CHAPTER 8

Visual Systems

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The child's visual perception is limited by immaturities in the nervous system beginning in the retina and continuing through the primary visual cortex to extrastriate visual cortex and beyond (see Fig. 8.1). At any given age during development, there are differential effects of these immaturities on different aspects of visual perception. Because the elimination of these constraints does not progress on a single developmental trajectory, it is never the case that the child's visual perception overall is a fraction of that of an adult (e.g., never half as good overall). Most—but not all—of these developmental trajectories depend on visual experience, that is, visual input is critical for tuning the neural underpinnings of most aspects of visual perception. When the input is missing, even for a brief period of time, the refinement fails to occur.

For this chapter, we have chosen visual capabilities that illustrate these points. We will begin by describing retinal limitations and their effect on the development of visual acuity. We will continue by describing limitations in the primary visual cortex, illustrated by their impact on the control of eye movements despite sufficient function to mediate orientation perception. The next section concerns limitations in both the dorsal and ventral streams of the visual cortex, illustrated by perceptual capabilities that require integration of local signals processed in the primary visual cortex by higher level visual areas to yield a perception of global motion and global form, respectively. Throughout, we summarize the evidence on neural limitations and relate it to developmental changes in visual capabilities. We also consider how a period of visual deprivation from bilateral congenital cataracts affects subsequent development. The cataracts blocked all patterned visual input during infancy until they were removed surgically and the eyes fitted with compensatory optical correction, usually contact lenses. Despite treatment early in infancy, these patients had later deficits in many aspects of vision. These "sleeper effects" indicate that early visual input—at a time when the visual nervous system is very immature—sets up the neural architecture for later refinement. In its absence, those later improvements fail to occur. In the last section, we consider general principles that emerge about brain development and visual perception.

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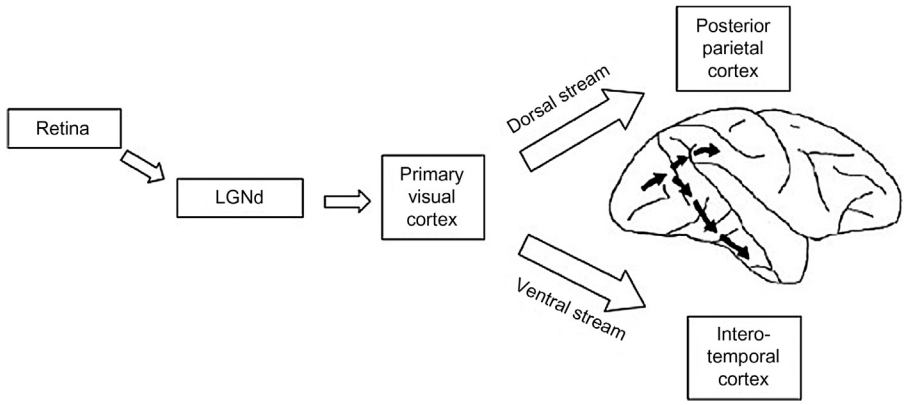

Figure 8.1 Simplified cartoon of the flow of visual information in the human adult brain from the retina to the dorsal lateral geniculate nucleus (LGNd) to the primary visual cortex and on to the extrastriate visual cortex where it divides into the dorsal visual stream projecting to the posterior parietal cortex and the ventral visual stream projecting to the infero-temporal cortex.

8.1 RETINAL LIMITATIONS

The retina of the human newborn is very immature, especially the central fovea, the portion of the retina that mediates the perception of fine detail. In the human adult, the central fovea forms a pit containing only densely packed cones. The outer segments are the same width as the inner segments (1.9 μm where 1 μm = 0.001 mm) resulting in very efficient transmission of light from the inner to the outer segment. The newborn, in contrast, has only the very beginnings of a foveal pit. Cones in the central retina are intermingled with, and covered by, outer ganglion cells (Yuodelis & Hendrickson, 1986). Moreover, cones in the central retina are much wider and shorter than those of the adult. Specifically, between birth and adulthood, the width of a foveal cone inner segment narrows on average from 6.5 to 1.9 μm, the length of the inner segment grows on average from 8.7 to 32 μm, and the length of outer segment grows on average from 3.1 to 45 μm (Yuodelis & Hendrickson, 1986). Moreover, in the central retina, the inner segments are more than five times wider than the outer segments at birth, but equivalent in width when mature. Together, these factors result in the newborn's central retina being 350 times less efficient than that of the adult at transmitting light (Banks & Bennett, 1988). The transition from the neonatal to the adult retina is gradual. Even at 45 months of age, immaturities in the central retina are evident: the outer segments of cones are only half as long as those of adults, the foveal pit still contains several layers of pale glia, and packing density is three-quarters of that in adults (Yuodelis & Hendrickson, 1986).

