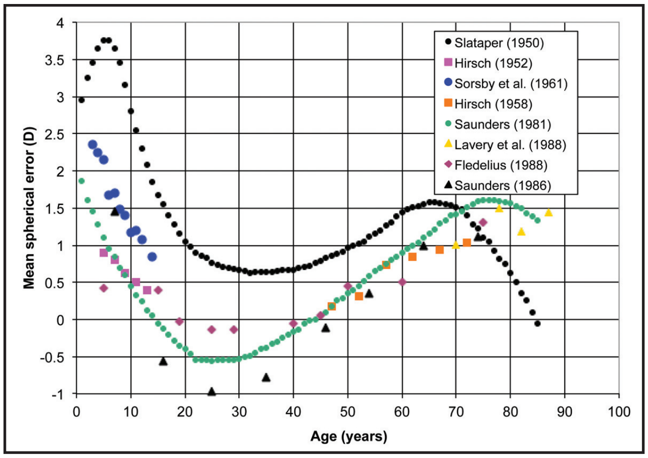

Figure 9-5. Changes in mean ocular refraction with age as found in different cross-sectional studies. The continuous curves are studies based on self-selected clinical patients, and the isolated symbols are based on randomly selected, non-clinical groups. Absolute levels of refraction differ between studies as a result of factors such as patient selection criteria and the use of either overall means or modes. (Adapted from Young FA, Leary GA. Refractive error development in relation to the development of the eye. In: Charman WN, ed. *Vision and Visual Dysfunction, Vol.1: Visual Optics and Instrumentation*. Basingstoke, UK: Macmillan; 1991:29-44.)

It is of interest that Donders[30] also noted the hypermetropic shift in midlife, which he termed *hypermetropia acquisita*. Such a shift may appear surprising, given the decrease in the radii of curvature of the unaccommodated lens with aging (see Table 9-1), which might be expected to lead to an increase in myopia. This "lens paradox," as it is often called, appears to occur due to the changes in the internal refractive index gradients of the lens (see Figure 9-3), which lead to a reduction in the contribution made by these gradients to the overall power of the lens.

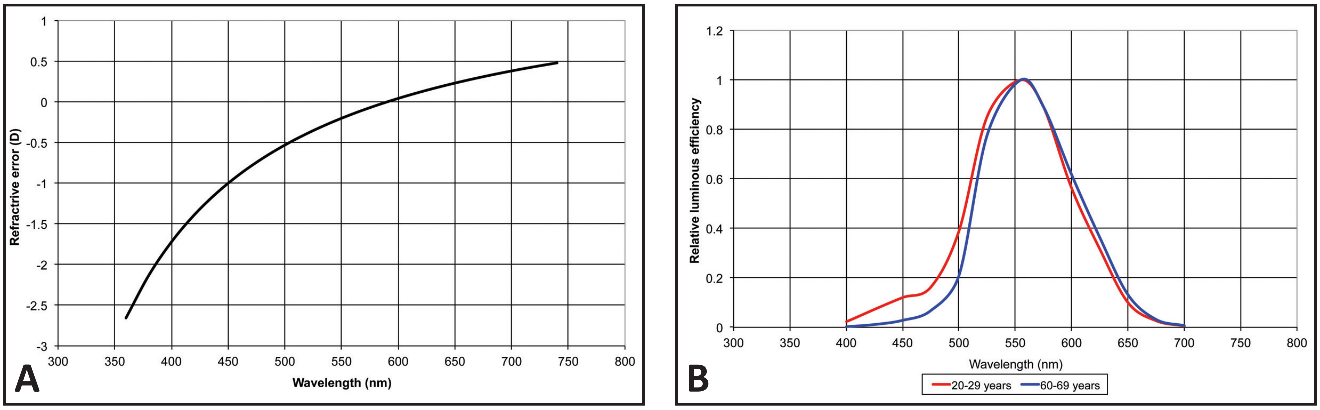

Figure 9-6. (A) Longitudinal chromatic aberration of the eye. This is independent of age. (Adapted from Thibos LN, Ye M, Zhang X, Bradley A. The chromatic eye: a new reduced-eye model of ocular chromatic aberrations in humans. *Applied Opt.* 1992;31(19):3594-3600.) (B) Relative photopic spectral luminous efficiency for 2 age groups: 2-degree field, flicker method. (Adapted from Sagawa K, Takahashi Y. Spectral luminous efficiency as a function of age. *J Opt Soc Am A.* 2001;18(11):2659-2667.)

Total Ocular Aberrations

Monochromatic higher-order wave aberrations at a constant pupil diameter vary idiosyncratically with the individual and tend to increase with age. In contrast, longitudinal chromatic aberration is constant through adult life and varies little across the population. Little is known about possible changes with age in the small amounts of subject-dependent transverse chromatic aberration that are experienced in foveal vision.

