

Part Three

Behavioral Processes and Aging

Seven

Vision and Aging

Frank Schieber

I. Introduction

Our ability to cope effectively with our environment begins with our capacity to process sensory input. Indeed, our senses have been carefully crafted by the forces of nature to effortlessly extract critical information from the world around us. Unfortunately, advancing adult aging brings with it systematic reductions in the efficiency of our sensory systems. This lost efficiency of low-level, automatic processing capacity often necessitates effortful, high-level compensatory processes that may tax already limited cognitive resources. Hence, sensory aging is of potentially great interest at all levels of psychological analysis (Schieber, 2003). The purpose of this chapter is to provide a contemporary overview of one aspect of sensory aging, namely age-related changes in vision and low-level visual information processing. This focus on vision does not discount the critical role played by the other major sensory systems, but instead is constrained by the author's breadth of expertise. This overview is by no means exhaustive. In fact, it merely samples

important domains of investigative inquiry with a focus on basic aspects of vision and aging that have received significant attention in the research literature during the past decade. Several excellent reviews of the sensory aging literature prior to the contemporary period are available for readers in need of a more comprehensive and historical introduction to the topic (recommended readings include Corso, 1981; Kline & Scialfa, 1997; Fozard & Gordon-Salant, 2001).

II. Structural Changes in the Visual System

Any comprehensive review of vision and aging must begin with a consideration of the myriad age-related changes that occur in the eye, retina, and ascending visual pathways in the nervous system. Careful attention to this topic is also important because of continuing, but unresolved, efforts to attribute various aspects of visual aging to optical versus neurological mechanisms.

A. Optical Changes

1. Cornea

Light first enters the eye through the cornea (see Figure 7.1). The cornea is the major refractive element of the eye, accounting for approximately two-thirds of the power required to focus incoming light onto the retina (Geldard, 1972). Small changes in the curvature of the cornea result in remarkable changes in the quality of the retinal image. The curvature of the cornea tends to increase beyond 50 years of age. Most of this change is restricted to the horizontal meridian (Baldwin & Mills, 1981; Fledelius, 1988). Hence, corneal astigmatisms that emerge in later life tend to occur along the horizontal meridian—just the opposite of the pattern observed in younger adults with refractive error (Morgan, 1993). Changes in the internal microstructure of the cornea (rather than its anterior surface) result in age-related increases in the intraocular scattering of light in persons over 60 years of age. However, the magnitude of this effect

is small compared to the amount of increased scatter introduced by the senescent lens (Guirao, Redondo, & Artal, 2000; Artal et al., 2002).

2. Iris and Pupil

The amount of light that enters the eye is regulated by the pupillary aperture in the pigmented iris muscle. Through dilation and constriction the pupil of the typical young adult is capable of regulating retinal illumination over a 16:1 range (Geldard, 1972). However, as one grows older the average diameter of the pupil for a given value of illumination tends to become smaller—a condition referred to as *pupillary miosis*. Age differences in pupil diameter are greatest under low-illumination conditions (Winn et al., 1994). For example, in dim light the expected diameter of the pupil falls from an average of 7–8 mm at age 20 to approximately 4 mm by 80 years of age (Loewenfeld, 1979). Effective pupil size may be reduced further by *ptosis*—a common condition among older persons

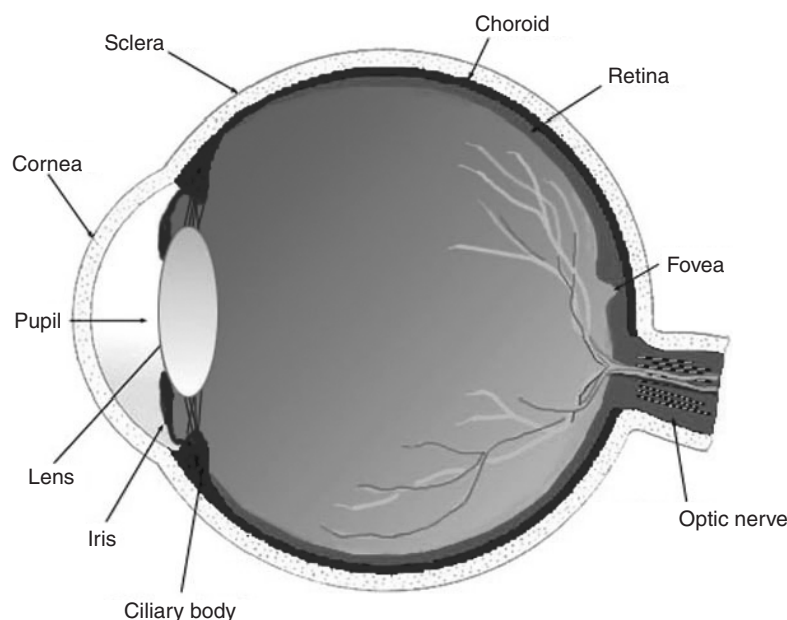


Figure 7.1 The human eye. Source: Webvision.

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in which lost elasticity of the eye lid causes it to “droop” into the pupillary aperture (Theodore, 1975).

3. Lens

Light passing through the pupil next encounters the crystalline lens of the eye. In response to contractile forces exerted by the ciliary muscles, the lens can alter its shape, thereby changing its focusing power. This process, called accommodation, enables the lens to dynamically increase its focusing power as required for doing work at near viewing conditions. In young children, the maximum amplitude of accommodation enables the lens to add approximately 20 diopters of focusing power to the eye, thus enabling them to focus upon objects as close as 5 cm away. However, the maximum amplitude of accommodation decreases in a linear fashion from childhood onward. By the midforties, the average person has lost so much accommodative power that they can no longer adequately focus upon objects within arm’s length (Hofstetter, 1965)—a normal age-related insufficiency of accommodation known as *presbyopia*. By the sixth decade of life, the amplitude of accommodation has been reduced to zero (Donders, 1864) and the need for reading glasses and/or bifocal lenses to perform near work becomes the norm. Presbyopia probably results from lost elasticity of the lens due to its continued growth across the life span rather than due to insufficiency in the ciliary muscles (Atchison, 1995).

The lens also becomes less transparent with increasing adult age (Weale, 1963). This increase in the optical density of the lens appears to be especially pronounced for short wavelength (i.e., blue) light (Said & Weale, 1959; Coren & Girgus, 1972). In addition to having increased optical density, the senescent lens also significantly increases the amount of off-axis

light scatter within the eye (Mellerio, 1971; Whitaker, Steen, & Elliott, 1993; Guirao et al., 1999). Another optical aberration occurring within the aging lens is the accumulation of metabolic by-products that fluoresce when stimulated by 345- (ultraviolet) and 420 (visible violet) -nm light energy (Satchi, 1973).

4. Retinal Illumination and Contrast

The combined effects of diminished pupil diameter and increased lens opacity yield a 0.5 log unit (threefold) reduction in retinal illuminance between 20 and 60 years of age (Weale, 1963). It has been estimated that lens opacity accounts for a 0.2 log unit reduction in retinal illuminance, whereas pupillary miosis accounts for the remainder of the age-related loss in light reaching the retina (Elliott, Whitaker, & MacVeigh, 1990). The contrast of the retinal image also declines with advancing age due to increased intraocular scatter (van den Berg, 1995), as well as a generalized reduction in the modulation transfer function of the eye (Artal et al., 2003). Optical analysis suggests that much of this age-related reduction in the contrast of the image formed on the retina would be mitigated by reducing the resting size of the pupil (e.g., Guirao et al., 1999). Indeed, a study by Sloane, Owsley, and Alvarez (1988) suggests that the pupillary miosis commonly observed in older adults may represent trade-off of the visual system between the demands of retinal illuminance and retinal contrast. Pupillary miosis does not represent an atrophic change in the iris, but rather a dynamic process acting to optimize retinal contrast and overall visual performance.

B. Sensorineural Changes

The most well-studied neural substrate of vision and aging is also the most accessible, namely the retina. The retina lines

the posterior hemisphere of the eye. It consists of two major topographical regions: the macula (the central 18°-wide region specialized for fine spatial resolution) and the peripheral retina. The macula is cone rich whereas the peripheral retina is dominated by rods. Cones are specialized for color vision and fine spatial resolution whereas rods are specialized for low-light levels. The photoreceptors (approximately 120 million rods and 5–8 million cones) are embedded in the retinal pigment epithelium (RPE). The RPE sits on the basement membrane of the retina (Bruch's membrane) and serves as a metabolic "conduit" between the photoreceptor outer segments and the choriocapillary layer on the other side of Bruch's membrane (and the eye-blood barrier). Nerve impulse generated by the rods and cones cascade through a network of bipolar, horizontal, and amacrine cells in the neural layer of the retina and ultimately converge upon the retinal ganglion cells whose axons project directly to the lateral geniculate nucleus (LGN) of the thalamus. From the LGN, visual information ascends to the primary visual cortex located in the occipital lobe of the brain (Bonnel, Mohand-Said, & Sahel, 2003).

1. Retinal Photoreceptors

There is converging evidence that adult aging is accompanied by a dramatic loss in the number of rods (Curcio et al., 1990; Gao & Hollyfield, 1992; Panda-Jonas, Jonas, & Jacobczyk-Zmija, 1995). Curcio and colleagues (1993) reported that rod density in the central retina (3–10°) declined by 30% between 34 and 90 years of age. Despite previous reports that aging was associated with a reduction in cone density as well (Gartner & Henkind, 1981), Curcio et al. (1993) found that cone density remained relatively stable across the same age range. One of the reasons for this inconsistency may be

due to the fact that there are wide individual differences in cone numbers. Given large individual differences in cone count, small but systematic declines with age would be difficult to find given the small number of observations typical of *in vitro* studies of this kind. However, other evidence indirectly supports concomitant age-related losses in cones. For example, foveal cone pigment density appears to decline with age (Kilbride et al., 1986; Eisner et al., 1988). However, electrophysiological evidence has failed to provide consistent evidence regarding macular cone function (Jackson et al., 2002; Seiple et al., 2003). Given that the cones appear to depend on a "survival factor" produced by surrounding rods (Fintz et al., 2003), it would be surprising if the age-related loss of rods did not portend subsequent cone loss at some to-be-determined time lag (see Curcio, Owsley, & Jackson, 2000).

