Low Vision and Plasticity: Implications for Rehabilitation

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Abstract
Low vision is any type of visual impairment that affects activities of daily living. In the context of low vision, we define plasticity as changes in brain or perceptual behavior that follow the onset of visual impairment and that are not directly due to the underlying pathology. An important goal of low-vision research is to determine how plasticity affects visual performance of everyday activities. In this review, we consider the levels of the visual system at which plasticity occurs, the impact of age and visual experience on plasticity, and whether plastic changes are spontaneous or require explicit training. We also discuss how plasticity may affect low-vision rehabilitation. Developments in retinal imaging, noninvasive brain imaging, and eye tracking have supplemented traditional clinical and psychophysical methods for assessing how the visual system adapts to visual impairment. Findings from contemporary research are providing tools to guide people with low vision in adopting appropriate rehabilitation strategies.
1. INTRODUCTION

In this review, we discuss plasticity and its implications for rehabilitation in low vision. For reviews of neuroplasticity in profound blindness, see Merabet & Pascual-Leone (2010) and Hirsch et al. (2015).

What do we mean by plasticity in the context of low vision? Eye disease or injury causes direct and observable changes to the anatomy of vision. These changes are usually early in the visual pathway and typically result in immediate and measurable changes in both anatomy and behavioral tests of visual function. For example, macular degeneration results in observable damage to the central retina and produces deficits in acuity and scotomas in the visual field. The visual system may adapt to damage caused by the pathology in ways that influence characteristics of neural processing and perception. These changes may occur spontaneously or may depend on the nature of subsequent visual experiences or training.

Our focus in this review is to consider plasticity as the changes subsequent to but not immediately attributable to the causal pathology. By analogy, the closure of a bridge on a busy city street has an immediate impact on the flow of traffic along the street just as macular degeneration affects the flow of visual information along the optic nerve. But the bridge closure can also have longer-term and widespread effects on the traffic flow on undamaged streets as drivers adapt their behavior to the bridge closure. Similarly, functioning visual pathways may adapt to pathology at an early anatomical site, such as the optics of the eye or retina. These plastic changes may be observed by studies of anatomy, neural processing, or visual behavior.

What are the impacts of visual experience and the age of onset of low vision on the potential for plasticity? Following the seminal deprivation studies on development of the primary visual cortex in kittens by Wiesel & Hubel (1965a,b), a prevailing view emerged that visual function in general would not recover from long-term deprivation after an early critical period. But recent evidence for improved perceptual function in adults with impaired vision reveals the possibility of adult plasticity. This is particularly important for rehabilitation, given that most low vision has its onset later in life. Ongoing research seeks to determine the anatomical sites and neural processing underlying this form of plasticity and to determine whether adult visual plasticity occurs spontaneously or requires explicit practice or training.

Rehabilitation deals with a person’s functional limitations by providing remedies and strategies for overcoming these limitations. Rehabilitation provides a visually impaired person with skills that allow functioning in the everyday world. Typically, rehabilitation focuses on prescribing mobility skills and devices (canes, dog guides, GPS, telescopes), reading aids and skills (magnifiers, training for eccentric viewing), and strategies for dealing with activities of daily living (cooking, shopping, grooming).

What is the significance of plasticity for rehabilitation? We may be able to quantify a change in a clinical outcome measure, such as acuity or a change in the pattern of brain activation in functional magnetic resonance imaging (fMRI), but does the change correspond to a useful improvement in real-world function? We typically regard the consequences of plasticity as providing rehabilitative benefits for the individual, but there may be conditions in which plasticity of the visual system forecloses options for later treatment or therapy.

In this review, we discuss the following questions:

■ What are the perceptual and physiological indicators of plasticity in low vision?
■ What roles do visual experience and age play in shaping these changes?
■ Are the changes spontaneous, or do they depend on training or practice?
■ Are the plastic changes relevant to rehabilitation?

Before turning to methods for measuring low vision, we summarize its definition and prevalence. Low vision is any chronic form of vision impairment not correctable by glasses or contact
lenses that adversely affects everyday functions. Low vision is sometimes defined as the inability to read newsprint at a distance of 40 cm with best refractive correction. This definition is used because most people with low vision have problems with reading (Elliott et al. 1997a, Owsley et al. 2009). When a more quantitative definition is required, low vision is often defined as visual acuity less than 20/60 (6/18) or a visual field with a maximum extent of less than 20° in the better eye. Note that the definition of low vision is more inclusive than the statutory definition of legal blindness—defined in the United States as a corrected visual acuity in the better eye of no more than 20/200, or a visual field of no more than 20°.

By a recent estimate, there are 285 million people worldwide with vision impairments: 39 million are blind, and 246 million have low vision (WHO 2014). These figures include many people in less-developed countries whose impaired vision is due to uncorrected refractive errors or untreated cataracts.

By conservative estimates, there are between 3.5 and 5 million people in the United States with low vision (Natl. Eye Inst. n.d.), and the number is rising as the U.S. population ages. Because the leading causes of visual impairment in the United States are age-related eye diseases—macular degeneration, glaucoma, diabetic retinopathy, and cataract—the prevalence of impaired vision rises steeply with age, with a greater prevalence among females (66%).

2. MEASURING PLASTICITY IN LOW VISION

2.1. Behavioral Measurements

Three types of behavioral studies have been used to examine plasticity in people with low vision: (a) interpreting differences in visual function between low-vision patients and normally sighted controls as evidence of plasticity; (b) longitudinal studies tracking changes in visual function not directly attributable to the underlying pathology; and (c) training or perceptual learning, intended to improve a specific aspect of visual function. These studies rely on a variety of outcome measures.

The most common outcome measures for evaluating functional changes are visual acuity, contrast sensitivity, and visual field. In research settings, acuity is usually measured with modern letter charts that follow robust design principles and that have high test-retest reliability. The best known are the Bailey-Lovie letter chart (Bailey & Lovie 1976) and the ETDRS chart (Ferris et al. 1982). Contrast sensitivity is often measured with letter charts as well, with the most widely used tests being the Pelli-Robson chart (Pelli et al. 1988) and the MARS Chart (Arditi 2005). A more complete characterization of low vision can be obtained from measurement of a contrast-sensitivity function (CSF) (Chung & Legge 2016). CSFs have rarely been measured for low vision because of the technical difficulty and patient time required. Recently, the quick-CSF method has been developed for use in clinical studies (Lesmes et al. 2010).