A very immature central retina at birth would be expected to coincide with very immature visual acuity. In fact, that is the case. Acuity in infants has been measured

Figure 8.2 An acuity card with wide stripes typical of those used with the Acuity Card Procedure. Each card contains a patch of black and white stripes on a plain gray background of equal mean luminance. The stripes are placed to the right or left of a central peephole through which a tester watches the baby. The stripes become narrower and narrower on subsequent cards.

typically with the Acuity Card Procedure (McDonald et al., 1985; Teller, McDonald, Preston, Serbis, & Dobson, 1986). The infant is shown a card with wide black and white stripes on one side (to the right or left of center) against a plain gray background of equal mean luminance similar to that shown in Fig. 8.2. Because infants have a looking preference for something patterned over something gray (Fantz, Ordy, & Udelf, 1962), they will look to the side with the stripes if they can see them. Once a tester decides that the baby can see the stripes, because the baby looks at them or smiles, or gives any consistent indication, subsequent cards are shown with stripes that get narrower and narrower, sometimes to the right and sometimes to the left of center. The measure of acuity is the finest stripes for which the infant shows a response that the tester can pick up reliably. This method has been used to show that acuity is about 40 times worse in the newborn infant than in the adult, with an eightfold improvement over the next 6 months, and gradual improvement thereafter, until acuity reaches adult levels after 4 years of age (Mayer et al., 1995). Studies of spatial contrast sensitivity indicate that acuity is fully adult-like around age 7 (Ellemberg, Lewis, Liu, & Maurer, 1999).

Through a series of calculations based on computational modeling, anatomical data, and various assumptions, Banks and Bennett (1986) concluded that retinal immaturities underlie much, but not all, of infants' immature acuity. Another way to test this hypothesis is to test grating acuity developmentally in humans who had a normal retina but a compromised higher visual system. We did just that by testing the grating acuity of children born with dense cataracts in both eyes. These patients were born with cataracts so dense that they had no pattern vision whatsoever until the cataracts were removed during the first year of life by surgically removing the natural lens of the eye and replacing it with a suitable optical correction, typically a contact lens. Anatomical and physiological studies of monkeys that had been comparably deprived (reviewed in Boothe, Dobson, & Teller, 1985), suggest that cataract-reversal patients have an entirely normal retina but a compromised visual cortex (see below for a detailed discussion of cortical abnormalities). If normal grating acuity depends only on a normal retina, cataract-reversal patients should develop normal grating acuity.

To find out, we tested the grating acuity of a group of bilaterally deprived infants within 10 minutes of their receiving their first contact lenses, after 1 hour of waking time, and 1 month later (Maurer, Lewis, Brent, & Levin, 1999). When infants first received their contact lenses after surgical removal of the cataractous lens, in other words, when they first could see patterns, grating acuity was no better than that of normal newborns despite the fact that the duration of deprivation, and hence the age of the infants at test, ranged from 1 week to 9 months. However, after only 1 hour of being awake, visual acuity improved significantly—as much as the improvement that occurs over the first *month* of life during normal development. To verify that the rapid improvement was caused by the hour of visual experience rather than a practice effect, we carried out a second experiment where we followed the same protocol for the immediate and 1-hour tests except that we patched one of the treated eyes so that, for each patient, one eye received the hour of visual experience while the other eye did not. The improvement in visual acuity was far greater in the experienced eye than in the patched eye, showing that the improvement was caused mainly by the onset of visual experience. Vision continued to improve at a faster than normal rate over the next month and, in a comparable group of cataract-reversal patients, acuity was entirely normal in 85% of patients by 12 months of age (Lewis, Maurer, & Brent, 1995). Thus, it appears that visual experience is necessary to trigger the onset of acuity development. But the visual system is experience-expectant, resulting in a rapid improvement in acuity once the appropriate experience occurs. Moreover, it seems that a normal retina is sufficient to mediate normal visual acuity up to 12 months of age. However, after 1 year of age, the improvement in acuity was slower than normal so that, by 2 years of age, the mean acuity was below normal, and by 3 years of age, 74% of treated eyes had acuity outside the normal range (Lewis et al., 1995). That deficit persists into adulthood (Ellemberg, Lewis, Maurer, Brar, & Brent, 2002). Thus, it is tempting to conclude that a normal retina alone is not sufficient to mediate normal grating acuity after 1 year of age, and the refinements in acuity that occur after 1 year of age depend also on cortical involvement. Indirect support comes from evidence that cataracts with onset after 1 year of age also lead to acuity deficits, with the critical period for damage from visual deprivation not ending until about 10 years of age, several years after adult-like acuity is achieved. That pattern suggests that visual input refines the cortical circuitry underlying acuity from infancy throughout the 7 years of normal development and then consolidates or crystalizes it for several years thereafter.

8.2 LIMITATIONS OF THE PRIMARY VISUAL CORTEX

The next way station where visual perception is limited during development is the primary visual cortex. The primary visual cortex is very immature at birth. Its volume

increases fourfold between birth and 4 months of age and continues to increase gradually until 4 years of age, at which time it is five times that of the newborn. Volume then gradually decreases by about 20% to reach the adult size by 11 years of age (reviewed in Huttenlocher, 1990). Synaptic density also reaches adult values at about 11 years of age by doubling during the first year of life and then decreasing by half over the next 10 years (reviewed in Huttenlocher, 1990). Moreover, neurons of the lateral geniculate nucleus (LGN) that feed into the primary visual cortex are smaller than those of adults until 2 years of age (Brauer, Leuba, Garey, & Winkelmann, 1985; Hickey, 1977), and studies of infant monkeys suggest that they are also less sensitive (Blakemore & Vital-Durand, 1986; Blakemore, 1990; Movshon & Kiorpes, 1993). Thus, it appears that the entire geniculo-striate pathway is very immature, especially during the first year or two of life.