2. Retinal Ganglion Cells

The activity of the photoreceptors is preprocessed in the neural network of retina consisting of interconnected bipolar, amacrine, and horizontal cells. Ultimately, this information converges upon retinal ganglion cells whose axons converge to form the optic nerve and ascend to the brain. Several studies have reported significant age-related reductions in the number of retinal ganglion cells subserving the macular region of the retina (Gao & Hollyfield, 1992; Curcio & Drucker, 1993). Consistent with these findings are reports of age-related losses in the number of axons in the optic nerve (e.g., Repka & Quigley, 1989) and age-related thinning of the neural layer of the retina (Lovasik et al., 2003).

3. Retinal Support Layers

Lipofuscin accumulates in cells of the retinal pigment epithelium (RPE) with

advancing age. Lipofuscin is a well-known biomarker of aging and reacts with light to form reactive oxygen species (free radicals) that can damage cell membranes (Bonnell et al., 2003). Abnormal deposits of insoluble material called drusen accumulate between the RPE and Bruch's membrane as people grow older. These drusen may induce local inflammation of retinal tissue and ultimately trigger an autoimmune immune response and subsequent retinal disease (Anderson et al., 2002). Bruch's membrane also accumulates significant deposits of cholesterol with advancing age, perhaps diminishing the efficient exchange of nutrients and metabolic by-products between the RPE and the choriocapillary layer (Curcio et al., 2001).

4. Primary Visual Cortex

Initial studies of the human visual cortex reported significant cell loss with advancing age. For example, Devaney and Johnson (1980) reported that the number of cells in the primary visual (striate) cortex declined by 25% as early as age 60. However, subsequent investigations have failed to observe systematic declines of neuron density in the visual cortex with aging (e.g., Haug et al., 1984; Leuba & Garey, 1987). In a comprehensive review of the literature, Spear (1993) concluded there was no consistent histological evidence in the primate to support the hypothesis of senescent cell loss in the ascending visual pathways between the retina and the visual cortex. Rather than general cell loss per se, Kim and co-workers (1997) suggested that age-related neural changes impacting visual function are more likely to involve extrastriate cortical areas or "reside on a different level of anatomical analysis, such as the level of synapses or receptors" (p. 126). Indeed, electron microscopy studies have revealed evidence of degenerative changes in dendritic processes and

decreased synaptic density for neurons in the primary visual cortex of "older" rhesus monkeys (Peters, Moss, & Sethares, 2001). Along a similar vein, Leventhal and colleagues (2003) reported age-related degradation of intracortical inhibitory processes related to GABA-receptor insufficiency in the visual cortex of nonhuman primates. Also consistent with the road map of Kim et al. (1997), Park and colleagues (2004) reported that despite histological evidence that neural density in the ventral (i.e., extrastriate) visual cortex is spared with aging, human **fMRI** studies reveal reduced functional efficiency in this region.

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III. Visual Impairment and Age-related Pathology

Although the emphasis of this volume focuses on normative changes accompanying adult aging, visual pathology is so common and so potentially debilitating in terms of everyday behavioral function that a brief review of the major age-related visual disorders appears both appropriate and necessary. The Eye Diseases Prevalence Research Group (EDPRG) has estimated that as of the year 2000 approximately 937,000 Americans older than 40 years of age were legally blind (corrected acuity 20/200 or worse in the better eye). Another 2.4 million Americans adults had low vision (corrected acuity worse than 20/40 but better than 20/200). This means that 1 of 28 adults living in the United States could be described as visually impaired (EDPRG, 2004a). A very large proportion of this age-related increase in the prevalence of visual impairment can be accounted for by three classes of ocular pathology: cataract, age-related maculopathy (ARM), and glaucoma (see Table 7.1). Cataract involves an excessive opacification of the lens and is, by far,

Table 7.1
 Prevalence (per 100) of Visual Pathology as
 a Function of Age (Years)^a

Age	Cataract	ARM-Dry	ARM-Wet	Glaucoma
50–54	5.1	0.34	0.23	0.91
60–64	15.5	0.56	0.38	1.57
70–74	36.9	1.66	1.15	2.79
80+	68.3	11.77	8.18	7.74

^aData from Eye Diseases Prevalence Research Group (2004 b,c,d).

the leading cause of age-related visual impairment. Fortunately, the deleterious effects of cataract are almost always reversible via outpatient surgical procedures (EDPRG, 2004b). The next most prevalent source of visual impairment among older persons is ARM, which involves a progressive and, as yet, incurable degeneration of central retinal structures. The risk of glaucoma also increases with age and involves the gradual and progressive destruction of the head of the optic nerve due to excessive pressure within the eye. Glaucoma responds reasonable well to drug therapy and much loss of function can be prevented if the disorder is detected early enough.

IV. Age-related Changes in Visual Function

A. Smooth Pursuit and Saccadic Eye Movements

The ability of the visual system to resolve color and fine spatial texture is mediated by the fovea. However, the centrally located fovea represents a very small region of visual space (approximately 2°). Optimal performance on many tasks depends on the ability of the oculomotor system to acquire, track, and maintain stimulus images on the foveal region of the retina. This acquisition and maintenance of the visual stimulus are mediated by two separate but complementary

perceptual-motor systems: the *smooth pursuit* and *saccadic* eye movement systems. The smooth pursuit system regulates large-amplitude, continuous motion processes that serve to track moving targets accurately and thus enhance visual performance by extending the functional range of foveal vision across a broader region of visual space. The saccadic eye movement system generates brief, high-velocity, ballistic excursions of the eye, which serve structured visual search (e.g., reading).

Smooth pursuit performance is typically quantified in terms of *pursuit gain* (the ratio of eye velocity divided by target velocity). Ideal smooth pursuit eye tracking performance is signified by unity gain. At increasing target velocities, eye tracking performance begins to lag behind target position/speed and pursuit gain declines proportionately. Previous studies have reported little or no age-related reduction in pursuit gain for target velocities below 5–10°/s but greater rates of decline in pursuit gain as target velocities increased beyond this level (e.g., Sharpe & Sylvester, 1978; Spooner, Sakala, & Baloh, 1980; Kanayama et al., 1994). Of special interest, Kaufman and Abel (1986) demonstrated that age-related declines in smooth pursuit eye movement performance were exacerbated in the presence of competing or distracting objects in the stimulus background. More recently, Moschner and Baloh (1994) measured pursuit gain in a large sample

of healthy young ($n=23$, mean age = 25) and older ($n=57$, mean age = 79) adults. The pursuit target oscillated sinusoidally along a $\pm 18^\circ$ horizontal meridian at velocities ranging from 11 to $45^\circ/\text{s}$ while eye tracking performance was monitored using standard electrooculographic techniques. Age-related declines in smooth pursuit performance were observed at all three target velocities, and the magnitude of this deficit increased significantly with target velocity. Pursuit gain fell to 0.50 in older observers at the highest target velocity, whereas the performance of the young observers at this speed (gain = 0.87) was better than that achieved by their older counterparts at the slowest target velocity.

Saccadic eye movements show much less dramatic change with advancing age. Small but significant age-related increases in the latency of saccade onsets have been reported by numerous investigators (e.g., Abel, Troost, & Dell'Osso, 1983; Warabi, Kase, & Kato, 1984; Huaman & Sharpe, 1994; Moschner & Baloh, 1994; Abrams, Pratt, & Chasteen, 1998). The magnitude of this age-related increase in the latency of saccadic eye movements varies from approximately 20 ms (Moschner & Baloh, 1994; Abrams et al., 1998) to 100 ms (Abel et al., 1983), depending on task and stimulus conditions. The peak velocities of saccadic eye movements (which are very fast) have also been shown to slow with age. For example, Pitt and Rawles (1988) reported that saccade velocity decreased by approximately 0.25 % per year from 20 to 68 years of age. Similar findings have been reported by Spooner, Sakala, and Baloh (1980). Despite reports of slowing, there is reasonable agreement that the spatial accuracy of saccadic target acquisition is well maintained into very old age (Warabi et al., 1984; Hotson & Steinke, 1988; Rosenhall et al., 1987; Moschner & Baloh, 1994), but see Sharpe and Zackon (1987). Related

studies have also found that stable fixation accuracy can be maintained by older subjects for task durations of at least 10 s (Kosnik et al., 1987).

Another characteristic of oculomotor function that appears to be affected by aging is the range or extent of upward gaze (i.e., the maximum vertical extent of visual fixation that can be achieved without the benefit of head movement). Chamberlain (1971) reported that the maximum extent of upward gaze declines from 40° at 5–14 years of age down to approximately 15° by 84 years of age. More recently, Huaman and Sharpe (1993) used a high-resolution magnetic search coil to reassess the maximum extent of upward and downward gaze in young (mean = 28.3 years), middle-aged (mean = 49.8), and older (mean = 71.9) adults. The limits of upward gaze reported were 43.1° , 42.0° , and 32.9° for the young, middle-aged, and older age groups, respectively. Similar findings were reported for downward gaze. An explanation of the differences across studies remains unclear. It should be noted, however, that a more recent study by Clark and Isenberg (2001) yielded results consistent with data of Huaman and Sharpe (1993).