Visual field perimetry is also an important outcome measure when low vision involves field loss. The Humphrey Visual Field Analyzer is often the instrument of choice for subjects with peripheral field loss or hemianopia. In the case of central field loss, the Humphrey 10-2 test, which focuses on the central 10° of the visual field, may be used. Recently, microperimeters have been introduced to evaluate the sensitivity of the central field, such as the Nidek MP-1 and MAIA. These microperimeters can deliver test stimuli to known retinal locations despite unstable eye movements. They do so by imaging and tracking retinal landmarks to compensate for eye movements. Microperimeters can also measure fixation stability, a property that has received increasing attention in low-vision research.

Other outcome measures may be more appropriate for evaluating performance on specific tasks related to real-world activities. For example, several tests have been developed to assess reading performance, including the Bailey-Lovie near-vision chart (Bailey & Lovie 1980), the
MNREAD visual acuity chart (Mansfield et al. 1993, Mansfield & Legge 2007), the Radner reading test (Radner et al. 2002), and the IReST reading tests (Trauzettel-Klosinski et al. 2012). Other psychophysical laboratory tests for evaluating performance on specific real-life tasks include visual search (Wiecek et al. 2012), face recognition (Bullimore et al. 1991), pedestrian mobility (Marron & Bailey 1982, Kuyk et al. 1996, Turano et al. 2004), the visual span for letter recognition (Legge et al. 2001), and the useful field of view as a predictor of driving ability (Ball et al. 1993).

Even if we observe an improvement in the objective measurements discussed so far, do people report a benefit or improved ability in performing tasks of daily living? Quality-of-life questionnaires are widely used to quantify subjective benefits. In the United States, two questionnaires have been developed for assessment of the health-related quality of life of people with low vision. These are the 25-item version of the National Eye Institute Visual Function Questionnaire (NEI VFQ-25) (Mangione et al. 2001) and the Veterans Affairs Low-Vision Visual Functioning Questionnaire (VA LV VFQ-48) (Stelmack et al. 2004). Rasch analysis is often used to improve the validity of the scoring method of questionnaires (Massof & Fletcher 2001, Dougherty & Bullimore 2010, Marella et al. 2010). Linking the low-vision patient’s experience, measured with quality-of-life questionnaires, with objective behavioral or brain-imaging measures may be one way of determining the relevance of plastic changes to rehabilitation.

2.2. Imaging Measurements

In the past 30 years, new imaging techniques have been used to study changes in retinal or cortical structures in low vision. In the 1980s, the first scanning laser ophthalmoscope (SLO) was developed to allow simultaneous stimulus presentation and imaging of the retina (Timberlake et al. 1982). Subsequently, SLO has become an invaluable instrument for determining the location of the preferred retinal locus (PRL) of patients with central field loss. Other techniques have also been used for this purpose, such as a modified fundus camera (White & Bedell 1990). More recently, other retinal-imaging instruments have found their way into low-vision research, including the Nidek MP-1, MAIA, OCT/SLO (Seiple et al. 2013), tracking SLO (Kumar & Chung 2015), and the adaptive-optics SLO (Duncan et al. 2011). Because of the trade-off between field size and spatial resolution, the best choice of instrument often depends on the research question being addressed.

Until recently, there was little opportunity for direct study of changes in visual cortex following the onset of impaired vision. The advent of noninvasive brain-imaging methods has made it possible to study brain structure and functional response in low vision. As examples of structural imaging, Ptito et al. (2008) used whole-brain magnetic resonance imaging voxel-based morphometry to observe the afferent projections to the visual cortex in people with congenital blindness; this showed significant atrophy of the optic nerves, optic chiasm, optic radiations, V1, V2, and MT. Hernowo et al. (2014) used similar methods to observe atrophy of the early visual pathway in macular degeneration. Diffusion tensor imaging was used to confirm a loss of white matter integrity in optic radiations in blind subjects compared with normal control subjects (Wang et al. 2013, Dietrich et al. 2015), with people who lost their vision after age 18 years showing more loss than congenitally blind individuals (Wang et al. 2013).

Functional methods, including positron emission tomography and fMRI, have been used to study cortical organization following vision loss. Examples are reviewed in Section 5. A common measurement used by brain-imaging studies to represent the level of activation of specific brain regions is the change in the hemodynamic response, more specifically, the blood-oxygen level-dependent (BOLD) response. The underlying assumption is that active neurons require more oxygen; thus the relative levels of oxyhemoglobin and deoxyhemoglobin can represent cortical...
activity. Computational methods based on measurements of population receptive fields (PRFs, the fMRI analog of receptive fields of single neurons measured in electrophysiology) have been used to document changes in the visual cortex associated with pathology (Wandell & Winawer 2015). The PRF specifies a region of visual space (location and extent) within which stimuli produce activation in a cortical voxel. PRFs generally increase in size from central vision outward, but the relationship differs across cortical maps in the visual hierarchy. Analysis of PRFs in low vision may reveal how the visual cortex changes in response to reduced visual input.

3. SIGHT RESTORATION FOLLOWING EARLY DEPRIVATION

Humans are born with immature pattern vision. Full-term newborns have behaviorally measured grating acuities of only 0.7 cycles/degree (Snellen equivalent of 20/860) compared with adult values of 30 cycles/degree or more (Brown & Yamamoto 1986), and contrast sensitivities of only 2 compared with adult values of at least 100 (Brown et al. 2015). Development of normal adult visual function requires visual experience. Infants born with dense congenital cataracts, for example, experience reduced adult acuity with any delay in cataract removal. In one study, adult acuity averaged 20/80 for removal of congenital cataracts at ages 14 to 31 weeks (Birch et al. 2009).

Visual functions develop at different rates, reaching adult levels as early as two months for critical flicker fusion, six to seven years for acuity and contrast sensitivity (Ellemberg et al. 1999), the grade school years for the visual span for reading (Kwon et al. 2007), and the teens for face perception (Lewis & Maurer 2009), but see McKone et al. (2012) for a countering view on the maturation of face perception. Given these differences, it is expected that the age of onset of visual impairment influences the extent of subsequent plastic changes and the potential for recovery of normal vision if restorative treatment becomes available.