One consequence arising from the immaturity of the visual cortex (and/or its input to subcortical eye movement areas) is the asymmetry of optokinetic nystagmus (OKN). OKN is a series of reflexive eye movements elicited by a repetitive pattern, such as stripes, moving across the visual field. The eyes alternately follow the movement of a stripe and then quickly saccade back to pick up fixation on another stripe. When visually normal adult humans, monkeys, or cats view moving stripes binocularly, OKN is symmetrical: it can be elicited easily when stripes move leftward or rightward. The same is true with monocular viewing: OKN can be elicited easily whether stripes move from the temporal visual field toward the nasal visual field or when they move in the opposite direction (nasally to temporally) (e.g., Braun & Gault, 1969; Lewis, Maurer, Smith, & Haslip, 1992; Pasik & Pasik, 1964). However, cats and monkeys made dependent only on subcortical pathways because of lesions of the primary visual cortex show normal monocular OKN for stripes moving temporally to nasally but no OKN for stripes moving in the opposite direction (Wood, Spear, & Braun, 1973; Zee, Tusa, Herdman, Butler, & Gucer, 1987). OKN to stripes moving nasally to temporally depends on an intact projection from LGN to primary visual cortex and then down to subcortical areas known to be involved in the mediation of OKN, namely the dorsal terminal nucleus of the accessory optic tract and the nucleus of the optic tract in the pretectum (reviewed in Maurer & Lewis, 1993). Measurements of the development of symmetrical OKN tested monocularly provide a window on the functional integrity of this pathway.

OKN eye movements can be elicited in humans even at birth, both when the stripes move from left to right or from right to left, providing that the infant is looking with both eyes open and that the width of stripe is above threshold, that is, for the newborn, quite wide (Atkinson, 1979; Krementizer, Vaughan, Kurtzberg, & Dowling, 1979; van Hof-van Duin, 1978). However, with monocular viewing, OKN is asymmetrical: it can be elicited easily when stripes move from the temporal visual field toward the nasal visual field but not at all when stripes move in the opposite

direction (nasally to temporally) (e.g., Atkinson, 1979; Lewis et al., 1992; van Hof-van Duin, 1978). Not until 3 to 6 months of age does monocular OKN become symmetrical for wide stripes (Atkinson & Braddick, 1981; Atkinson, 1979; Lewis et al., 1992; Naegele & Held, 1982, 1983; Roy, Lachapelle, & Lepore, 1989; van Hof-van Duin & Mohn, 1984, 1985, 1986). However, even at 24 months of age, a small asymmetry exists so that the narrowest stripes eliciting OKN nasally to temporally are a bit wider than those eliciting OKN temporally to nasally (Lewis, Maurer, Chung, Holmes-Shannon, & Van Schaik, 2000). These results suggest that the visual cortex or pathways through it to the pretectum are very immature during early infancy and still not fully mature by 24 months of age. The resulting asymmetrical OKN could be mediated entirely by subcortical pathways from retina to the pretectum that favor OKN to stripes moving temporally to nasally (Hoffmann, 1989; reviewed in Lewis et al., 2000). Alternatively, asymmetrical OKN could be mediated by a cortical pathway that is especially immature at eliciting OKN to temporalward motion (reviewed in Lewis et al., 2000).

Our successful quantification of OKN asymmetry in individual normal infants suggests that this may be a useful tool for evaluating the degree of cortical insult caused by infantile ocular disorders such as cataract. We tested the symmetry of monocular OKN in 51 patients who had been deprived of patterned visual input at some point during childhood because of dense cataracts in one or both eyes (Maurer, Lewis, & Brent, 1989). OKN was asymmetrical in virtually every eye tested of the 23 patients born with cataracts in one eye and of the six patients born with cataracts in both eyes, regardless of the duration of deprivation (ranging from 1.4 to 29 months), regardless of acuity (ranging from 20/20 to light perception), and in unilateral cases, regardless of the patching regime. To determine the sensitive period for developing symmetrical OKN, we also measured the symmetry of OKN in patients born with normal eyes who later were deprived of pattern vision because of a trauma to one eye causing a cataract ($n = 13$) or who developed dense cataracts in both eyes ($n = 9$). We found that OKN was asymmetrical if the cataract occurred before 18−30 months of age, but not if its onset was later. These results suggest that patients who are deprived of patterned visual input beginning any time during the first 1−2 years of life, that is, any time during the normal period of development, suffer from abnormalities in the primary visual cortex and/or in its projection to the pretectum.