B. Light Sensitivity

The human retina consists of two complementary subsystems that enable it to efficiently process light signals over a remarkable range of stimulus intensities. The *scotopic* system receives input from the rods and performs best under low light levels (10^{-6} to 10^1 cd/m²). The *photopic* system receives input from cones. Unlike the scotopic system, the photopic system is characterized by keen spatial resolving power and fine color discrimination. However, these enhanced capabilities come at a cost. The photopic system requires much more light (10^0 to 10^7 cd/m²) in order to respond effectively

(Geldard, 1972). When the eye is fully adapted to low light levels (e.g., a darkened hallway or a rural roadway at night), our ability to detect and recognize gross features in the visual environment is ultimately determined by the overall efficiency of the scotopic system. The ability to discriminate fine detail and/or color is dependent on the operating efficiency of the photopic system.

Older adults consistently report problems performing important visual tasks under dim lighting conditions and at night (Kline et al., 1992; Mangione et al., 1998; Owsley et al., 1999). Low illumination levels are related to age-related vehicular crashes and injuries related to falls (Massie, Campbell, & Williams, 1990; McMurdo & Gaskell, 1991). Much of this problem may be attributable to age-related declines in the sensitivity of the scotopic system, as well as the rate at which scotopic sensitivity dynamically adjusts to decreases in background illumination (i.e., dark adaptation). Studies in this area have improved our understanding of the special needs of older observers under low-light levels. Perhaps as important, these studies have enabled investigators to make headway in deciphering the relative roles of optical versus neural factors in mediating age-related declines of visual function.

1. Scotopic Sensitivity

In order to demonstrate the smallest amount of light that can be reliably detected by the human visual system, one must establish special conditions that manifest the ultimate sensitivity of the scotopic system: (1) allow the visual system to adapt to complete darkness for 30–40 min to allow for the full regeneration of rod photopigments necessary for the transduction of light and (2) present the target approximately 10° from central fixation in order to stimulate the retina in

the region of maximum rod density. Classical studies meeting these criteria have revealed age-related reductions in scotopic sensitivity ranging from 0.3 to 2.0 log units in magnitude (e.g., McFarland et al., 1960; Gunkel & Gouras, 1963). However, until very recently, investigators were unable to determine whether these changes were attributable only to age-related optical factors or whether neurophysiological mechanisms might also be involved.

Jackson and colleagues (1997) measured scotopic sensitivity in young (mean = 27 years) and old (mean = 70 years) adults while carefully controlling for optical factors that might contribute to age-related reductions in performance. For example, all participants were carefully screened for manifest ocular disease. Pupils were dilated to a minimum of 6 mm to eliminate the effects of pupillary miosis. Individual differences in lenticular density were measured and used to statistically adjust stimulus dosage at the level of the retina. Finally, all participants were refracted to the test distance to control for refractive error. Even after applying these unprecedented levels of controls to eliminate systematic effects due to optical factors, these investigators still observed a 0.5 log unit loss in scotopic sensitivity in their older group. Similar findings were reported in another carefully controlled study by Sturr and co-workers (1997). In a follow-up study, Jackson and Owsley (2000) replicated and extended these findings in an examination of scotopic and photopic sensitivity in a sample of 94 observers ranging in age from their twenties through eighties (photopic sensitivity data are discussed later). After controlling for all known optical sources of variation in data, they found that scotopic sensitivity declined at a rate of 0.08 log units per decade between 20 and 90 years of age—replicating the same 0.5 log unit (three-fold) reduction across the observed life

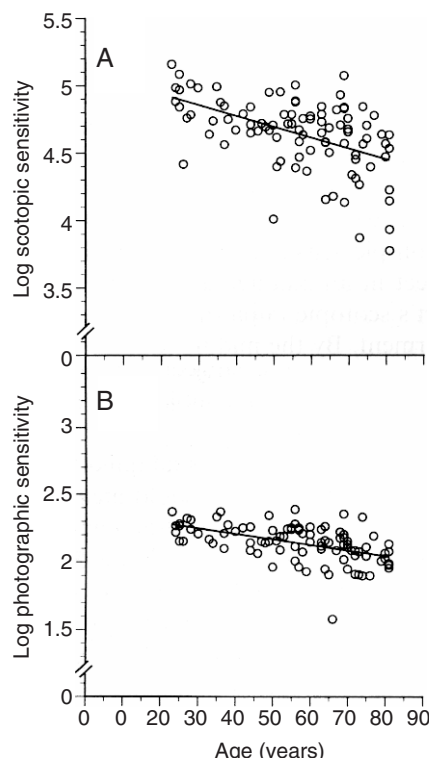


Figure 7.2 Scotopic (A) and photopic (B) sensitivity as a function of age. Source: Jackson and Owsley (2000).

span (see Figure 7.2). The authors interpreted these results as strong evidence for the involvement of neural mechanisms in the mediation of age-related losses in visual sensitivity under low luminance conditions. Although they could not rule out involvement of postreceptoral processes involving retinal ganglion cells, optic nerve, or cortical dysfunction given previously noted age-related changes at these levels, these investigators make a strong case for their “retinoid deficiency hypothesis” (see Jackson, Owsley, & Curcio, 2002). This hypothesis suggests that rods are more likely to suffer from the deleterious effects of age-related changes in retinal support structures responsible for satisfying the high metabolic demands of photoreceptor transduction.

2. Photopic Sensitivity

The limits of photopic light sensitivity are typically measured using stimuli delivered to the fovea—the rod-free central region of the retina that sets the limits on form and color+ vision. Jackson et al. (1997), as described earlier, conducted a carefully controlled assessment of age differences in photopic sensitivity by determining the minimum amount of retinal stimulation needed to detect a brief 1.7° light flash presented against a uniform 10 cd/m² background. Their results are depicted in of Figure 7.2B. Photopic sensitivity declined with age at a rate of 0.04 log units per decade. Thus, age-related declines in photopic sensitivity progress at half the rate observed for scotopic sensitivity. Even greater reductions in the rate of age-related losses of photopic sensitivity have been reported by Coile and Baker (1992). These finding are consistent with the hypothesis put forward by Jackson et al., (2002) that senescent processes differentially affect rod relative to cone function.

C. Spatial Resolution

1. Visual Acuity

Perhaps the best-known index of general visual function, visual acuity is a measure of one’s ability to resolve fine spatial detail. Visual acuity is traditionally expressed in terms of the *minimum angle of resolution* (MAR) in minutes of arc. Normal visual acuity for the general population is 1 minarc, namely the smallest spatial contour that can be resolved subtends 1 min of arc (so-called 20/20 vision). It should be noted that many persons demonstrate spatial resolutions better than 1 minarc. In order to achieve optimal reliability, state-of-the-art visual acuity assessment instruments use charts with the same number of

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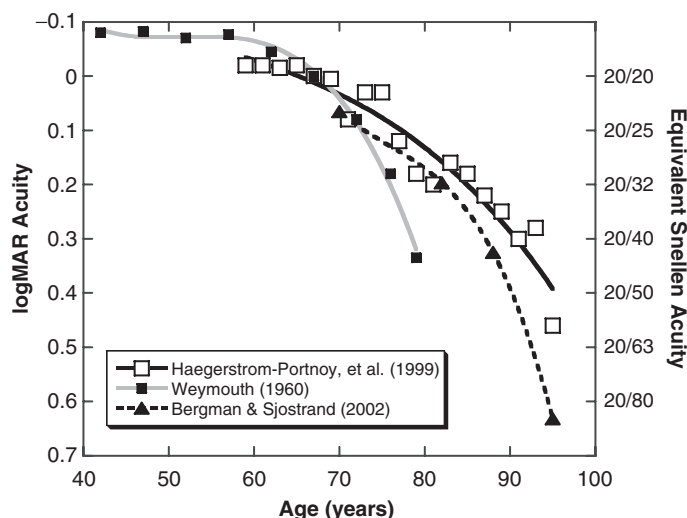


Figure 7.3 Findings from three studies of aging and visual acuity.

letters per line and with letter size and spacing decreasing in 0.1 log unit steps (see Rosser, Laidlaw, & Murdoch, 2001).

Pitts (1982) provided a comprehensive review of the classical literature on age-related changes in visual acuity. Findings across studies revealed a consistent pattern: Corrected visual acuity remained excellent (i.e., 1 minarc) into the sixth decade of life. Beginning in the sixth decade, corrected visual acuity began to decline at an accelerated pace. At this point, however, the consensus among the studies began to break down as the rate of senescent decline varied widely across investigations. Figure 7.3 compares the classical findings of Weymouth (1960) to recent results reported from Europe and North America. The two contemporary studies appear to be consistent and depict an aging visual system that is somewhat more robust than the classical findings (also see Elliott, Yang, & Whitaker, 1995). Both of the contemporary studies used large, representative community-resident samples and modern letter-by-letter scoring criteria. Classical studies, like Weymouth's, typically employed data collected in a clinical setting and used less precise line-by-line scoring

systems—factors that may have contributed to an overly pessimistic estimate of acuity declines expected with advancing adult age. Averaged across studies, Haegerstrom-Portnoy et al. (1999) and Bergman and Sjostrand (2002) data indicate that representative corrected visual acuity declines from 1.2 minarc (20/24) at age 70 to 3.5 minarc (20/71) at 95 years of age (see Table 7.2).

The estimates presented in Table 7.2 reveal that relatively unimpaired levels of visual acuity can be maintained “for most persons” through the use of eyeglasses and/or contact lenses until 88 years of age. However, “average” values provide little information by which to assess the range of functional impact resulting from age-related declines in visual acuity. Fortunately, Bergman and Sjostrand (2002) provided a detailed breakdown of the distribution of acuity across the range of ages sampled in their study. A summary of their findings is presented in Figure 7.4, where the relative proportion of persons falling into three functional categories of vision are reported across age groups. The three categories of visual acuity, namely 20/25 or better, 20/26-20/66 20/66, or worse,

Table 7.2
 Representative Corrected Acuity as a Function of Age from Two
 Contemporary Studies

Age	Hagerstrom-Portnoy et al. (1999)		Bergman & Sjostrand (2002)		Pooled estimate	
	MAR ^a	Snellen	MAR ^a	Snellen	MAR ^a	Snellen
70	1.2	20/24	1.16	20/23	1.18	20/24
82	1.5	20/30	1.57	20/31	1.54	20/31
88	1.7	20/35	2.08	20/42	1.91	20/38
95	2.9	20/58	4.26	20/85	3.57	20/71

^aMinimum angle of resolution (minute of arc).