Studies revealing critical periods for normal visual development in humans and studies of the irreversible effects of early deprivation on cells in the visual cortex of kittens (Wiesel & Hubel 1965a,b) led to the view that recovery of visual function following long-term visual deprivation was absent or largely incomplete. Case studies on attempts at vision restoration in humans following long periods of congenital or early-onset blindness revealed poor outcomes (von Senden 1960 (1932), Valvo 1971, Gregory & Wallace 1963). More details are available from a recent case study of subject MM. He had normal vision until age 3.5 years when a chemical accident caused the loss of one eye and serious corneal damage to the other. He retained light perception but no pattern vision. More than 40 years later, limbal stem-cell surgery and a corneal transplant restored his vision in the sense of good image formation on the retina. Behavioral measurements made within two years after the surgery revealed grating acuity of only about 1.2 cycles/degree (Snellen equivalent of 20/500), nearly normal color and motion perception, and the ability to recognize two-dimensional shapes, but severe impairment in higher-level visual functions including three-dimensional object and face recognition (Fine et al. 2003). Brain imaging (fMRI) revealed that MM’s visual cortex was highly responsive to motion cues, but it did not show normal category-specific responses for faces and objects within the ventral visual pathway (Fine et al. 2003). Testing ten years later revealed no clear improvement in MM’s acuity or higher-level visual function (Huber et al. 2015). The conclusion from these case studies is that, if experience-dependent visual functions do not develop normally prior to visual deprivation, they do not develop following vision restoration. Some capacities that had matured may also decline through long periods of deprivation, such as visual acuity in the case of MM.

Given these findings, some recent results imply surprising adult plasticity after long-term deprivation. Amblyopia is the reduced acuity and contrast sensitivity in one eye, which often
accompanies strabismus or a major difference in the refractive powers of the two eyes (anisometropia). The conventional view is that treatment for amblyopia is ineffective after a critical period ending at about seven years, but recent studies indicate that some types of extensive visual practice (Levi & Li 2009, Hess et al. 2011) can yield measurable improvements in acuity in the amblyopic eye and stereoacuity in adults with amblyopia. Although amblyopia is not considered to be a form of low vision, these improvements are evidence for plasticity in adult visual function.

Further evidence for plasticity of visual function in late childhood or early adulthood comes from Project Prakash (Sanskrit for light). This project has the humanitarian goal of providing sight-restoring surgery to the large population of children in India with congenital cataracts and other forms of treatable early blindness, and the scientific goal of studying the recovery of vision when sight is restored following early and prolonged deprivation (Sinha et al. 2013). The project has revealed forms of plasticity in both low-level sensory function and higher-level perception.

Kalia et al. (2014) used the quick-CSF method to measure CSFs following cataract removal in 11 Prakash patients with blindness from dense bilateral congenital cataracts. Surgery occurred at ages 8 to 17 years, past the age at which normally developing contrast sensitivity reaches the adult level. Prior to surgery, all of the subjects had little or no pattern vision, with acuities listed as hand motion or finger counting. Postsurgical acuities ranged from 20/428 to 20/103. Kalia et al. also measured postsurgical CSFs separated by six months to determine whether vision would continue to improve beyond levels achieved immediately after surgery. They found that 5 of the 11 patients had significant postsurgical improvements over the six-month period. Peak contrast sensitivities increased as much as 30-fold for two subjects. The high-frequency cutoffs (the highest frequency visible at maximum contrast) also improved for the five subjects but did not exceed 2 to 4 cycles/degree, compared with cutoff frequencies of 20–30 cycles/degree for normally sighted controls. The continuing improvement in vision following surgery of the five subjects implied that not all of the gain was due to better retinal-image quality; at least a portion of the improvement was likely cortical in origin. Kalia et al. did not find a significant correlation between the extent of improved contrast sensitivity and the age at surgery, time since surgery, presurgery acuity, or type of cataract. Perhaps the absence of significant correlations was due to the relatively small sample of subjects in the study. The results, however, indicate that development of contrast sensitivity can be delayed until at least late adolescence.

Project Prakash has also addressed a famous philosophical question, posed by William Molyneux in 1688. Suppose a person is born blind and learns shapes by touch, and then has vision restored. Would the person be able to recognize the shapes with vision? Held et al. (2011) reported on experiments with Prakash subjects following cataract surgery. They were tested in a match-to-sample task. The subject saw or touched a sample object and then had to choose it from a pair of objects presented either by touch or by vision. Immediately after surgery, subjects performed well in matching touch to touch and vision to vision but not in the transfer task; that is, they could not reliably use vision to select the object they had just touched. These results provide a negative answer to Molyneux’s question. But within a few weeks, the same subjects were able to accurately perform the cross-modal matching task, implying that they had developed the ability to incorporate visual information into multimodal object representations.

The Prakash results indicate significant plasticity in the acquisition of visual function after a lengthy period of congenital or early-onset visual impairment. This is promising for rehabilitation. Surgical treatments or therapies may yield improvement in vision even in adulthood. But, coupled with the results from subject MM and prior case studies, it is clear that the restoration of vision falls well short of the capacities of normally sighted adults. In Section 5 below, we discuss why complete sight recovery might not occur in cases of early-onset low vision.
4. VISUAL PLASTICITY FOLLOWING ADULT-ONSET DEPRIVATION

The concept of a critical period refers to the developmental stage in which visual experience, normal or abnormal, has a major impact on the organization of the cortical network for visual processing. Beyond the critical period, the cortical network is more stable and less adaptable to altered sensory input (Wandell & Smirnakis 2009). For this reason, we expect to observe less visual plasticity following late-onset low vision from eye diseases, such as cataract and age-related macular degeneration (AMD). In the following, we present evidence that some degree of visual plasticity is still present in adults with normal or low vision.

This issue is of great scientific interest and also has important implications for rehabilitation. If a person’s visual system does not adapt or change following adult-onset low vision, then a cure for the disorder would be expected to restore normal vision. A common example is age-related cataract. Typically, visual functions return to normal following successful cataract surgery. But if the aging visual system is unable to adapt, patients with adult-onset low vision may not benefit from functional improvements as a result of interventions, including training or perceptual learning. Given the practical implications, it is imperative to understand the adult brain’s potential for adapting to modified visual experience and to understand how this potential may decline in the senior years when the prevalence of visual impairment increases (Congdon et al. 2004).

There are different ways to alter the visual experience of an individual. One way is through training or perceptual learning where a subject is tested repeatedly with the same or similar stimuli. In normally sighted adults, performance improvements can be observed following an intensive period of training, often over a week or several weeks, for tasks such as position judgment (Li et al. 2004), orientation discrimination (Lu & Dosher 2004), face identification (Gold et al. 1999), texture identification (Gold et al. 1999), and letter recognition (Chung et al. 2004, 2005) even in older adults (Andersen et al. 2010, Yu et al. 2010).