Note however that the cortex is functional at least to some extent right from birth. Even newborns can discriminate oblique stripes oriented to the left from those oriented to the right (Atkinson, Hood, Wattam-Bell, Anker, & Tricklebank, 1988; Slater, Morison, & Somers, 1988), and this discrimination is based on orientation cues rather than on differences in local contrast between stimuli (Maurer & Martello, 1980). Studies of monkeys show that the primary visual cortex is the first structure in the geniculostriate pathway capable of mediating such a discrimination

(e.g., Hubel & Wiesel, 1968). By 4 weeks (youngest age tested), infants show changes in visually elicited cortical brain waves (known as visually evoked potentials or VEPs) when the orientation of a striped stimulus changes (Braddick, Birtles, Wattam-Bell, & Atkinson, 2005). However, the refinement of orientation tuning is not complete until middle childhood because, even at 5 years of age, the minimum tilt that can be discriminated from vertical is 4–5 times larger than in adults (Lewis, Kingdon, Ellemberg, & Maurer, 2007).

8.3 LIMITATIONS BEYOND THE PRIMARY VISUAL CORTEX

After information is processed in the primary visual cortex, it proceeds to higher-order visual areas along two streams: a dorsal stream and a ventral stream. The dorsal stream is comprised of middle temporal area (MT/MST complex, also sometimes referred to as V5), V3a, and other inputs to the posterior parietal cortex. The ventral stream is comprised of V3v, V4, and other inputs into the inferior temporal cortex. Although there are many interactions between the two streams, they are broadly specialized for detecting "where" a stimulus is located (dorsal: direction of motion, location, integration with action) and "what" the stimulus is (ventral: the identity of objects and faces). What is common across the two streams is that at higher levels of the visual nervous system the size of receptive fields increases, most neurons receive inputs from both eyes, and, as a result, information is increasingly integrated across time and space. Thus, detailed information processed in small regions of space at the level of the primary visual cortex is integrated to support more global percepts. Here we will illustrate the role of brain development in higher order visual cortex in mediating improvements in the detection of global motion (dorsal stream) and global form (ventral stream).

8.4 DORSAL STREAM LIMITATIONS: EXAMPLE OF GLOBAL MOTION

Sensitivity to local motion depends on neurons in the primary visual cortex that are tuned to direction. These neurons have small receptive fields and each one responds only to information in a small receptive field, often connected to only one eye (Movshon, 1990). Sensitivity to the overall, or global, direction of motion requires additional processing in the dorsal stream involving especially the MT/MST complex (also referred to as V5) where there is a convergence of inputs across receptive fields and eyes onto neurons turned to direction and speed (Hess, Hutchinson, Ledgeway, & Mansouri, 2007; Maunsell & Van Essen, 1983; Newsome & Pare, 1988). Higher areas in the dorsal stream (V3, TOC, LO, V6) also respond selectively to coherent global motion (Biagi, Crespi, Tosetti, & Morrone, 2015). Studies of the monkey indicate that neurons in MT already show directional-selectivity similar to

that seen in adults by 1 week of age (youngest tested) (Movshon, Rust, Kohn, Kiorpes, & Hawken, 2004).

In nonhuman primates, there is converging evidence for the earlier development of MT/MST and other parts of the dorsal stream than of the ventral stream. Thus, in the macaque, local glucose utilization reaches adult levels throughout the dorsal pathway by 3 months of age, at which point the levels throughout the ventral pathway are still quite immature (Distler, Bachevalier, Kennedy, Mishkin, & Ungerleider, 1996). Correspondingly, behavioral studies indicate earlier evidence of the perception of global motion than of global form: when tested at 10–11 weeks of age, monkeys can detect the global direction of both translational and rotational motion but most fail comparable tests for detecting the structure of global form even in the concentric patterns that are easier for adult monkeys to detect than are linear patterns (Kiorpes, Price, Hall-Haro, & Movshon, 2012). Even at 15 weeks, only half of the tested monkeys were able to detect structure in the concentric patterns. Thereafter, there is steady improvement in threshold sensitivity for both global motion and global form, with a greater improvement in sensitivity for motion than form.

Anatomical studies in marmoset monkeys, like those in the macaque, suggest especially early development of MT/MST. Thus, labeling of cortical cells for neurofilament on the day of birth reveals activity in only V1 and an area identified by anatomical landmarks as MT (Bourne & Rosa, 2006). Subsequently, levels increase at the same rate in V1 and MT (or even slightly faster in MT), with gradual emergence, sequentially, in V2, V3, V4, and inferior temporal cortex, that is, the ventral stream. A similar pattern emerged in a study of the calcium binding proteins calbindin and paravalbumin and the neurofilament specifically of pyramidal neurons (Mundinano, Kwan, & Bourne, 2015): even before birth, all three were evident in V1, MT/MST, and DM (a dorsal medial area) but nowhere in the ventral stream. Based on the patterns of postnatal development, the authors speculate that MT and DM drive development of the dorsal stream, whereas the ventral stream's development occurs hierarchically beginning with V1 and continuing, in sequence, V2, V3, V4, and the inferotemporal cortex.

The largely parallel development of V1 and MT/MST in the marmoset summarized in the previous paragraph leads to the obvious hypothesis that, from birth, neurons in MT/MST respond to input from V1 neurons. However, an anatomical tracer study suggests an alternative possibility (Warner, Kwan, & Bourne, 2012): during the first few weeks after birth, the predominant input to MT/MST is via the pulvinar, which itself receives direct input from the retina. With age, the pulvinar inputs recede and the V1 inputs increase in magnitude. These results raise the possibility that early behavioral sensitivity to global motion is mediated by a pathway that bypasses V1.