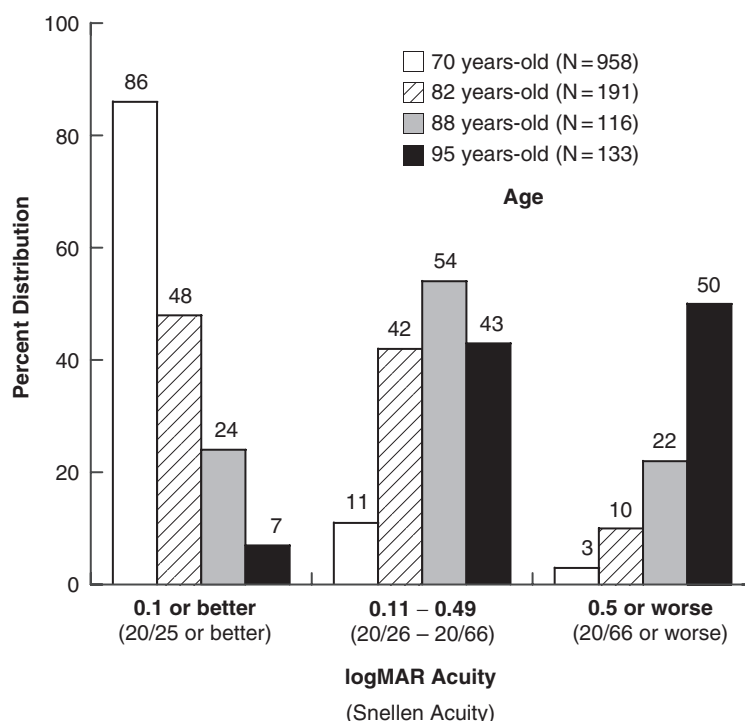


Figure 7.4 Functional levels of visual acuity distributed by age. Data from Bergman and Sjöstrand (2002).

represent levels of general visual function that can be described as “good,” “marginal,” and “impaired,” respectively. Those falling into the “marginal” group would encounter problems reading small text or road signs from a great distance. Many with “marginal” acuity would be challenged when applying for

or renewing their driver’s license, as the minimum “uncorrected” visual acuity criterion for most state departments of transportation is 20/40 (2 minarc). However, this criterion has gradually been relaxed over the past decade and few drivers with acuities worse than 20/66 will ultimately be denied a drivers

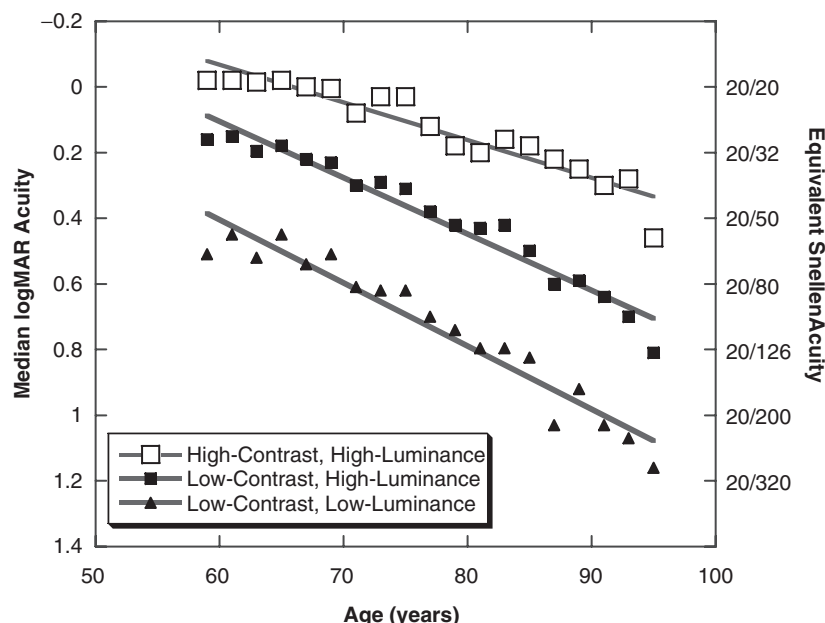


Figure 7.5 Visual acuity as a function of age and varying stimulus contrast and luminance. Source: Haegerstrom-Portnoy, Schneck, and Brabyn (1999).

license (Rosenbloom, 2004). Persons with visual acuities in the “impaired” region would likely have difficulty obtaining a driver’s license and could also be expected to be challenged with many activities of daily living.

Several studies have reported that age differences in visual acuity are exacerbated under challenging viewing conditions such as low stimulus contrast and/or low luminance (e.g., Richards, 1977; Sturr, Kline, & Taub, 1990). Haegerstrom-Portnoy, Schneck, and Brabyn (1999) collected acuity data from a large ($N=900$) sample of healthy older adults while varying background luminance (150 versus 15 cd/m^2) and/or letter contrast (90% versus 16% nominal contrast). Results of this study are summarized in Figure 7.5. Upon inspection of Figure 7.5, it is obvious that acuity declines at low target contrast and falls even farther when luminance is reduced as well. However, what is less apparent is that the slopes of these three acuity-

by-age functions significantly differ from one another. Acuity is lost at a rate of 5.5 letters (0.11 logMAR) per decade for the traditional high-contrast, high-luminance stimulus condition. However, this rate of loss increases to 8 letters (0.16 logMAR) per decade in the low-contrast, high-luminance condition. An even greater rate of loss (9 letters or 0.18 logMAR per decade) is observed when both contrast and luminance are reduced.

In discussing their findings, the authors pointed out an inconsistency in the research literature, namely previous studies indicating that age differences in acuity were exacerbated under low-contrast conditions used *binocular* viewing conditions (i.e., Richards, 1977; Taub & Sturr, 1991; Haegerstrom-Portnoy et al., 1999). However, previous studies using *monocular* viewing conditions failed to observe accelerated age-related loss upon switching from high- to low-contrast stimuli (i.e., Brown & Lovie-Kitchin, 1989; Owsley et al., 1990).

Haegerstrom-Portnoy et al. (1999) concluded that this pattern of results is strongly consistent with the hypothesis that the efficiency of the binocular summation process is compromised with advancing age. This conclusion is also supported by studies of age differences in contrast sensitivity reported previously by Owsley and Sloane (1990) and Pardhan (1996) (see later). Regardless of the theoretical significance, the data presented in Figure 7.5 teach a practical lesson: standard tests of visual acuity under ideal conditions may not be the best indicators of functional status under the more challenging conditions typically encountered in real world settings (Schieber, 1988).

2. Contrast sensitivity

The ability to detect and recognize objects in the visual environment varies considerably as a function of target size, contrast, and spatial orientation (see Olzak & Thomas, 1985). As a consequence, knowledge about one's visual acuity (the ability to resolve small, high-contrast targets) is not always predictive of real world visual performance involving objects of various size and low-to-moderate contrast (e.g., Ginsburg et al., 1982; Watson, Barlow, & Robson, 1983). The *contrast sensitivity function* (CSF) complements and extends the information provided by simple measures of acuity by assessing an individual's visual efficiency for the detection of targets over an extended range of size and/or orientation. The CSF is determined by measuring the minimum contrast needed to detect idealized spatial targets (sine wave gratings) that vary in their spatial periodicity [cycles per degree of visual angle or (c/deg)]. Contrast thresholds are typically collected using sine wave grating stimuli that vary in spatial frequency from 0.5 c/deg (very wide) to 16–32 c/deg (very narrow). Because high

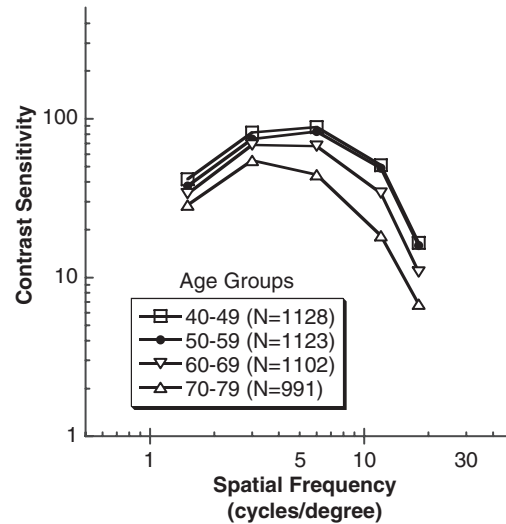


Figure 7.6 Contrast sensitivity functions of four age groups. Source: Nomura et al. (2003).

levels of visual sensitivity are associated with low-contrast thresholds, a reciprocal contrast sensitivity score (1/threshold contrast) is computed and plotted as a function of target spatial frequency, yielding the now familiar inverted U-shaped CSF (e.g., see Figure 7.6).