Another way to alter visual experience is through a systematic restriction of a subject’s normal visual environment. After exposure to orientation-specific deprivation for four hours, Zhang et al. (2009) observed an increased sensitivity to the deprived orientation, compared with the orthogonal control orientation. Kwon et al. (2009) used special goggles to expose their subjects to a low-contrast environment for four hours. Following this exposure, their subjects showed improved contrast-discrimination performance and also increased fMRI BOLD responses in cortical areas V1 and V2. These changes were attributed to an increase in response gain in visual cortex associated with the reduced contrast in the subjects’ visual environment. The enhanced responsiveness of the human visual cortex to weak inputs is consistent with the findings of Boroojerdi et al. (2000). They measured threshold amplitudes of transcranial-magnetic stimulation pulses applied to the visual cortex to produce phosphenes (the perception of flashes of light). They found a progressive increase in phosphene sensitivity during three hours of light deprivation, with sensitivity returning to the baseline value approximately three hours after light deprivation ceased. Cross-modal changes in the visual cortex are also observed following light deprivation. After five days of being blindfolded, normally sighted subjects performed better on a tactile discrimination task, accompanied by an increase in fMRI BOLD signal within the occipital cortex in response to tactile stimulation (Merabet et al. 2008). The increase in BOLD signal disappeared within 24 h following deprivation. These findings provide evidence for visual plasticity in the normal adult brain.

In the aforementioned studies, the visual deprivation was artificial and temporary, lasting only several hours to a few days. The effects may not be representative of the visual deprivation experienced by people with naturally occurring visual impairments. We now turn to evidence that visual plasticity is also observed in low vision.

Age-related cataracts, a major cause of visual impairments in the elderly population, are unusual in having a highly successful treatment option—cataract extraction and implant of an artificial
intraocular lens. Although patients often have the cataracts for years before they undergo surgery, postsurgical visual functions (acuity, contrast sensitivity) usually return to normal values (Rubin et al. 1993, Elliott et al. 1997b), with a subjective improvement in vision (Javitt et al. 1993). There are interesting perceptual biases associated with having a cataract and its subsequent removal. Cataracts produce retinal-image blur and tend to filter out short-wavelength (blue) light. Cataract patients often report their environment to be richer in warm colors but report changes to cooler and more vivid colors immediately following cataract removal. When cataract patients are asked to make settings such that visual stimuli appear achromatic, the settings tend to be in the bluish region but shift to the yellow region after the cataract surgery to compensate for the additional short-wavelength light reaching the retina (Delahunt et al. 2004). Before cataract surgery, patients judge slightly blurred natural-scene images as best focused, but these images are reported to appear too sharp following cataract surgery (Parkosadze et al. 2013). Although acuity may return to normal values within days after cataract surgery, these subjective biases in color and sharpness perception may persist for months. The subjective biases before and after cataract surgery indicate that the visual pathways are capable of adjusting to the modified visual environment produced by eye disease or eye therapy, even in old age.

There is evidence that subjects with macular degeneration can improve their reading performance as a result of perceptual learning. Chung (2011) first showed that reading speed measured using the rapid serial visual presentation (RSVP) paradigm improved by an average of 53% following six weekly sessions of training in subjects with AMD or Stargardt disease (an early-onset form of macular degeneration). Subsequent studies using smaller print size (Tarita-Nistor et al. 2014) or testing only subjects with Stargardt disease (Nguyen et al. 2011) confirmed the effectiveness of perceptual learning for enhancing reading speed. Seiple et al. (2011) found that an oculomotor training task was more effective than RSVP training in improving reading speed for subjects with AMD. Rosengarth et al. (2013) also reported that following eccentric viewing training, AMD subjects showed improvements in their fixation stability, visual acuity, and reading speed, with the improvement in fixation stability having a positive correlation with changes in fMRI BOLD signals during training (but not after training) in V1, V2, and V3. The reading improvements exhibited by people with macular degeneration are of roughly the same magnitude as improvements exhibited by adults with normal vision in a similar task (Chung et al. 2004) and may reflect the same underlying capability of adult vision for perceptual learning. It is encouraging from a rehabilitation standpoint that visually impaired individuals can benefit from this approach.

Cortical lesions can also cause low vision, with strokes, traumatic brain injuries, and tumors being the primary etiologies. The hallmark consequence of cortical visual impairment is homonymous hemianopia—a loss of visual field in the same visual space in the two eyes. There is no treatment for homonymous hemianopia, but patients are often taught to make more scanning eye movements into the blind side as a compensatory strategy. In 1998, Sabel and his colleagues (Kasten et al. 1998) suggested that vision restoration therapy (VRT), a training program that is similar to repeatedly performing perimetry (measurement of the visual field), with emphases at the transition zone between seeing and nonseeing areas, was effective in enlarging the visual field. However, the improvements were only observed with the training program and could not be replicated in standard clinical perimeters (Kasten et al. 1998) or when eye movements were monitored carefully using an SLO (Reinhard et al. 2005). To date, it remains unclear whether the improvements demonstrated by Sabel and his colleagues were due to a true plasticity of the neurons at the transition between the seeing and nonseeing areas, artifacts of uncontrolled eye movements, or attentional or even placebo effects (Horton 2005a,b; Glisson 2006; Reinhard et al. 2005). A recent study showed that a greater expansion in the visual field border was observed when
VRT was combined with transcranial direct current stimulation (a noninvasive cortical stimulation technique), compared with VRT plus sham stimulation (Plow et al. 2012).

5. CORTICAL PLASTICITY IN LOW VISION

Some forms of low vision arise from the genetic abnormalities of visual development associated with unusual organization of the visual cortex. Albinism is a genetic abnormality characterized by a disorder in the synthesis of the pigment melanin. Typically, people with albinism have low vision, characterized by foveal hypoplasia, nystagmus, strabismus, and reduced acuity (Summers 2009). They have abnormal projections of optic nerve fibers from the retina to visual cortex (Guillery et al. 1975). In normal human vision, fibers from the temporal retina project to the ipsilateral hemisphere, whereas nasal fibers have contralateral projections, crossing at the optic chiasm. In humans with albinism, some of the temporal fibers also cross at the optic chiasm to the contralateral hemisphere. An fMRI study revealed that the abnormal projection produces representation of a portion of the temporal hemifield, extending up to 14° across the midline, superimposed on the representation of nasal hemifield in the contralateral area of V1 (Hoffmann et al. 2003). This arrangement could potentially result in spatial distortions in perception if the cortical cells in the overlap region respond to inputs from both hemifields. But Klemen et al. (2012) demonstrated orderly perceptual representations of space in subjects with albinism. This is evidence that humans with albinism have interleaved maps for the nasal and temporal projections in the overlap region.