Figure 9-6A shows mean values of ocular longitudinal chromatic aberration.[31] It can be seen ocular longitudinal chromatic aberration amounts to approximately 2.00 D across the visible spectrum. The associated optical blur is, however, reduced by the relative insensitivity of the eye to the extremes of the visible spectrum. Figure 9-6B shows examples of the photopic relative luminous efficacy curves[32] for younger and older patients and emphasizes that spectral sensitivity is heavily weighted toward the center of the visible spectrum, so that out-of-focus images produced by the extremes of the spectrum contribute little to retinal illuminance; note the effects of lens absorption on sensitivity to blue light in the older patients.

The monochromatic wave aberration increases with age. In the younger eye, the aberrations of the cornea are approximately balanced by those of the internal optical elements, which have an opposite sign. However, as the eye ages, the lenticular aberration changes. This destroys the balance between corneal and internal aberration, although the corneal aberration remains roughly constant.[33] In particular, the spherical aberration of the eye becomes more positive with aging. Typical summary data for the total mean ocular root-mean-square higher-order wavefront aberration of the whole eye as a function of age group and pupil diameter are shown in Figure 9-7.[34] The levels of aberration associated with spherical defocus of 0.125, 0.25, and 0.50 D are included for comparison. It can be seen that the blurring effects of higher-order aberrations are likely to be modest in younger eyes but start to be important after the age of approximately 50 years, particularly at larger pupil diameters.

However, as noted previously, the effect of increases in total aberration across the pupil of the older eye is mitigated by the decrease in pupil diameter at a fixed light level. For example, Figure 9-2A shows that under conditions where the pupil of a 20 year old is 5 mm in diameter, it is only 4 mm in a 60 year old. The result is that, with natural pupils, optical image quality that is governed by wave aberration, chromatic aberration, and diffraction at any specific light level is much less affected by age than might be expected from Figure 9-7. Because corneal aberrations are approximately constant with age, an implant lens with appropriate aberration characteristics designed to correct those of the cornea should prove of lasting value.

Scattered Light

A variety of diffractive and refractive forward-scattering effects cause relative wide-angle light scattering to occur in the eye media, particularly in the cornea and lens. Scattered light may also originate at the retina. Scattering remains relatively constant through earlier adult life[35] but increases fairly

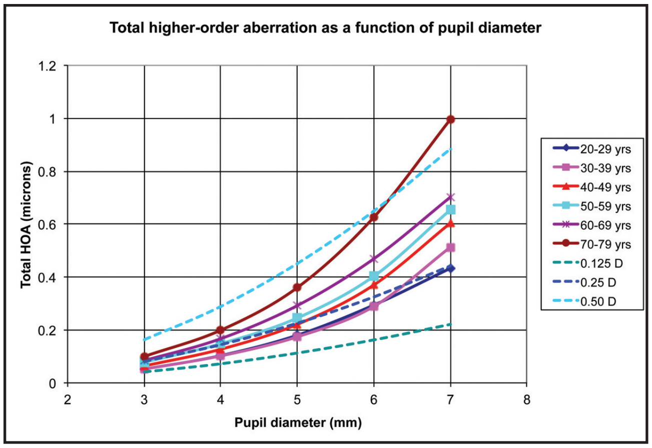

Figure 9-7. Plots of mean total root-mean-square higher-order wavefront aberration as a function of pupil diameter for different age groups. For comparison, the dashed curves show the wavefront aberration associated with defocus levels of 0.125, 0.25, and 0.50 D. (Adapted from Applegate RA, Donnelly WJ, Marsack JD, Koenig DE. Three-dimensional relationship between higher-order root-mean-square wavefront error, pupil diameter, and aging. *J Opt Soc Am A.* 2007;24(3):578-587.)

rapidly after the age of approximately 45 years.[36,37] This straylight reduces the contrast of any retinal image, hence contrast sensitivity, and causes disability glare problems with strong light sources, such as the sun at low elevations or car headlamps at night.

Total Ocular Transmittance

Total ocular transmittance is largely dependent on the changes in lenticular transmittance. Useful summaries and models of the change have recently been presented by van de Kraats and van Norren.[38] The reduced ocular transmittance reduces the retinal illuminance, thus adversely affecting mesopic and scotopic vision. Further, the relatively greater losses at the blue end of the spectrum adversely affect spectral luminous efficiency and color vision (see Figure 9-6B).[32] However, the reduction in shorter wavelength light caused by the older lens, in combination with the absorbing effects of macular pigment, may reduce the effects of blur due to longitudinal chromatic aberration and offer some protection to the retina against the effects of shorter-wavelength light. Removal of the crystalline lens followed by its replacement with a clear poly(methyl methacrylate) IOL removes these beneficial effects, and the use of IOL materials that block both the near ultraviolet and the blue end of the spectrum is now common. Although debate continues about the most desirable form for the spectral transmittance of IOLs,[39] it does not appear that any