Numerous studies reveal a consistent pattern of age-related change in the CSF collected under photopic conditions (foveal presentation at moderate to high luminance levels). Contrast sensitivity declines by approximately 0.3 log units across the later half of the adult life span. Large magnitude losses such as these are typically reported for targets of intermediate and high spatial frequency (4–18 c/deg) (Owsley, Sekuler, & Siemsen, 1983; Elliott, 1987; Schieber et al., 1992; Elliott & Whitaker, 1992). However, several studies also report smaller age-related losses at low (less than 2 c/deg) spatial frequencies (Nameda, Kawara, & Ohzu, 1989; Ross, Clarke, & Bron, 1985; Sloane, Owsley, & Alvarez, 1988). Results from a large-scale study of contrast sensitivity reflect this consensus view. Nomura and co-workers (2003) used a clinical contrast sensitivity

assessment chart (Vistech VCTS 6500) to assess normative differences in the CSF in a sample of 4344 adults ranging in age from 40 to 79 years of age. Referring to Figure 7.6, observers in their sixties demonstrated a 0.1 log unit loss in contrast sensitivity at intermediate and high spatial frequencies (6, 12, and 18 c/deg). Those in their seventies, the oldest group sampled, demonstrated an even greater loss (0.3 log unit) across the same range of spatial frequency. [Note: The apparent age-related loss in contrast sensitivity at the lowest spatial frequency probably resulted from the small number of stimulus cycles used in the 1.5-c/deg targets on the Vistech chart; see Savoy and McCann, (1975)].

Data of Nomura et al. (2003) also serve to demonstrate the relatively weak correlation between visual acuity and contrast sensitivity that is often observed, especially among older adults. Figure 7.7 shows the distribution of contrast sensitivities required to detect an 18-c/deg target among persons with "good" (20/20 or better) visual acuity. Even among groups of adults screened to preclude problems with visual acuity, large

variations in contrast sensitivity performance remain. It is noteworthy, for example, that 21% of those in their seventies failed to detect the 18-c/deg target at the highest contrast available (i.e., 33%), despite having acuities of 20/20 or better. This pattern of results is consistent with numerous studies that have shown that contrast sensitivity is a good predictor of age differences in visual performance even when subjects are equated for high-contrast visual acuity. For example, Owsley and Sloane (1987) reported that age-related problems in the detection and recognition of human faces could be accounted for by contrast sensitivity losses at intermediate spatial frequencies in observers with good visual acuity. Similar findings have been reported for performance with other domains of real world visual stimuli (e.g., Evans & Ginsburg, 1985; Kline et al., 1990).

Clear evidence shows that much, if not most, of the age-related loss of photopic contrast sensitivity can be explained by changes in the optical properties of the senescent eye. Numerous studies have demonstrated that systematic age differences in the quality of the retinal image due to pupillary miosis, lens opacification, and increased intraocular scatter contribute significantly to age-related reductions in contrast sensitivity (e.g., Owsley et al., 1983; Hemenger, 1984; Guirao et al., 1999). However, much debate remains regarding the relative contribution of neural factors (e.g., changes in the retina, optic nerve, and/or visual cortex) to age differences in contrast sensitivity. Numerous studies that have attempted to isolate the neural contributions to contrast sensitivity change by controlling for senescent changes in preretinal optics (e.g., Elliott, Whitaker & MacVeigh, 1990; Sloane, Owsley & Alvarez, 1988) or through bypassing the optics of the eye altogether by stimulating the retina via laser interferometry (Dressler & Rassow, 1981;

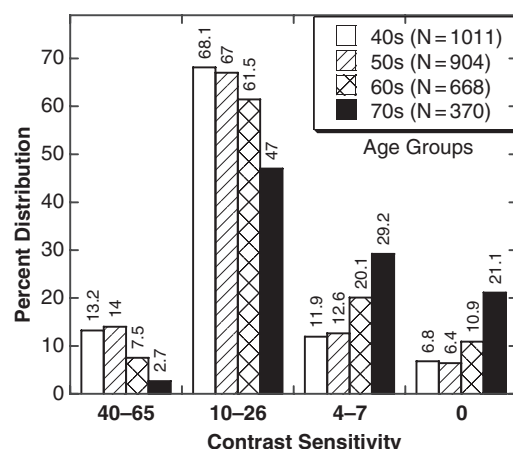


Figure 7.7 Distribution of contrast sensitivity by age for an 18 c/deg target from observers with visual acuity of 20/20 or better. Source: Nomura et al. (2003).

Kayazawa, Yamamoto, & Itoi, 1981; Morrison & McGrath, 1985; Burton, Owsley, & Sloane, 1993) have reported highly inconsistent conclusions. Estimates of the relative contribution of neural mechanisms toward explaining age-related losses in contrast sensitivity range from "zero" (Hemenger, 1984) to "substantial" (Morrison, & McGrath, 1985).

One conclusion that is certain is that the optics versus neural mechanisms debate will continue. It is hoped that, investigators will begin to use more diverse and powerful tools in their attempts to resolve these long-standing questions. In fact, several studies have demonstrated that such developments are already underway. Studies have used structural equation modeling in an attempt to disentangle the relative contributions of optical versus neural mediators of age differences in contrast sensitivity (e.g., Scialfa, Kline, & Wood, 2002). Other investigators have begun using "ideal observer models" of spatial vision together with noise-masking paradigms in an attempt to separate variations due to *internal noise* (which includes senescent optics effects) versus *computational efficiency* (nonoptical factors) (e.g., Pardhan et al., 1996; Bennett, Sekuler, & Ozin, 1999). These approaches hold great potential but will require long-term, programmatic efforts of research to rigorously evaluate the numerous (and yet to be tested) assumptions upon which they are based.

3. Vernier Hyperacuity

Vernier hyperacuity applies to a family of tasks in which the observer is required to detect deviations from perfect linear alignment among two or more suprathreshold visual objects (see Figure 7.8 for sample stimulus configurations). Normal observers are capable of detecting when one of the stimuli deviates from

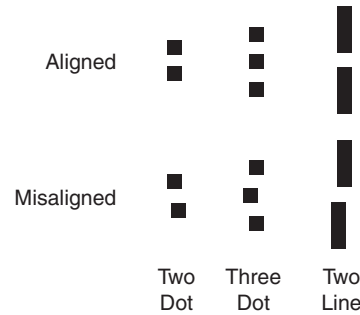


Figure 7.8 Schematic drawing depicting sample vernier acuity alignment stimuli. Stimulus elements are suprathreshold (typical stroke width of 3–5 minarc) separated vertically by a small gap (e.g., 4 minarc). The horizontal alignment of one of the elements is varied. The misalignment is exaggerated for illustration purposes.

true linear alignment by as little as 5 to 10 s of arc. This is a tiny fraction of the smallest spatial gap that can be resolved by a person with 20/20 visual acuity (i.e., 60 s of arc). Hence, this exquisite sensitivity for relative spatial position has been referred to as "hyperacuity" (Westheimer, 1975). Performance on these types of tasks is quantified using two complementary measures: vernier acuity and vernier bias. As classically measured using the method of adjustment, the *vernier acuity* value is an index of the precision of a group of alignment judgments (i.e., the standard deviation). *Vernier bias*, however, refers to the average absolute deviation from true alignment (i.e., a measure of accuracy rather than precision).

Vernier hyperacuity has developed a bit of a mystique among vision researchers interested in aging. It has been claimed that vernier acuity has somehow escaped the deleterious effects of aging, unlike virtually every other measure of spatial vision, and has remained "forever young" (see Enoch et al., 1999). This interpretation is not surprising given the remarkable consensus in the research literature during the 1990s. Numerous studies have consistently reported that vernier acuity

remained unchanged throughout the adult life span (e.g., Whitaker, Elliott, & MacVeigh, 1992; Lakshminarayanan, Aziz, & Enoch, 1992; Lakshminarayanan & Enoch, 1995; Vilar et al., 1995; Kline et al., 2001). All of these studies, with the exception of Odom and colleagues (1989), reported that vernier bias also remained stable with advancing adult age. The remarkable stability of vernier hyperacuity across the life span has often been attributed, in part, to its demonstrated insensitivity to optical degradations that might blur or reduce the intensity and/or contrast of the retinal image (Williams, Enoch, & Essock, 1984; Enoch et al., 1999). However, two studies appear to challenge the conclusion that vernier hyperacuity is resistant to aging.

Li, Edwards, and Brown (2000) hypothesized that one possible reason that previous investigations of vernier hyperacuity had failed to observe an age-related decline was because they failed to vary stimulus offset (or misalignment) in fine enough steps to reveal the true sensitivity of their youngest observers. In effect, they suggested that previous studies had systematically underestimated the sensitivity of young observers, thereby masking any true age difference. Indeed, a comparison of the minimum offset increments used in many of these studies reveals that stimuli were manipulated in a relatively crude manner (see Garcia-Suarez et al., 2004). Li et al. (2000) proceeded to measure vernier hyperacuity in a sample of 60 observers equally spaced across the life span between 21 and 75 years of age. Hyperacuity was assessed using a three-line vertical stimulus array in which the alignment of the central stimulus segment was offset between 0 and 18 s of arc (in increments of 6 arc s). The method of constant stimuli was employed using a two-alternative forced-choice procedure in which the observer's task was to report whether the middle line in the vernier stimulus array was

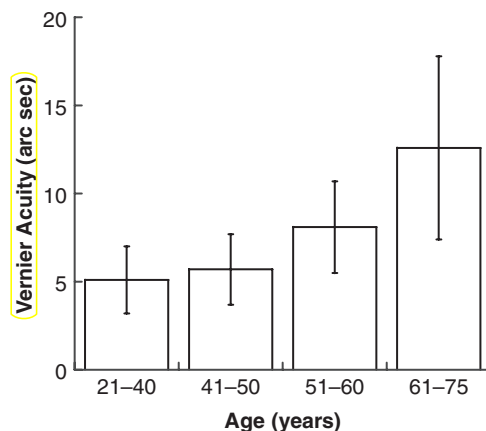


Figure 7.9 Mean vernier acuity and SD bars for four age groups. Source: Li, Edwards, and Brown (2000).