The impact of early-onset low vision on neuroplasticity was examined in subject MM, discussed in Section 3. Although surgery restored visual perception across MM’s visual field, fMRI revealed an absence of cortical response to visual stimuli in the posterior region of the occipital pole (Levin et al. 2010). This is the cortical region, which contains a large representation of foveal vision in normally sighted subjects. The authors speculated that MM’s V1 abnormalities resulted from the lack of small neuronal receptive fields responsible for coding high spatial-frequency information. It is possible that these cells were not fully mature when MM lost his vision at age three or that they were particularly vulnerable to the lack of high spatial-frequency visual input.

Another case study involved subject S who had normal vision until age six when a permanent corneal disease reduced his acuity to 20/1000 and his contrast sensitivity to about 10% of normal. By age six, it is likely that his contrast sensitivity and acuity had attained adult levels. In an fMRI study at age 56 years, S was tested with both visual and tactile inputs (Cheung et al. 2009). Similarly to MM, posterior areas of occipital cortex normally responsive to foveal stimuli were unresponsive to visual input, whereas more anterior areas were activated by visual input. Additional tests showed that the foveal projection area was responsive to tactile input. The authors interpreted their findings as indicating that cortical neurons with small receptive fields for encoding high spatial frequencies had been redeployed for encoding tactile features, while neurons with large receptive fields, tuned to coarse visual details, remained responsive to visual input. S was a proficient Braille reader, but it is unclear what role, if any, Braille reading played in the responsiveness of the foveal projection area to tactile stimuli.

Although several studies have revealed responses in the “visual cortex” of blind subjects to tactile and auditory stimuli (Sadato et al. 1996, Merabet et al. 2009), the Cheung et al. (2009) study was one of the first to reveal the potential of the visual cortex for cross-modal sharing of function in low vision. Cunningham et al. (2015) have recently demonstrated a correlation between the extent and amplitude of tactile responses in V1 and the extent of field loss and acuity reduction in a group of subjects with late-onset retinitis pigmentosa (a disorder that typically results in peripheral field loss).
In another recent case study, Dormal et al. (2015) made behavioral and brain-imaging measurements of subject KL both before and after sight-restoring surgery. KL had a history of low vision from early infancy. At the age of 48 years, she received a Boston keratoprosthesis (a type of artificial cornea). Prior to surgery her acuity was 20/500. fMRI revealed both auditory and visual responses in the primary visual cortex. Analogous to the cross-modal tactile responses in S’s visual cortex, the amplitude of KL’s auditory response was greater in the posterior compared with the anterior calcarine sulcus. Unlike S, however, the cross-modal auditory response overlapped with the visual response in the primary visual cortex. Following the surgery, KL’s acuity increased dramatically, reaching 20/30 after seven months, but the auditory response in the primary visual cortex remained elevated. KL also exhibited auditory responses in higher-level visual areas prior to surgery, but auditory responsiveness in the higher-level areas declined following the surgery.

These case studies may indicate that the visual cortex, if deprived of high-resolution input during early life, can repurpose itself for tactile or auditory processing. It is not known how this cross-modal functioning affects the potential for visual rehabilitation. If tactile or other nonvisual use excludes visual functioning and is irreversible, sight-restoring surgery or the deployment of retinal implants may be limited in the capacity for vision recovery.

Most low vision has its onset after vision has fully matured. fMRI has been used to study retinotopic organization in adults with peripheral-field loss including glaucoma (Duncan et al. 2007) and retinitis pigmentosa (Cunningham et al. 2011). fMRI has also been used to study the effects of central field loss. Macular degeneration is the most common cause of central field loss. The primary visual cortex contains a retinotopic map in which the central field is highly magnified relative to peripheral vision. A macular scotoma of 15° would mean that about 50% of V1 does not receive visual input. The region of the visual cortex associated with a retinal scotoma is termed the lesion projection zone or LPZ. Tests of possible reorganization (sometimes termed remapping) of the visual cortex in macular degeneration have centered on the question of whether the LPZ responds to visual stimuli presented to peripheral vision outside the scotoma. Some studies have reported substantial responses in the LPZ (Baker et al. 2005, 2008; Schumacher et al. 2008). Other studies have found no evidence for remapping (Sunness et al. 2004, Baseler et al. 2011) or incomplete remapping (Liu et al. 2010). In the most extensive study, Baseler et al. (2011) used fMRI to study 16 subjects with macular degeneration and 12 age-matched controls. They found no evidence that stimuli presented in peripheral vision outside the scotoma activated the LPZ. Their study used passive stimulation not requiring a behavioral response. There is evidence, however, that cortical responses in the LPZ can be elicited in active visual tasks, such as picture recognition, which might implicate top-down feedback from higher visual centers normally masked by stronger stimulus-driven activation (Masuda et al. 2008, Liu et al. 2010).

If the LPZ in most adults with acquired central field loss remains unresponsive to visual stimuli, implying a lack of remapping of the peripheral field into the LPZ, it is possible, but not yet demonstrated, that the cortical circuitry for vision remains intact and capable of resuming normal visual processing. This would be advantageous if retinal implants, optogenetic treatments, or other remedies could reinstate visual signaling along the retino-cortical pathway. This possibility is consistent with the discussion in Section 4 concerning the successful prognosis for cataract surgery. It remains to be understood how the age of onset of eye disease, its severity, and its duration interact in determining when the visual cortex reorganizes in some functionally irreversible way or retains its capacity for normal visual functioning.

6. OCULOMOTOR AND PERCEPTUAL PLASTICITY

People with central field loss often rely on a region outside the scotoma as a reference location for seeing—the preferred retinal locus (PRL). To qualify as a stable PRL, eye movements should
consistently bring the object of interest to this region instead of to the fovea—a form of plasticity termed oculomotor re-referencing. Adopting a set of stringent criteria to define oculomotor re-referencing, including the complete lack of foveating saccades, White & Bedell (1990) reported that only 7 of their 21 subjects with long-standing macular disorders demonstrated evidence of complete oculomotor re-referencing, casting doubt on plasticity of the oculomotor system in this context. However, by comparing whether microsaccades of fixational eye movements were directed to the fovea or the PRL, Chung (2013a) reported a re-referencing index (on the basis of landing errors of microsaccades with respect to the fovea and those with respect to the PRL, as well as the distance between the fovea and the PRL) of 82% for a group of 23 eyes with central field loss. This finding implies a high degree of oculomotor re-referencing.