Human infants do not show any evidence of cortical directional selectivity in VEP signals until about 7 weeks of age, several weeks after orientation selectivity is evident

(Braddick et al., 2005). Initially the selective VEP responses are evident only for elements moving at about 6 deg/s; with age, the effective velocity range extends to slower and faster speeds. Those data are consistent with behavioral evidence that infants fail tests of motion selectivity before about 7 weeks of age. In these studies, infants were given a choice of looking at a form defined by dots moving in a direction opposite the dots forming the background, contrasted with a field of uniformly oscillating dots. Infants show no preference for the side with the motion-defined form until about 7–8 weeks of age (Wattam-Bell, 1996a,b), with the preference first evident for velocities of about 5–10 deg/s and slowly expanding to higher and lower velocities (reviewed in Braddick, Atkinson, & Wattam-Bell, 2003). However, even at 9–12 weeks, infants show no evidence of discriminating between opposite directions of uniform motion (Armstrong, Maurer, Ellemberg, & Lewis, 2011; Wattam-Bell, 1996a).

In contrast, even by 7 weeks of age, infants can integrate motion signals into a global percept. This is surprising since one would have thought that integrating motion signals into a global percept would develop well after the ability to process the direction of motion in a simple stimulus such as stripes. However, the underlying processing for the integration of motion signals may be different in infants and adults. For example, a recent study using functional Magnetic Resonance Imaging (fMRI) of 7-week-old infants looked for differential cortical activation for coherent versus randomly moving dots. There was selective activation in both infants and adults throughout the dorsal stream, including areas identified anatomically as MT and V3, LO (lateral occipital cortex), TOS (transverse occipital sulcus), and V6 (Biagi et al., 2015). However, the low correlation of MT activity with V1 activity in the fMRI data and in separate resting state data suggested a different pattern of organization in the infants that might involve input to MT that bypasses V1, as has been documented in the immature marmoset (see above). Reorganization of the neural underpinnings for the perception of global motion is also suggested by a VEP study of global motion versus global form at 4–5 months of age: infants' responses to the two types of stimuli were distinct (and stronger to global motion than global form—see below) but their topography was different from that in the adult group (Wattam-Bell et al., 2010). From the locations, the authors speculate that global motion might be mediated in MT for infants but in the higher, more medial dorsal areas (V3, V6) in adults.

Little is known about the development after infancy of the cortical networks for the perception of global motion perception, except that individual differences in sensitivity to global motion among children 5–12 years old are correlated positively with the surface area of the parietal lobe and negatively with that of the occipital lobe (Braddick et al., 2016b) and in a complex pattern with measures of signal transmission (fractional anisotropy) of the superior longitudinal fasciculus that connects the parietal lobe to more anterior areas involved in attention and decision making (Braddick et al., 2016a). Behaviorally, sensitivity improves throughout early

childhood, such that older children can detect the global direction of movement with an increasing percentage of randomly moving dots (reviewed in Hadad, Schwartz, Maurer, & Lewis, 2015). When sensitivity is adult-like depends on whether the moving elements have a limited lifetime (appear for only a short time before being replaced by another moving element) to eliminate local motion cues, on the speed at which the elements move, and on their density. With some parameters, children perform as well as adults as early as 3 years of age (Parrish, Giaschi, Boden, & Dougherty, 2005); with others, they continue to improve until about 12 years of age (Hadad, Maurer, & Lewis, 2011).

Patients treated for bilateral congenital cataracts have only small deficits in the perception of local motion, especially at slow velocities (Ellemberg et al., 2005) and can perceive the direction of global motion when most of the elements move in the same direction. However, when coherence is reduced by having a larger percentage of the dots move in random directions, they have difficulty seeing the direction of motion and, even when tested as adults, their sensitivity is roughly five times worse than that of adults with normal eyes (Ellemberg et al., 2002; Hadad, Maurer, & Lewis, 2012). The deficits occur even when the speed is in the range where the patients have minimal deficits in perceiving local motion, even in patients with steady fixation, and even when deprivation ended during the first two months of life, that is, before the onset of sensitivity to global motion in the child with normal eyes. This is an example of a sleeper effect (Maurer, Mondloch, & Lewis, 2007): visual input during early infancy, before a capability is even manifest, is essential for the *later* development of normal function. Presumably the early input establishes, or maintains, the necessary neural architecture, likely involving MT/MST, for later refinement. One possibility is that early input plays a role in the reorganization of cortical inputs to MT/MST seen in the marmoset (Warner et al., 2012) from mainly inputs bypassing V1 (e.g., the pulvinar) to predominantly V1 inputs. That hypothesis is bolstered by evidence that long-term binocular deprivation in cats leads to functional reliance on pathways bypassing V1 (Zablocka & Zernicki, 1996; Zablocka, Zernicki, & Kosmal, 1976, 1980; Zernicki, Zablocka, & Kosmal, 1978).

This deficit in patients treated for bilateral congenital cataract is much larger than their deficit in sensitivity to global form (see below), a pattern supporting the dorsal vulnerability hypothesis described below. Consistent with extrastriate deficits, patients treated for bilateral congenital cataract, unlike controls, fail to show late components of the event related potential (ERP) that differentiate in controls between different kinds of global motion (random, inward radial, outward radial) (Segalowitz, Sternin, Lewis, Dywan, & Maurer, 2017).