displaced to the left or the right. Probit analysis was used to fit a "percent right" psychophysical function to the data. Vernier acuity was defined as half the distance between the 25 and 75% points on this psychophysical function. The "point of subjective equality" (i.e., the 50% point on the psychophysical function) was used as the index of vernier bias. Contrary to many previous studies, the precision of vernier localization was reduced significantly in older observers (see Figure 7.9). Vernier acuity levels remained unchanged between the ages of 21 and 50 but increased significantly between 51 and 75 years of age. A control experiment in which 8 young observers viewed stimuli through a 3-mm artificial pupil and a neutral density filter that reduced light transmission by approximately 60% indicated that expected age-related reductions in retinal illumination could not account for the observed age differences in performance. Finally, mean absolute misalignment error (vernier bias) did not vary as a function of age. Li et al. (2000) concluded that when one uses carefully designed psychophysical procedures and sufficiently small increments of misalignment in the vernier stimulus configuration that age-related declines in

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vernier acuity will be observed. A more recent study by Garcia-Suarez, Barrett and Pacey (2004) appears to confirm this conclusion. Using a three-point vernier alignment stimulus, the method of constant stimuli, and a very small misalignment increment step size (4.6 s of arc), they assessed vernier acuity in 18 young (mean age = 26.3) and 18 old (mean age = 72.0) highly practiced observers. Like the Li et al. (2000) study, they reported a 0.22 log unit age-related elevation in vernier hyperacuity threshold (7.22 and 11.98 arc s for young and old, respectively). Together, these two studies appear to have dramatically altered the landscape regarding the nature of age-related differences in vernier hyperacuity. Both groups of investigators have attributed these newly discovered age-related declines in spatial hyperacuity to senescent reductions in the efficiency of neural mechanisms. In a follow-up study, Li, Edwards, and Brown (2001) reported electrophysiological evidence consistent with this interpretation.

D. Color Vision

The human visual system is capable of remarkably precise discrimination on the basis of color. Observers with normal vision can distinguish among more than 100,000 hues generated from various combinations of three primary color light sources (Geldard, 1972). Most large-scale investigations of age differences in color discrimination have been based on the *Farnsworth-Munsell 100 Hues Test* (FM-100). The FM-100 is one of a family of color confusion tests in which small (1.4°) color-coated stimuli are arranged by the observer to form a linear sequence in which the most similar stimuli are placed side by side to form a predetermined color gradient. All test colors are of intermediate lightness and color saturation (Munsell value = 5, chroma = 5). The test is scored by counting the number of

sequential arrangement errors, as well as noting their placement within each of four regions of color space. A total error score of 100 or more is indicative of anomalous performance (Farnsworth, 1943). Cody, Hurd, and Bootman (1990) demonstrated that older adults with poor FM-100 test scores were more likely to make errors discriminating between medicine capsules with similar color coding especially under relatively low illumination levels. Large-scale studies reported similar patterns of age-related loss in color discrimination performance (Verriest, van Laetham, & Uvijls, 1982; Roy et al., 1991). There is a linear increase in the number of color discrimination errors between 30 and 80 years of age. Much of this age-related change appears to be due to weakness in blue–yellow color mechanisms. That is, the color weakness that emerges with advancing adult age appears to mimic *tritanopia*—a relatively rare form of color anomaly resulting from a weakness in short wavelength (blue) cones. Thus, the typical older observer will tend to have difficulties discriminating between colored surfaces that differ by trace amounts of blue or yellow pigmentation. Representative results from a study of age differences in color discrimination using the FM-100 instrument by Kinnear and Schraie (2002) are presented in Figure 7.10. Note how the proportion of errors due to confusions along the blue–yellow axis of color space increases with age at a faster rate than errors along the red–green axis.

Much of the loss in the capacity for fine color discrimination appears to be related to senescent increases in the density of the ocular media, especially the yellowing and darkening of the crystalline lens. For example, Verriest (1963) reported that the performance of young persons on the FM-100 test became similar to that typically observed in the aged when those younger participants viewed test stimuli through a filter that selectively absorbed

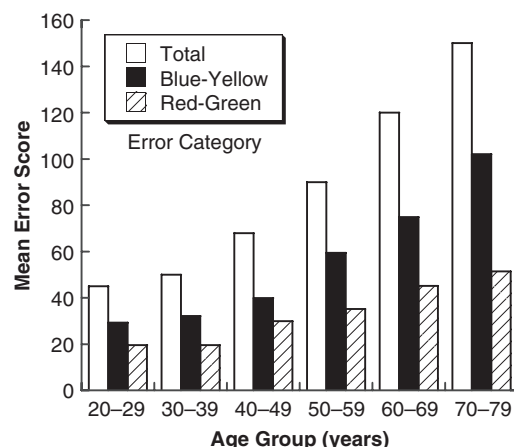


Figure 7.10 Error scores on Farnsworth–Munsell 100 hues test of color discrimination as a function of age. Source: Kinnear and Sahraie (2002).

short wavelength light. Knoblauch et al. (1987) demonstrated that the tritan-like color discrimination errors of older observers were mimicked in young and middle-aged adults when the FM-100 test was administered under conditions of reduced illumination (i.e., 5.7 lux instead of the traditional test illumination of 200 lux). This suggests that gain in the blue–yellow system may change more rapidly with reductions in the light adaptation level of the retina than gain in the red–green opponent color channel. A large-scale study of age differences in FM-100 color discrimination (Mäntyjärvi, 2001) used an illumination of 1000 lux instead of the conventional 200 lux standard illuminant. Compared to previous large-scale studies, the size of the aging effect appeared to be reduced.

Nonetheless, evidence shows that some of this loss in color discrimination performance may result from senescent changes in cones or postreceptoral processes. Numerous studies have reported age-related declines in the efficiency of the short wavelength (blue) cone system (Eisner et al., 1987; Haegerstrom-Portnoy, Hewlett, & Barr, 1989; Johnson et al., 1988). Other studies report

similar rates of age-related loss in sensitivity for middle wavelength (green) and long wavelength (red) cone systems as well. Werner and Steele (1988) examined age differences (10 to 84 years of age) in the sensitivity of short, medium and long wavelength cones using a chromatic adaptation isolation technique. Increment thresholds for monochromatic light stimuli selected to stimulate one class of cones were collected using a bright chromatic background adaptation field selected to reduce the sensitivity of the remaining two cone types. For example, use of a bright yellow background field suppresses the sensitivity of medium and long wavelength cones, thus allowing the short wavelength cones to set the limits on sensitivity for stimuli with wavelengths under 510 nm (Werner, Peterzell, & Scheetz, 1990). Increment thresholds collected in this fashion revealed similar patterns of age-related loss in sensitivity for all three cone types. More specifically, sensitivity losses of 0.12, 0.14, and 0.14 log units per decade were observed for short, medium, and long wavelength cones, respectively. Most of this loss in cone sensitivity observed across the life span could not be attributed to age-related changes in ocular media density.

Some of the most interesting research conducted recently on the topic of aging and vision has been concerned with the psychophysical scaling of suprathreshold color appearance. For example, Scheffrin and Werner (1993) used a psychophysical scaling procedure to examine potential age differences in the appearance of real world broadband stimuli (“color chips”) sampled from various regions across color space. Young (mean age = 21.3) and older (mean age = 71.9) participants screened for normal color vision and good ocular health rated the hue and colorfulness (i.e., color saturation) of five representative hues presented at three levels of lightness (i.e., apparent brightness of a reflective

surface). Hue scaling was accomplished by rating each sample based on four color components: %-redness, %-greenness, %-yellowness and %-blueness (the basic dimensions of opponent-color theory, described later). Participants were encouraged to use only two of these components when possible with the constraint that the sum of their hue component ratings for each stimulus must equal 100%. Given some practice, this scaling procedure has been demonstrated to yield reliable results (Gordon & Abramov, 1988). No age differences in hue scaling were observed, indicating that basic color naming performance was maintained through 70+ years of age. However, a different pattern of results was observed for judgments of perceived colorfulness or saturation. In a separate set of trials that followed the hue scaling procedure, these same observers rated each sample on a two component color saturation scale: %-chromatic versus %-achromatic appearance. Again, the sum of these components was constrained to 100%. The older observers consistently reported significant reductions in the strength of the chromatic component of their perceptual experience, especially at the lower lightness levels. In a follow-up study, Kraft and Werner (1999) found that this age-related reduction in the apparent colorfulness of chromatic stimuli was observed only in older adults with remarkable reductions in lens transmittance [i.e., less than 4% transmittance for short (420 nm) wavelength light].

The opponent-process theory, the most broadly supported theory of color vision, holds that output from long wavelength (L) and middle wavelength (M) cones are combined into a single push-pull, or opponent, "red-green" channel. Similarly, a second "blue-yellow" opponent channel is organized by contrasting input from short wavelength ("blue") cones with the summed inputs of L and M

cones (to form the "yellow" side of the opponent channel). A simplified prediction from this opponent-process theory of color vision holds that when activity within the red-green and blue-yellow channels is in equilibrium then no color contrast information is available. Such a condition leads to the perception of an achromatic stimulus. This is the so-called "white point" or "achromatic locus." Because the achromatic locus depends on the relative sensitivity of all three types of cones, as well as the dynamic response of both opponent-color processes, any systematic change in its color space coordinates with age would be indicative of compromised integrity of color perception. Indeed, well-known changes in the senescent lens, such as the differential absorption of short wavelength light, predict specific shifts in the coordinates of the achromatic locus with increasing adult age. That is, the combination of red, green, and blue color primaries needed to yield an achromatic experience would require a greater proportion of blue light to compensate for lens absorption (see Enoch et al., 1999). Yet, remarkably, Werner and Scheffrin (1993) have demonstrated that the achromatic locus remains unchanged with advancing adult age. They assessed the position of the "white point" in CIE color space at three levels of retinal illuminance (10, 100, and 1000 Trolands) in 50 observers ranging from 11 to 78 years of age. All observers were determined to be in good ocular health. No reliable age differences in the position of the achromatic locus were observed. The average CIE 1931 coordinates of the achromatic locus was $x=0.31$, $y=0.31$ at all three levels of illuminance. Similar results consistent with the interpretation that the overall integrity of color vision is well maintained during senescence had been reported previously by Scheffrin and Werner (1990) using a related paradigm

to explore the sensitivity of the blue–yellow color contrast coding mechanism.