Because people with central scotomas must use their peripheral vision for seeing, the normal periphery has been used as a model to study their visual behavior. Emerging evidence suggests that some properties of the PRL more closely resemble those of the fovea than that of the normal periphery. For instance, the recognition of an object in peripheral vision is more difficult when it is flanked by nearby stimuli—the crowding effect (see Levi 2008 for a review). To alleviate the crowding effect, neighboring objects need to be separated by critical spacing from one another. Toet & Levi (1992) showed that the two-dimensional shape of the crowding zone (critical spacing measured along different meridians with respect to an object of interest) is typically circular at the normal fovea but elliptical in the normal periphery, with the ellipses elongated along the radial direction toward the fovea. Chung (2013b) showed that the crowding zones at the PRL of individuals with macular disorders were less elliptical than expected at the same eccentricity in the normal periphery. The smaller than expected extent of spatial interaction might explain why people with macular disorders did not benefit from increased line spacing in a reading task, contrary to observations in the normal periphery (Chung et al. 2008, Calabrèse et al. 2010). Note that not all visual functions measured at the PRL are better than the expected values based on the same eccentricity in the normal periphery. For example, Chung (2013b) found that acuities measured at the PRLs of subjects with macular degeneration were worse than the acuities at corresponding locations in the normal periphery. Acuities might be poorer because most PRLs are located at the edge of a scotoma where the retina may not be healthy. Moreover, it is generally believed that acuity is limited by cell densities (cones, ganglion cells) that are not amenable to plasticity in adulthood.

The adoption of the PRL as an oculomotor reference location is strong evidence for visuomotor plasticity. The question of how the PRL develops is intriguing to the clinical community because of its relevance to rehabilitation and to the scientific community because of its relevance to plasticity (Cheung & Legge 2005). Crossland et al. (2005) tracked the natural development of the PRL in 25 individuals with recent-onset bilateral macular diseases and found that all of them developed a PRL within six months (no interventions were provided). In contrast to Crossland et al. (2005), in a study by Kwon et al. (2013), rapid development of PRL-like behavior was observed in normally sighted subjects viewing with an artificial central scotoma. These subjects were trained for several hours in a search task and spontaneously learned to bring targets of interest to a specific location near the boundary of the artificial scotoma. This type of oculomotor learning occurred even when training with the artificial scotoma was interspersed with long periods of normal foveal vision (Walsh & Liu 2014). It remains to be determined how the rapid oculomotor learning observed with artificial scotomas in the Kwon et al. study relates to the development of PRLs over a longer time course in people with naturally occurring central scotomas.

PRLs are usually located near the boundary of a scotoma, but what factors determine where on the boundary? A function-based hypothesis predicts that the location of a PRL should be optimized for a specific task. For reading, a PRL left or right of the scotoma would mean that text letters on the attended line would disappear into the scotoma, so placement of the PRL above or below the scotoma would be advantageous. But 34–60% of patients adopt a PRL left of their scotoma...
(Guez et al. 1993, Fletcher & Schuchard 1997, Cacho et al. 2007). Some others have reported that the majority of patients with macular disease, especially those with Stargardt disease, had a PRL below their scotoma (White & Bedell 1990, Reinhard et al. 2007, Greenstein et al. 2008). It has also been proposed that the PRL location is task or stimulus dependent (Lei & Schuchard 1997) and that subjects may use more than one PRL. For instance, the PRL for fixation may differ from that for reading (Crossland et al. 2011).

Alternatively, the location of a PRL may be chosen to optimize acuity or some other general visual capacity. However, by measuring acuities at various locations around a scotoma, Chung & Bernard (2013) found that the PRL does not occur at the location with the best acuity.

An important implication of the oculomotor system’s plasticity is that patients can benefit from training. Indeed, eccentric viewing is a standard rehabilitation technique for patients with central scotomas. Eye movement training has also long been recognized as beneficial for low-vision patients (Freeman & Jose 1997). Seiple et al. (2011) compared the effectiveness of eccentric viewing training, eye movement training, and RSVP reading training, and they reported that eye movement training led to the highest improvement in reading speed. A goal of eye movement training is to improve fixation stability. There is a positive correlation between fixation stability and acuity (Tarita-Nistor et al. 2008) and between fixation stability and reading speed (Rubin & Feely 2009), leading to the belief that stable fixation is desirable. However, to date, the functional role and impact of fixational eye movements are still largely unknown. It is debated whether the greater fixation instability in low-vision patients is detrimental by causing poor visual performance or advantageous in sustaining vision by preventing image fading (Déruez et al. 2004).

As discussed in Section 4, the reading performance of people with central field loss from macular degeneration can benefit from perceptual learning (Chung 2011, Nguyen et al. 2011, Tarita-Nistor et al. 2014). Attempts at improving the effectiveness of the standard RSVP training method are currently underway, including training to read vertical text if the PRL is located left or right of the scotoma (Subramanian et al. 2014) and by aligning words with respect to their optimal viewing position (Astle et al. 2015).

Another approach for helping individuals with field loss, relying on visual plasticity, is remapping stimuli from the nonseeing areas to the seeing areas. These techniques are effective only if subjects can learn to integrate distorted and/or displaced visual input with direct visual input. Using an artificial central scotoma, Wensveen et al. (1995) investigated the benefit of remapping print obscured by the scotoma to reappear at the scotoma margin. The resulting print appeared highly distorted and may account for the modest improvement in reading speed. For peripheral field loss, prisms have been used to shift images from the nonseeing area to the seeing area. A complication is that patients need to learn to distinguish which image is in the veridical position and which one is remapped from the nonseeing area, sometimes leading to confusion. Spatial multiplexing (Peli 2001) is a method of remapping the unseen peripheral field as a superimposed representation within the functioning central field. This method, similar to augmented reality, requires the subject to wear a head-mounted display with a camera to capture wide-field images. Given the challenges of these remapping methods, training patients with peripheral field loss (restricted field or hemianopia) to make more scanning eye or head movements toward the nonseeing area continues to be the most common rehabilitation strategy (Trauzettel-Klosinski 2011).

7. VISION RESTORATION TECHNOLOGIES AND SENSORY SUBSTITUTION DEVICES

The most prevalent causes of low vision are retinal diseases. Promising technologies are being explored to restore, replace, or bypass damaged retinal tissue to provide visual input for perception.
These approaches are discussed in recently published special issues of the journals *Vision Research* (Fine et al. 2015) and *Translational Vision Science and Technology* (Lasker 2014). The technologies include implanted visual prostheses, gene therapy, optogenetics, photopharmacology, stem-cell therapy, endogenous neuron regeneration, and neuroprotection. Relevant to all of these technologies is the emerging research on retinal remodeling, the morphological and neuronal changes in the inner retina, which may occur following photoreceptor damage (Strettoi 2015). We restrict the discussion here to prosthetics, the most highly developed of these technologies.