Patients treated for bilateral cataracts with postnatal onset between 1 and 10 years of age have acuity deficits (see above) but entirely normal sensitivity to global motion (Ellemberg et al., 2002; see also Fine et al., 2003). That pattern indicates that there are different sensitive periods for damage from visual deprivation for different visual

capabilities, as would be expected from the fact that each depends on a different cortical network. The short critical period for global motion also illustrates that a long developmental trajectory (e.g., global motion *and* acuity) does not necessarily indicate a long critical period for damage (short for global motion; long for acuity).

8.5 VENTRAL STREAM LIMITATIONS: EXAMPLE OF GLOBAL FORM

Sensitivity to global form depends on the integration of information about the orientation of local elements, likely transmitted by V1 neurons. As early as 3 weeks of age (youngest tested), VEP responses of infants respond to changes in the orientation of gratings (Braddick, 1993) and in the same age range, infants show behavioral evidence of discriminating orientation (see above). Throughout the first few months, infants are more likely to show VEP and behavioral responses to a change of orientation than to a change in the direction of moving elements (Armstrong et al., 2011; Braddick et al., 2005). However, the perception of global form requires the integration of information about the orientation of individual parts of a stimulus. Detection of global structure is mediated in the ventral stream, with sensitivity to the patterning of orientation information first evident in V3 and V4 and increased responsiveness in the LOC (lateral occipital complex) in the occipitotemporal cortex (Ostwald, Lam, Li, & Kourtzi, 2008). As summarized above, in non-human primate models, the development of these ventral structures lags behind the development of the dorsal pathway. Consistent with that evidence, at 4–5 months, human infants show reliable cortical VEP responses to changes in the direction of motion (see above), but less reliable, more diffuse responses to changes in global form: the cortical VEP of over 90% of infants showed a response to the reversal of motion direction but only half showed any differential VEP response to concentrically arranged elements versus a random arrangement (Wattam-Bell et al., 2010). Moreover, the infants' response to global form was more central and diffuse than that of adults (Wattam-Bell et al., 2010).

It is difficult to deduce the age at which infants first show behavioral evidence of detecting the structure of global form because their visual preferences and dishabituation when tested with contrasting forms can often be based on local, as opposed to global, properties of the stimulus. For example, newborns' preference for face-like arrangements of pattern elements can be largely, if not wholly, explained by low-level features like visible spatial frequencies in the upper half of the image that are congruent with the shape of the contour and contained in features that are dark on a light background (Cassia, Turati, & Simion, 2004; Cassia, Valenza, Simion, & Leo, 2008; Farroni et al., 2005; Mondloch et al., 1999). In addition, these early preferences may be mediated by the superior colliculus (Morton & Johnson, 1991), which may be more mature at birth than the primary visual cortex. Over the next few months, face detection improves so that by 3 months infants prefer face-like structures even when low-level features cannot explain the preference (Mondloch et al., 1999).

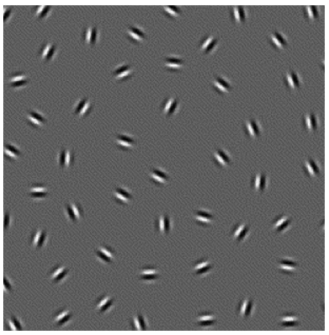

Figure 8.3 Gabor patches arranged as a circle within a background of randomly ordered Gabor patches (after Gerhardstein et al., 2004, *Vision Research*, *44*(26), 2982, Fig. 8.1).

This response to face structure is likely cortically mediated and depends on visual experience during the first three months of life: in infants whose visual experience was shortened by a period of visual deprivation caused by bilateral congenital cataracts, the preference is not evident at 3 months of age and only emerges after several months of "catch-up" visual experience (Mondloch et al., 2013).

As with faces, there is clear evidence of sensitivity to global structure in the arrangement of dots and lines at 3–4 months of age. For example, at 12 weeks, but not 10 weeks, infants dishabituate to a pattern of randomly oriented lines after habituation to a concentric arrangement (Curran, Braddick, Atkinson, & Wattam-Bell, 2000). At this age, infants can also discriminate a completely random pattern of Gabor patches from a stimulus with Gabor patches forming a circle that are arranged on a background of randomly ordered Gabors like that shown in Fig. 8.3 (Gerhardstein, Kovacs, Ditre, & Feher, 2004).