As noted by Enoch et al. (1999), the stability of the achromatic locus initially suggests that adult aging is not associated with alterations in the basic mechanisms mediating the appearance of colored stimuli. However, upon more careful consideration it becomes obvious that one or more mechanisms must be operating to compensate for lenticular changes in short wavelength opacity. Indeed, it appears that the same mechanisms of local retinal adaptation responsible for the phenomenon of *color constancy* (the invariance of color perception across relatively wide changes in the spectral composition of naturally occurring illumination) may also serve to compensate for systematic age-related changes in the spectral transmission properties of the ocular media. Indeed, Werner and Scheffrin (1993) have presented quantitative evidence that modulation of the relative sensitivities of the three cone types could compensate for changes in the spectral content of the retinal image caused by age-related changes in the lens. Enoch et al. (1999) suggested that the mechanisms responsible for everyday color constancy may compensate for a broad range of senescent changes in the visual system in the service of maintaining robust color perception. A recent study by Delahunt et al., (2004) provides additional support for the working hypothesis that color constancy mechanisms contribute to the maintenance of robust color perception in old age. Cataracts tend to differentially absorb short wavelength light but to a much greater extent than normal aging of the lens. Immediately following cataract surgery, patients often report large shifts in color appearance. Predictably, blues and blue–greens are particularly affected. Delahunt et al. (2004) quantified these shifts in color appearance by assessing the achromatic locus (described earlier) before and after

cataract surgery in four patients (ranging from 63 to 84 years of age). Prior to surgery, participants reported near-normal achromatic loci in color space, indicating good compensation for short wavelength absorption by their cataracts. However, immediately following surgery the achromatic locus shifted toward the yellow region of color space. Such a shift was consistent with the hypothesized compensation in the blue–yellow mechanisms in response to the alterations in retinal illuminance resulting from cataracts. This postoperative shift in the achromatic locus gradually subsided over the course of the 12-month follow-up assessment. Of particular interest was the fact that the achromatic locus measured in the companion eye of each participant failed to demonstrate any of the postoperative shift or recovery for the achromatic locus. Consistent with the conclusions of Werner and Scheffrin (1993), lack of an interocular transfer for the postoperative shift in color appearance suggests that the compensatory processes responsible for maintaining robust senescent color perception occur at the level of the retina rather than in the visual cortex. Also of note, the time course of these adaptation processes occurs on a scale many orders of magnitude longer than that typically associated with classical mechanisms of color constancy such as von Kries adaptation.

E. Temporal Resolution

One of the most fundamental changes in vision that accompanies normal adult aging is the systematic loss of the ability to detect and efficiently process rapid temporal changes in the environment. Temporally contiguous visual events that would be seen as separate and distinct by young observers often appear “fused” or indistinguishable by older individuals (Kline & Schieber, 1982). This loss of

temporal resolving power in the visual system manifests itself in the form of apparent age-related slowing in higher order visual processes such as the sequential integration of form (see Kline & Orme-Rogers, 1978; Kline & Schieber, 1980) and backward masking (Kline & Birren, 1975; Walsh, 1976), but is also readily observed in the form of age-related deficits that emerge in rudimentary visual functions such as flicker sensitivity, dynamic visual acuity, speed, and motion perception. Theoretical speculations regarding the mechanism (or mechanisms) mediating this age-related loss in visual temporal resolution are numerous. Some investigators have explained such age differences within the context of general slowing due to random brain cell loss (Cerella, 1990); increased stimulus persistence (Botwinck, 1973); selective loss of transient/magnocellular channel sensitivity (Kline & Schieber, 1981); and, more recently, diminished temporal contrast sensitivity (Shinomori & Werner, 2002).

1. Flicker Sensitivity

The classical method for assessing the temporal resolution of low-level visual processes is the *critical flicker frequency* (CFF) threshold. The CFF represents the minimum frequency of a pulsating (high contrast) light source at which the light appears to be perceptually "fused" into a continuous, rather than flickering, stimulus. Because the stimulus at the CFF threshold is still physically oscillating between "on" and "off" states, it is thought to represent the temporal periodicity at which the visual system can no longer reliably detect rapid changes in scene luminance. There is a well-documented decline in the CFF threshold with advancing age (Brozek & Keys, 1945; McFarland, Warren, & Karis, 1958; Huntington & Simonson, 1965). For example, Wolf and Shaffra (1964)

collected CFF thresholds from a sample of 302 observers ranging from 6 to 95 years of age. Flicker sensitivity (and CFF thresholds) decreased gradually between childhood and 60 years of age and declined rapidly thereafter. Experimental manipulations have clearly revealed that much of the age-related decline in the CFF threshold can be attributed to reduced levels of retinal illumination accompanying normal changes in the ocular media and pupillary miosis. However, studies by Weekers and Roussel (1945) and McFarland et al. (1958) support the notion that a significant proportion of the age differences in the CFF may be attributable to senescent changes in the visual nervous system (see Kline & Schieber, 1985).

More contemporary studies of aging and flicker sensitivity have abandoned the CFF threshold paradigm in favor of measuring the *temporal contrast sensitivity function* (tCSF)—a more comprehensive assessment of visual system response in the temporal domain (Wright & Drasdo, 1985; Mayer et al., 1988; Tyler, 1989). When assessing the tCSF, the brightness of a small ($2\text{--}5^\circ$) self-luminous circular target is sinusoidally modulated at a given temporal frequency around a baseline luminance value. Next, the minimum luminance contrast modulation required to detect the presence of flicker is determined for a range of temporal frequencies, which typically extends from 1 to 50 cycles per second (Hz). Wright and Drasdo (1985) collected tCSFs from 70 observers sampled equally across the life span between 10 and 79 years of age. They reported an age-related decrease in temporal contrast sensitivity that grew in magnitude as temporal frequency was increased from 3.3 to 30 Hz. Tyler (1989) measured tCSFs in a large sample of observers ranging from 5 to 75 years of age. He reported a "leftward shift" in the shape of the tCSF among older observers, i.e., a migration of peak

temporal contrast sensitivity from higher to lower temporal frequencies. Tyler (1989) interpreted this age-related leftward shift in the shape of the tCSF as evidence for a generalized slowing in visual information processing and estimated the magnitude of this slowing to approach 20% by 75 years of age. However, Mayer and colleagues (1988) provided evidence that the qualitative difference in the shape of the tCSF for older observers that emerged in these studies could be accounted for primarily on the basis of age-related reductions in retinal illumination.

In a follow-up study, Kim and Meyer (1994) collected tCSF data from 89 observers ranging from 18 to 77 years of age. They used a bright (120 cd/m^2), long wavelength (660 nm) stimulus and statistically controlled for individual differences in pupil size to minimize the influence of any age-related variations in retinal illumination. Upon carefully controlling for differences in retinal illumination, they found a small but statistically significant age-related reduction in temporal contrast sensitivity across the range of temporal frequencies observed. The magnitude of this generalized loss of temporal contrast sensitivity was approximately 0.08 log units per decade beyond 44 years of age. Unlike Tyler's (1989) study, they failed to observe a leftward shift in the overall shape of the tCSF in their older observers and concluded that aging was characterized by a reduction in sensitivity of temporally tuned visual mechanisms rather than a generalized "slowing" in the rate of visual processing. Additional data analyses conducted by Kim and Mayer (1994) provided support for this interpretation of their data. According to linear systems theory, the *impulse response function* (IRF) quantitatively describes the response of the visual system to temporally modulated stimulus input. The classic IRF is biphasic, consisting of

an initial excitatory response followed shortly by an inhibitory rebound (see in Figure 7.11B for some examples of an impulse response function). Within constraints, the IRF can be derived via Fourier transformation of the tCSF curve. Upon deriving IRFs in this way for each age group, a clear pattern emerged. Both the peak amplitude and the area of the first (excitatory) lobe of the IRFs for older observers were significantly diminished in magnitude. However, neither the latency of the peak response nor the zero crossing (point of transition between excitatory and inhibitory lobes) slowed with advancing adult age.

Shinomori and Werner (2003) noted that there are some mathematical and conceptual problems associated with the derivation of the temporal impulse response (tIRF) from the temporal contrast sensitivity function (tCSF) using Fourier transformation. tIRFs derived in this manner appear to preserve response amplitude but may not adequately preserve phase (i.e., temporal) information. They proposed that an alternative psychophysical method and modeling approach (see Burr & Morrone, 1993) could be used to assess potential age differences more accurately in the tIRF of the visual system. This approach is based on the *two-pulse contrast detection function* (2PCDF). The 2PCDF is generated by measuring the minimum contrast needed to detect the occurrence of a pair of rapidly presented light pulses as a function of the variable interstimulus interval separating these pulses. The shape of the tIRF can be derived more accurately from 2PCDF data because the autocorrelation function is preserved (for technical details, see Shinomori & Werner, 2003). Two-pulse contrast detection functions were collected from 70 observers between 16 and 86 years of age. Pulses with a duration of 1.2 ms and interstimulus intervals ranging from 6.7 to 180 ms were employed. Representative