Brindley & Lewin (1968) first demonstrated the plausibility of a visual prosthesis. They implanted an array of 80 electrodes intracranially to stimulate the visual cortex of a volunteer who was blind from advanced glaucoma. They showed that direct stimulation of neurons in her visual cortex resulted in percepts of light (phosphenes) having topographic regularity in the visual field. Most recent implants have targeted sites earlier in the visual pathway, which require less invasive surgery and take advantage of preserved neural processing beyond the implant. Two types of retinal implants are currently in clinical trials—subretinal and epiretinal. The Alpha IMS is an example of a subretinal implant (Stingl et al. 2015). It uses an array of light-sensitive microphotodiodes, coupled with stimulators, subretinally implanted adjacent to the retinal pigment epithelium. The goal is to replace damaged photoreceptors with artificial stimulation of bipolar cells. The advantages of the subretinal design are that no external camera is required for image capture and the patient can use natural eye movements to look around. However, the implant surgery is difficult, and the longevity of the implants is uncertain. The Argus II is an example of an epiretinal implant (Ho et al. 2015). It uses an array of 60 stimulating electrodes placed on the vitreous side of the retina. Retinal ganglion cells are targeted for stimulation, thereby bypassing degeneration earlier in the retinal network. A camera, mounted on spectacle frames, captures an image, which is processed and then transmitted to the electrode array. Viewing direction is determined by head orientation. The epiretinal surgery is more straightforward, and patients have retained functioning implants over a period of three or more years.

The major issues concerning prosthetic implants and the other vision technologies are the functional values of the restored vision they provide. To date, results from gene therapy and prosthetic implants (the most advanced of the technologies) have yielded very low vision—termed ultra low vision because it is hard to measure with standard clinical tests. Stingl et al. (2015) reported on 29 subjects with end-stage retinal degeneration treated with the Alpha IMS subretinal implant. Of these subjects, 25 achieved light perception and localization of light sources. Thirteen reported useful daily-life experiences, including vision of “picture frame on the wall, fluorescent tubes, kitchen objects, plates in a good contrast, bottles, cup handle, washbasin, and bottles on shelves” (Stingl et al. 2015, p. 154). Only four of these subjects had measurable acuities (orientation of Landolt rings) of 20/2000, 20/2000, 20/606, and 20/546.

For purposes of rehabilitation, tactile and auditory sensory substitution devices are being developed as alternatives to vision technologies. These devices do not require surgery, are less expensive, and can be used on an elective basis, but they require substantial practice to be useful. BrainPort is a tactile-imaging device. It transforms a camera video image to a tactile image rendered with an array of 20 × 20 electrotactile stimulators felt by the tongue. Subject testing has been conducted with the BrainPort (Nau et al. 2013), using tests similar to those used with vision prosthetics. Subjects with light perception or worse, trained for 15 hours, could locate the direction of light sources, determine the orientation of low spatial-frequency grating patterns, and determine the orientation of very large tumbling E’s (Nau et al. 2013).

vOICe ([http://www.seeingwithsound.com](http://www.seeingwithsound.com)) is a software application for transforming two-dimensional grayscale camera video images into a stereo soundscape. The image, sampled at a default resolution of 176 × 64, is mapped into sound using binaural direction, pitch, and loudness.
Some practiced users are capable of impressive feats of object and scene recognition. Striem-Amit et al. (2012) measured the acuity of nine early blind subjects who had extensive training on vOICe (averaging 73 hours over several months). Acuities, measured as the orientation of an isolated E target in a 66° field, ranged from 20/200 to 20/600.

Caution may be in order in assessing acuities for isolated optotypes in the ultra low–vision context. Performance depends on complex interactions between the field of view sampled by the technology, the number and distribution of imaged samples, the user’s control over zooming and scanning, and the extent of the user’s training.

Echolocation can be used to obtain information about objects in space. It involves no surgery or equipment cost but requires substantial practice. Human echolocators emit sounds (tongue clicks, claps, cane taps) and gather information about the properties and layout of objects in space from the returning echoes. For a review, see Kolarik et al. (2014). Training in echolocation as rehabilitation for blind people is available through World Access for the Blind. Teng et al. (2012) measured an analog of vernier acuity in six blind highly trained echolocators. The threshold horizontal offsets for the best three subjects ranged from 1.2° to 1.9°.

There is evidence that the ventral visual cortex is activated in trained blind subjects by tactile stimulation with a tongue display unit similar to the BrainPort (Ptito et al. 2012), by image soundscapes from vOICe (Merabet et al. 2009, Striem-Amit & Amedi 2014), and in echolocation (Thaler et al. 2011, Arnott et al. 2013). The functional roles of these activations are yet to be determined. Perhaps cross-modal recruitment of visual areas in blind subjects enables more efficient or accurate processing of the spatial content of the tactile or auditory images. The activity in visual cortex may also be playing a role in the construction of a multisensory spatial representation of objects in space.

It has also been proposed that the responses of the visual cortex in blind subjects to tactile and auditory images are accompanied by visual qualia, that is, subjective experiences equivalent to conscious visual perception. According to the Law of Specific Nerve Energies, proposed by Johannes Müller in the nineteenth century, the attributes of the sensory qualia are determined by the nervous pathway activated, or in more modern terms, by the area of the brain activated. Conscious percepts arising from activity in visual cortex would qualify as visual perception. If so, we may then wish to expand our exploration of low vision to include spatial images arising from retinal implants, tactile displays, audio soundscapes, and echolocation, which appear to be processed along the visual pathway.

8. CONCLUDING REMARKS

We have discussed behavioral and neural indicators of plasticity in low vision and considered their implications for rehabilitation. New research methodologies, including retinal imaging and non-invasive brain imaging, have supplemented traditional clinical tests of acuity, contrast sensitivity, and field, as well as psychophysical methods for probing plasticity. We now return to the four questions raised in the Introduction.

8.1. What Are the Perceptual and Physiological Indicators of Plasticity in Low Vision?

Plastic changes due to low vision are observed in behavioral, oculomotor, and brain-imaging studies. There is evidence that the retinotopic organization of V1 can be disrupted in early-onset low vision. Case studies provide evidence that V1 cross-modal changes occur in low vision as well as in blind subjects. This may include sharing of the occipital cortex by vision and other sensory
modalities (tactile, auditory). This cross-modal sharing may also extend to extrastriate cortical sites along the visual pathway involved with object recognition and motion processing.