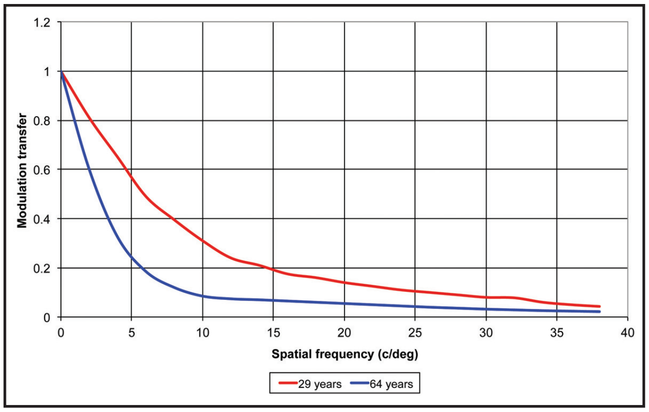

Figure 9-8. Mean ocular modulation transfer functions for young (mean age: 29 years) and older (mean age: 63 years) patients, as measured by the double-pass method with a 4-mm pupil diameter and 633-nm monochromatic light. (Adapted from Artal P, Ferro M, Miranda I, Navarro R. Effects of aging in retinal image quality. *J Opt Soc Am A.* 1993;10(7):1656-1662.)

of today's lenses have significant adverse effects on scotopic vision.[40]

Decreasing lenticular transmittance in blue light may be of importance in relation to problems with circadian rhythms in the elderly. Some retinal ganglion cells contain a rhodopsin-based visual pigment having peak sensitivity around 460 nm. It appears that signals from these cells may be responsible for light-induced melatonin suppression and the maintenance of the circadian rhythm. Senescent miosis combined with reduced blue transmittance may result in an inadequate photon flux in the relevant waveband reaching the retina, causing a disruption of normal circadian rhythms and sleep patterns.[41]

Overall Retinal Image Quality

Overall retinal image quality in monochromatic light is affected by both wavefront aberration and scattered light. Because both increase with age, it would be expected that image quality at a constant pupil diameter would be reduced in the older eye.

Image quality can be directly measured using double-pass techniques. These demonstrate the marked reduction in monochromatic modulation transfer at all spatial frequencies that occur in the normal, older eye in comparison with a younger eye of the same pupil diameter (Figure 9-8).[42]

However, as discussed previously, the reduction in pupil diameter at constant scene luminance with age goes some way toward reducing the wave aberration in the older eye, albeit at the expense of reduced

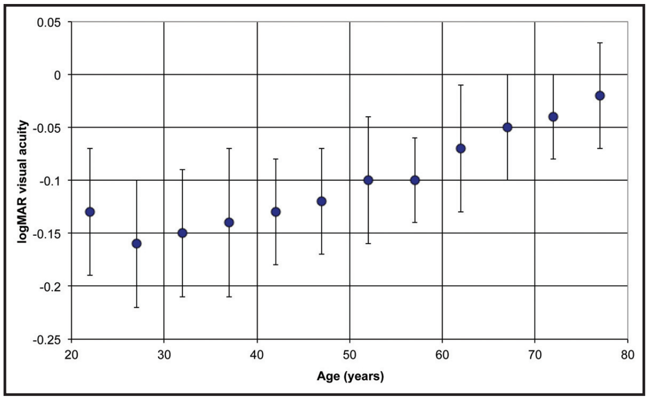

Figure 9-9. Mean and standard deviation of the high-contrast logMAR visual acuity of normal eyes. Each point refers to the mean of eyes within a 5-year interval. Chart luminance is 160 cd/m^{-2}. (Adapted from Elliott DB, Yang KCH, Whitaker D. Visual acuity changes throughout adulthood in normal, healthy eyes: seeing beyond 6/6. *Optom Vis Sci.* 1994;72(3):186-191.)

retinal illuminance, which is further reduced by the decreased ocular transmittance.

Overall Visual Performance

As demonstrated, a variety of optical changes occur in the normal, aging eye. Overall visual performance depends on both optical and neural factors. It is, however, worth noting that, apart from the loss of the ability to accommodate and despite the optical changes, many aspects of visual performance remain remarkably robust against aging, provided that an appropriate spherocylindrical optical correction is used. For example, Figure 9-9 shows changes in photopic, high-contrast visual acuity (VA) with age in healthy eyes.[43] Although there is a modest decline in acuity in later years, mean acuity remains better than 0 logMAR (20/20) almost throughout life. Other studies have found a more rapid decline after the age of 50 years,[44,45] perhaps due to the inclusion of some eyes with pathology.

Elliott et al[43] suggested that the data shown in Figure 9-9 are best fitted by a bilinear model, where:

$$\text{logMAR VA} = -0.0049.\text{age} - 0.025 \text{ (up to 29 years)}$$

$$\text{logMAR VA} = +0.0029.\text{age} - 0.250 \text{ (after 29 years)}$$

Visual acuity under low contrast or glare conditions shows a steeper decline with age. Further aspects of age-related changes in visual performance, arising from both optical and neural factors, are discussed in Chapter 10.

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