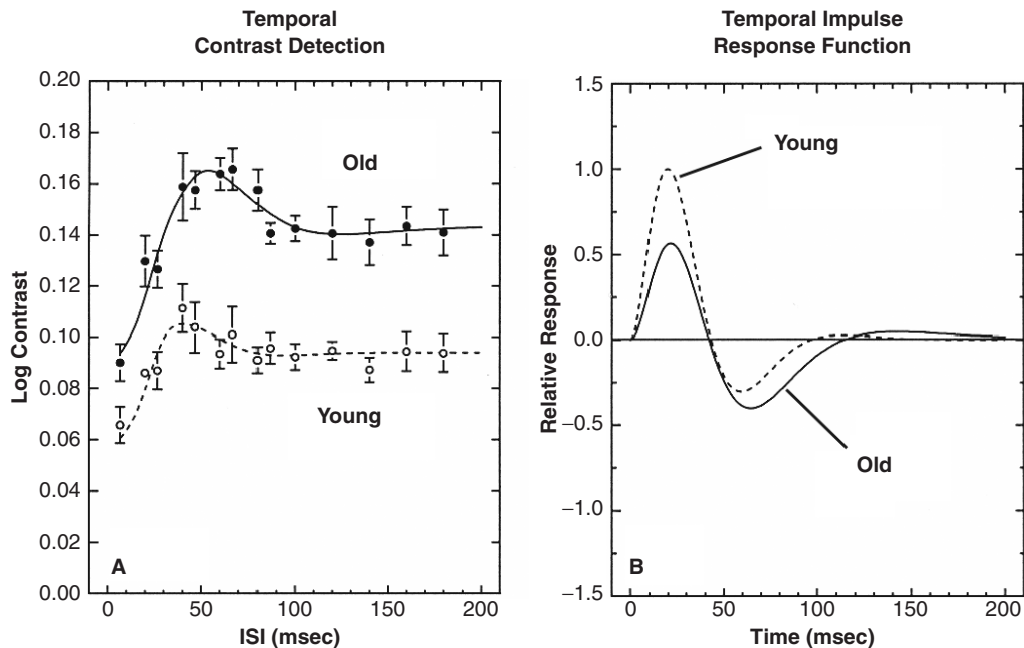


Figure 7.11 Two-pulse contrast detection functions (A) and derived temporal impulse response functions (B) for a representative young and older observer. Source: Shinomori and Werner (2003).

2PCDFs and derived tIRFs for a young and an older observer, respectively, are depicted in Figure 7.11. The investigators reported that two-pulse contrast detection thresholds were uniformly elevated with advancing adult age. More interesting, given the current context, was the finding that tIRFs of older observers consistently demonstrated reduced amplitude for the initial excitatory phase with no systematic age differences in the latency of the peak response (or zero crossing between response phases). Hence, Shinomori and Werner (2003) were able to independently confirm the previous conclusion of Kim and Mayer (1994) using an entirely different approach. It would appear, therefore, that age-related changes in flicker sensitivity are indicative of reduced contrast sensitivity within temporally tuned visual mechanisms instead of a more generalized slowing in visual processing. It should be noted, however, that this does

not preclude visual slowing as a mediator in other areas of visual perception. Finally, Shinomori and Werner (2003) also noted that the second, or inhibitory, lobe of the tIRF was attenuated greatly or entirely missing in many of their oldest participants. A visual system characterized by such a loss in late, inhibitory processes appears to be consistent with the classical notion of increased “stimulus persistence” in senescent visual information processing (see Kline & Schieber, 1982).

2. Motion Sensitivity

Numerous studies have reported age-related declines in motion detection and discrimination thresholds. One of the most basic approaches to measuring motion sensitivity involves the assessment of the *oscillatory motion displacement threshold* (OMDT). In this paradigm, the spatial position of a

stimulus is displaced rhythmically along the horizontal or vertical meridian in a sinusoidal fashion. The minimum amplitude of displacement required to reliably discriminate moving from stationary stimuli is then determined as a function of the temporal frequency of spatial oscillation. Buckingham, Whitaker, and Banford (1987) collected OMDTs from a sample of young (mean age: 20.7), middle-aged (mean age: 48.0), and older (mean age: 69.7) observers using a low spatial-frequency sine wave grating target that oscillated at frequencies ranging from 1 to 20 Hz. Marked and consistent age-related declines in motion sensitivity were observed across the full range of temporal frequencies. For example, at 8 Hz mean OMDTs of 39, 52, and 97 s of arc were observed for the young, middle-aged, and older observers, respectively. Similar findings have been reported by other investigators who have used careful experimental controls indicating that such sizable age differences in OMDT measures of motion sensitivity are independent of age-related changes in retinal illumination and/or refractive error (e.g., Elliott, Whitaker, & Thompson, 1989; Schieber et al., 1990; Kline et al., 1994). As such, OMDT appears to represent a class of hyperacuity phenomena that may provide a uniquely powerful approach to separating the effects of neural versus optical aging in the visual system.

Several groups of investigators have used random dot motion paradigms to study age differences in the sensitivity of *global motion* detection mechanisms. Observers are presented with a large number of small, randomly positioned dots on a computer display. Using basic animation techniques, these dots then take "random walks" across the display during brief stimulus exposures. Under the conditions just described, the observer can not perceive a principal direction of motion in the flow of dots (because there is none). More and more "correlated

motion" is gradually added to the stimulus display (by having more and more dots move in a given direction across subsequent frames) until the observer can reliably detect a directional trend in the overall pattern of motion. The amount of correlated motion required to correctly discriminate the direction of flow is the *global motion threshold*. Motion in this case is said to be global, as it represents a statistical trend in the entire population of dot stimuli rather than within any given dot *per se*. Dots contributing to the correlated direction of flow are assigned randomly from one frame of the animation sequence to the next. Trick and Silverman (1991) used this technique to assess age differences in global motion sensitivity. They found that motion sensitivity declined by 50% between 25 and 80 years of age. Gilmore and colleagues (1992) also found sizable age-related declines in global motion sensitivity using a random dot motion paradigm. However, such reductions in global motion sensitivity were limited to the female members of their sample of older observers. Unfortunately, the earlier study by Trick and Silverman (1991) did not report any analyses of their data based on gender. However, two studies using the random dot paradigm that did conduct such analyses have also reported the same interaction, namely global motion sensitivity was reduced in older females but not in older males. Experimental controls in both of these studies indicated that these gender-specific losses were independent of ocular factors, such as differences in retinal illuminance or refractive error (Schieber et al., 1990; Atchley & Anderson, 1998). In the Schieber et al., (1990) study of global motion sensitivity, oscillatory motion detection thresholds (OMDTs) for single dot stimuli were also measured. A strong age-related increase in OMDTs of equal magnitude was observed among both older males and females. This pattern of results suggests that the global

motion deficit observed in older females is probably mediated by a reduced efficiency of "spatial pooling" or integration of the motion signal over large regions of space at a location in the nervous system beyond the primary visual cortex. Previous studies using the random dot paradigm indicate that a likely site for this visual processing deficit in older females is the medial temporal (MT) area (see Newsome & Pare, 1988; Zeki et al., 1991).

3. *Speed Perception*

Clear and compelling evidence shows age-related declines in motion sensitivity under ideal laboratory conditions. What is less clear, however, is how this motion sensitivity deficit scales to real world judgments of absolute and relative speed such as those necessary for safe and efficient operation of a motor vehicle. Scialfa and co-workers (1991) examined age differences in the magnitude estimations of vehicle velocity for actual automobiles traveling around a test track at speeds varying from 15 to 50 mph (24–80 kph). Young (mean age: 22.2) observers tended to underestimate the speed of slowly moving vehicles (15 mph) and overestimate the speed of the most rapidly moving vehicle (50 mph). Older (mean age: 65.3) observers demonstrated a similar, but less severe, slow underestimation/fast overestimation bias. The resulting psychophysical functions relating perceived speed to actual speed suggested that older observers were less sensitive to relative changes in velocity, but, at the same time, demonstrated more accurate absolute judgments of speed at the two ends of the velocity range examined. Staplin, Lococo, and Sim (1993) conducted a series of complementary simulation and field studies that strongly suggested that the perceptual basis of "time-to-collision" and "traffic gap acceptance judgments" changes significantly with advancing adult age.

Relative to younger drivers, older observers seated in a stationary vehicle tended to underestimate the time required for an approaching vehicle to reach their current position. Although less accurate, these judgments erred on the conservative side (i.e., persons making such errors would be less rather than more likely to become engaged in a traffic conflict). Andersen and co-workers (2000) reported similar findings in a part-task visual simulation of vehicular deceleration while approaching a stop sign at an intersection. These authors speculate that such losses in the accuracy of effortless visual perceptual guidance may require compensatory cognitive effort while negotiating intersections. The full costs of such perceptual to cognitive trade-offs remain to be determined.

4. *Form from Motion*

Spatial vision is principally concerned with the processing and recognition of forms defined by variations in luminance and/or wavelength across space (i.e., luminance and/or color contrast). However, form perception can also be quite robust for motion gradients across space. For example, observers typically have no difficulty perceiving the border between two adjacent regions in a field of random dots moving at sufficiently different angular velocities (Regan & Hong, 1990). Several studies suggest that the ability to perceive motion-defined contours declines dramatically with advancing adult age. For example, Wist, Schrauf, and Ehrenstein (2000) assessed the ability of observers to discriminate between simple geometric forms defined by briefly presented (240 ms) motion gradients (stationary versus moving) in a high-density random dot field. The amount of motion contrast required to discriminate such motion-defined stimuli increased dramatically between 20 and 70 years of age. In fact, half of the observers

over the age of 70 were completely unable to discriminate motion-defined form when 100% of the dots in the foreground region moved at a robust level of $1.3^\circ/\text{s}$. In a related study, Andersen and Atchley (1995) reported that older observers had difficulty perceiving motion-defined, three-dimensional-corrugated surfaces. Similarly, Norman, and colleagues (2004) reported that older observers demonstrated difficulty recognizing short animated sequences (6 frames across 240 ms) depicting "biological motion." However, much of this age-related deficit disappeared for longer animated sequences (i.e., 10 frames across 400 ms). The variety of age-related problems observed with the perception of *form-from-motion* stimuli suggests that additional investigations into these phenomena hold great promise for helping to better understand the neurophysiological mechanisms mediating visual aging.

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