Behavioral data from Project Prakash and the extensive eye-movement and behavioral studies of PRL formation in macular degeneration indicate that adult visual function can improve following the onset of low vision. Enhancements are observed in contrast sensitivity, form recognition, eye-movement control, and reading.

8.2. What Roles Do Visual Experience and Age Play in Shaping These Changes?

For the most part, genetic abnormalities in the visual pathway, such as those found in albinism, appear to be irreversible, although the possibility of treating inherited disorders such as Leber’s congenital amaurosis (a form of retinitis pigmentosa) with gene therapy is being studied (Bainbridge et al. 2008, Maguire et al. 2008, Jacobson et al. 2015).

A wealth of evidence indicates that when long-term low vision begins in childhood, whether it is congenital or begins later, subsequent sight-restoring surgery falls well short of yielding normal adult vision. Nevertheless, early long-term deprivation may be followed by some level of recovery of visual function in adulthood. It remains to be determined how much recovery is possible, the conditions promoting recovery, and the factors that put the brakes on greater recovery.

Late-onset low vision may result in less plastic reorganization of vision and greater potential for restored vision, as in the many cases of good outcomes following surgery for age-related cataracts. Similarly, the evidence for limited retinotopic reorganization in AMD may indicate less plasticity in occipital function in late-onset low vision. But evidence for PRL development in AMD indicates that behavioral and oculomotor adaptations can occur even in cases of age-related low vision.

8.3. Are the Changes Spontaneous, or Do They Depend on Training or Practice?

It is likely that changes in cortical functioning, such as the cross-modal sharing of predominately visual areas with tactile or auditory processing, result from the long-term modification of sensory inputs associated with low vision. These changes appear to occur without explicit training.

Similarly, the adoption of a PRL in macular degeneration often occurs spontaneously, although the process is sometimes facilitated through training of eccentric viewing by a rehabilitation specialist.

Specific skills, such as reading with peripheral vision in cases of central field loss, can be enhanced by explicit training, sometimes referred to as perceptual learning. It remains unclear how specific this training needs to be and what visual characteristics are most conducive to improvement.

Perceptual learning and changes in perceptual response following periods of modified visual input are forms of visual plasticity present in normal adult vision and also in adult-onset low vision. Perceptual learning may play a useful role in low-vision rehabilitation.

As discussed in Section 7, there is evidence that visual information can be recoded using auditory or tactile forms of stimulation, potentially taking advantage of processing in visual cortex. It appears that successful use of these sensory substitutions for vision requires extensive training and practice.

8.4. Are the Plastic Changes Relevant to Rehabilitation?

It is important for eye care clinicians, rehabilitation and educational specialists, and patients to understand the nature and potential for changes in visual functioning following the onset of low
vision. It is also important to understand the likely benefits of potential sight-restoring surgery or therapies.

Finding quantitative metrics for assessing the impact of plasticity on rehabilitation remains elusive. The recent development of visual-function questionnaires evaluating visual abilities may prove useful in forging such links (Goldstein et al. 2015).

Visual plasticity has a dual nature when it comes to rehabilitation. Reorganization of visual processing in the presence of low vision, such as cross-modal sharing of visual pathways with tactile or auditory processing, may be of enormous adaptive advantage in the everyday lives of people with low vision. But this reorganization may get in the way of potential sight-restoring therapies or prosthetics later on. By contrast, decreased plasticity, typical of late-onset low vision from cataract or AMD, may leave the visual pathways more intact and receptive to sight-restoring therapies. The duality of plasticity means that the age of onset of low vision and the number of years of vision impairment play important roles in determining the potential functional benefits of retinal implants, gene therapy, optogenetics, and other innovative treatments for vision loss. Consideration of this duality may help specialists and their low-vision patients decide whether to undertake sight-restoring treatment, devote effort to training with a sensory substitution device, or concentrate on more traditional strategies for adapting to low vision.

SUMMARY POINTS

1. Low vision is any type of visual impairment that affects activities of daily living. It can result from eye disease, eye injury, or genetic abnormalities of the visual pathway.
2. In the context of low vision, plasticity refers to adaptations of visual processing following the onset of eye pathology, not directly due to the underlying disorder. Plasticity may be observed as changes in perception, oculomotor behavior, or neural processing.
3. Modern methods of retinal imaging, brain imaging, and eye tracking have supplemented traditional clinical measures of acuity, contrast sensitivity, and field status for identifying and tracking changes over time in low vision.
4. Complete recovery of normal adult visual function does not seem possible after a prolonged period of vision impairment beginning in the childhood years of visual development. In such cases, the retinotopic organization of the early visual cortex may be modified, including sharing of cortical resources with auditory or tactile processing.
5. Once vision has matured to adult levels, the organization of the visual cortex appears to remain quite stable, despite prolonged periods of vision impairment. This stability is advantageous in cases where pathology early in the visual pathway can be corrected, as in cataract extraction. The stability, however, might stand in the way of functionally beneficial cortical reorganization in late-onset low vision.
6. Although the organization of the visual cortex remains stable in adulthood, the visual system has some capacity for adapting to its inputs. Recent studies of perceptual learning and oculomotor behavior reveal that some improvements in visual functions are possible in cases of adult-onset low vision. The boundaries of these improvements are yet to be determined.
7. Vision restoration is being attempted with vision prosthetics, but as yet, prosthetics provide only very low levels of visual function. Sensory substitution devices recode visual input in tactile or auditory formats and require extensive practice for useful operation.
FUTURE ISSUES

1. What biological constraints reduce plasticity of the adult visual brain? What limits do these constraints impose on improved perceptual or oculomotor function following the onset of low vision in adulthood?

2. Can methods be found to release the brakes on plasticity for people with adult onset of low vision? If so, what benefits would result for rehabilitation?

3. What are the capabilities of the normal adult visual system for perceptual learning and for adapting to modified input? Can these capabilities be leveraged for enhanced low-vision rehabilitation?

4. What are the functional benefits, if any, of shared processing of auditory and tactile inputs by visual pathways in low vision? What are the implications for low-vision rehabilitation?

5. How do the age-related differences in plasticity of the visual pathways affect the potential benefits of visual prostheses and sensory substitution technologies?

6. We need better methods for translating laboratory measures of low-vision plasticity into improved rehabilitation strategies.

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Errata

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