After infancy, sensitivity to global form increases gradually for many years, such that children become increasingly able to detect the form in higher levels of background noise. For example, Gunn et al. (2002) measured the highest level of background noise (randomly oriented line segments) that children can tolerate and still detect a concentric array of line segments. Children's thresholds were higher than those of adults until 6 years of age. Studies using Glass (Glass, 1969) patterns have documented a slightly longer developmental trajectory. Glass patterns use pairs of dots to form concentric or linear patterns within a background of randomly oriented dot pairs (see Fig. 8.4). Thresholds can be calculated by randomly orienting varying percentages of the signal dot pairs. In human adults, these patterns selectively activate area V4 in the ventral stream (Wilkinson et al., 2000). Children 9 years old, but not 6 years old, are as sensitive to the form in Glass patterns as are adults (Lewis et al., 2004). Similarly, between 5 and 13 years of age, children become able to find a form defined by oriented Gabors with increasingly greater background noise (Kovács, Kozma, Fehér, & Benedek, 1999). In addition to changes in V4, the long developmental trajectory may reflect, in part, the slow development of

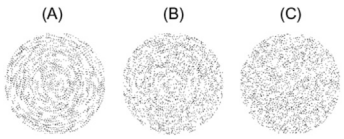

Figure 8.4 Concentric Glass Patterns. (A) 100% signal—every dot has a mate on the radial axis. (B) 50% signal—50% of the dots have a mate on the radial axis and the remaining dots are noise dots that are unpaired and distributed randomly. (C) Noise—there is 0% signal and every dot is distributed randomly.

long-range horizontal connections within V2, a relay station in the ventral stream between V1 and V4 (Burkhalter, Bernardo, & Charles, 1993; Taylor, Hipp, Moser, Dickerson, & Gerhardstein, 2014). Despite the long developmental trajectory for sensitivity to global form, the maturation of sensitivity to global motion takes even longer, at least under some testing conditions (Hadad et al., 2015).

Early binocular deprivation from congenital cataracts prevents the normal development of sensitivity to form in Glass patterns (Lewis et al., 2002). Such patients can distinguish concentric Glass patterns from random control patterns, but their threshold for tolerating randomized dot pairs within the patterns is 1.6 times higher than that of adults with normal vision. This contrasts with a threshold elevation of 4.9 for the perception of global motion (see above). Thus, early visual input is necessary to set up the neural architecture, likely involving V4, for the later integration of local elements into a global form structure, but there is more resiliency in the ventral system for processing global form than in the dorsal system for processing global motion. That conclusion is consistent with recent evidence that, unlike global motion, Glass patterns evoke a N170 (a negative ERP component about 170 msec after the onset of the stimulus) in adults treated for bilateral congenital cataracts. However, unlike controls, the amplitude of the signal is the same for random patterns and patterns in which the dot pairs define a circular pattern (Segalowitz et al., 2017). As with global motion, the effect of early visual deprivation may be to prevent the normal alteration of the balance of inputs to the relevant higher cortical areas (V4 in the case of global form) from extrageniculate pathways to pathways through V1. Studies of binocularly deprived cats provide further evidence that early visual deprivation prevents the normal alteration of the balance of inputs to extrastriate cortex: form perception in binocularly deprived cats, unlike that in visually normal cats, is resistant to lesions of V1 but is eliminated by lesions to the superior colliculus and pretectum (Zablocka et al., 1976, 1980).

8.6 DORSAL STREAM VULNERABILITY

Comparisons of sensitivity to global motion and global form in a variety of special populations have consistently reported that the deficit in global motion is much larger

than the deficit in global form (reviewed in Braddick et al., 2016a). This has led to the "dorsal vulnerability" hypothesis (Braddick et al., 2003), which manifests as especially poor sensitivity to global motion, as well as poor visuo-motor control. In addition to patients treated for bilateral congenital cataract, especially poor sensitivity to global motion has been reported in patients who missed early binocular input because of misaligned eyes (Ho et al., 2005; Simmers, Ledgeway, & Hess, 2005) or who experienced unusual visual experience at birth because of extremely premature birth (e.g., Taylor, Jakobson, Maurer, & Lewis, 2009). In other conditions, the vulnerability is less clearly related to abnormal input, and may be better explained by a genetic abnormality that especially impacts the more slowly developing parts of the brain. The impact is likely greater on the dorsal stream than on the ventral stream because, although the dorsal stream appears functional earlier than the ventral stream (Distler et al., 1996; Kiorpes et al., 2012; Wattam-Bell et al., 2010), its sensitivity increases more during development, at least in monkeys (Kiorpes et al., 2012), and it is adult-like in humans at a later age (cf., Hadad et al., 2011 versus Lewis et al., 2004). The larger changes in development over a longer period as an underlying cause of a dorsal stream vulnerability is consistent with the Detroit Model (Levi, 2005): the last person hired is the first to be fired. Similarly, aspects of vision that are slower to mature might be the most likely to be affected by insult. This analysis might explain the especially poor sensitivity to global motion in children and adults with Williams Syndrome, autism spectrum disorder, developmental dyslexia, Fragile X, and hemiplegia (reviewed in Braddick et al., 2016a; Hadad et al., 2015). Alternatively, it may be the postnatal reorganization of inputs to MT, similar to that documented in the marmoset (Warner et al., 2012), that makes sensitivity to global motion especially vulnerable in humans.

8.7 CONCLUDING REMARKS

A common assumption in the literature is that cortical development is hierarchical. For vision, that means that the development of V1 is primary and that only as its neurons become more refined can input to the next relay stations in V2 and V3 begin to function. Those relay stations, in turn, it is assumed, must mature before there can be any functional input to higher cortical areas in the dorsal and ventral streams, namely MT/MST and V4, respectively. Our summary of brain and behavioral development of vision illustrates the fallacy of this assumption: MT/MST is surprisingly mature at an early age—as mature, or even more mature, than V1. This is evident in electrophysiology in the monkey and human, in neuroanatomy in the marmoset, and in behavioral sensitivity in the infant monkey and human. It is a reminder that there are many inputs to extrastriate cortex, some of which bypass V1, and hence may allow precocious development. It seems plausible that those inputs

play a larger role in early development and after early binocular deprivation than they do in visually normal adults.

A similar caution against a hierarchical approach comes from a comparison of sensitivity to global versus biological motion in adults treated for bilateral congenital cataracts. In adults, a percept of a moving biological entity arises from the pattern of movement of the joints and occurs robustly even when the pattern is contained only in moving dots (Johansson, 1973). A critical structure for this decoding is the posterior region of the superior temporal sulcus, an area that receives its inputs from both the dorsal and ventral streams (Puce & Perrett, 2003). Since cataract-reversal patients have substantial deficits in global motion (dorsal stream) and in global form (ventral stream), from a hierarchical approach, one would expect that there also would be substantial deficits in sensitivity to biological motion. However, adults treated for bilateral congenital cataracts have completely normal sensitivity to biological motion, performing as accurately as controls even when a high percentage of noise dots are superimposed on the biological motion (Hadad et al., 2012). A similar caution comes from evidence that newborns are sensitive to biological motion (Simion, Regolin, & Bulf, 2008), months before they show the first evidence of sensitivity to global motion. This neonatal sensitivity, like the sensitivity after early binocular deprivation, may depend on alternative pathways than those that mediate the perception of biological motion in adults, such as those involving the cerebellum, premotor cortex, kinetic-occipital area, fusiform face area, ventral V3, and amygdala (reviewed in Hadad et al., 2012).

Studies of altered perception after early visual deprivation provide additional lessons. Here we reviewed evidence that deprivation of patterned visual input for as little as the first 2–3 months of life is sufficient to prevent the later development of normal acuity, OKN, and sensitivity to global form and global motion. Thus, in the child with normal eyes, visual experience in early infancy sets up the neural architecture for normal refinement, likely in the geniculostriate pathway and the extrastriate cortex to which it projects. In the absence of that input, there are *sleeper effects*: deficits in abilities that emerge only later, long after treatment. One reason suggested above is that early visual input may be critical for the reorganization of inputs to extrastriate cortex. Another—and not mutually exclusive possibility—is that early visual input is necessary to preserve the occipital cortex for visual responding and that in its absence, responding to other modalities is established, as in the congenitally blind (Ricciardi, Bonino, Pellegrini, & Pietrini, 2014). Support for this possibility comes from recent evidence that the cuneus (V3) responds to variations in auditory signals (moving footsteps and vowel sounds) in adults treated for bilateral congenital cataracts and in the congenitally blind but not in adults with normal eyes (Collignon et al., 2013, 2015). Similarly, in adults treated for bilateral congenital cataracts, adaptation to *auditory* motion elicits an unexpected *visual* motion aftereffect (Guerreiro, Putzar, & Röder, 2016). Processing of audio-visual signals is likewise reduced in ventral visual areas compared to responses

to the visual signal alone (Guerreiro, Putzar, & Röder, 2015), perhaps as a developmental adaptation to noisy V1 signals. These unusual crossmodal remappings may occur to a lesser extent when the early visual deprivation is restricted to one eye, such that input to higher visual cortical areas through the nondeprived eye is able to better preserve the visual pathway for later refinement and/or allow more normal crossmodal reorganization to proceed. This possibility is suggested by evidence of *smaller* deficits in the deprived eye(s) after monocular than after binocular deprivation in sensitivity to global form (Lewis et al., 2002) and to global motion (Ellemberg, 2002).

Another lesson from patients treated for dense cataracts is that there are multiple sensitive periods during which visual deprivation can prevent later normal development (Lewis & Maurer, 2005). Here we illustrated three patterns: a short critical period for damage to global motion (~6 months) despite postnatal onset of sensitivity and a long developmental trajectory in infants with normal eyes; a longer critical period for symmetrical OKN (~30 months), roughly coincident with the period of normal development; and an extremely long critical period for acuity (~10 years), lasting past the age at which acuity is adult-like (~7 years). The lesson is that there are multiple critical periods during which visual experience is necessary for normal development and that one cannot infer their timing by knowing the normal developmental trajectory.

In this chapter we used "marker tasks" to probe specific aspects of developing brain function (e.g., symmetrical OKN for V1 influence on eye movements; global motion for MT; global form for V4). However, studies of the mature brain indicate that these visual capabilities are mediated by an interactive network involving feedforward and feedback connections that are further modulated by attention and previous experience of objects and events. Thus, limitations in childhood and deficits after visual deprivation could arise from immaturities anywhere in the network. Developmental improvements in visual perception could arise not from maturation in the "marker" area but from other parts of the network or even from top-down influences of attention and conceptual knowledge. Nevertheless, the brain–behavioral correlations we described here are supported by converging evidence of direct brain-behavior associations during development from animal models, anatomy, electrophysiology, neuroimaging, and behavior. A full understanding, however, will require a consideration of how those changes fit into a complex brain network.

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