Chapter 3

The First Steps in Seeing

Le bon Dieu est dans le détail.

Attributed to Gustave Flaubert

Others assert that the devil is in the details. No matter who is responsible for the minutiae of reality, science is undoubtedly about details, gadgets, and mechanisms. While I can wax lyrically about consciousness, qualia, and zombies, some basic facts about the brain are essential for understanding how it works. Because the majority of this book is concerned with seeing, I'll start by describing retinal processing and eye movements. Subsequent chapters discuss those aspects of seeing that depend on the cortex. The story that emerges often clashes with the deep-seated intuitions you have about the way you see. perceive.

3.1 The Retina Is a Layered Structure

You see by way of light passing through the cornea and lens of your eye. These act as a camera, in which an inverted image of the scene is focused through the vitreous gel inside the eyeball onto the retina. Light traverses this miniature nervous system before individual photons are absorbed in the photoreceptors at the back of the retina (Figure 3.1). The optical signals are converted into electrical ones that are processed in a complex series of steps by horizontal, bipolar, amacrine, and ganglion cells. One census identified five dozen distinct cell types, each one probably with a distinct function. These large numbers are disconcerting for physicists and mathematicians trained to look for simple, powerful, and universal principles to explain the design and function of the brain. They also serve as a warning that the final count of distinct cellular actors for the forebrain could easily number in the many hundreds.[1]

[1]Mammalian retinae contain more than 50 distinct cell types, each with a different function (DeVries and Baylor, 1997; MacNeil and Masland, 1998; Masland, 2001; and Dacey et al., 2003). I will return to the theme of cell types in Section 4.3.

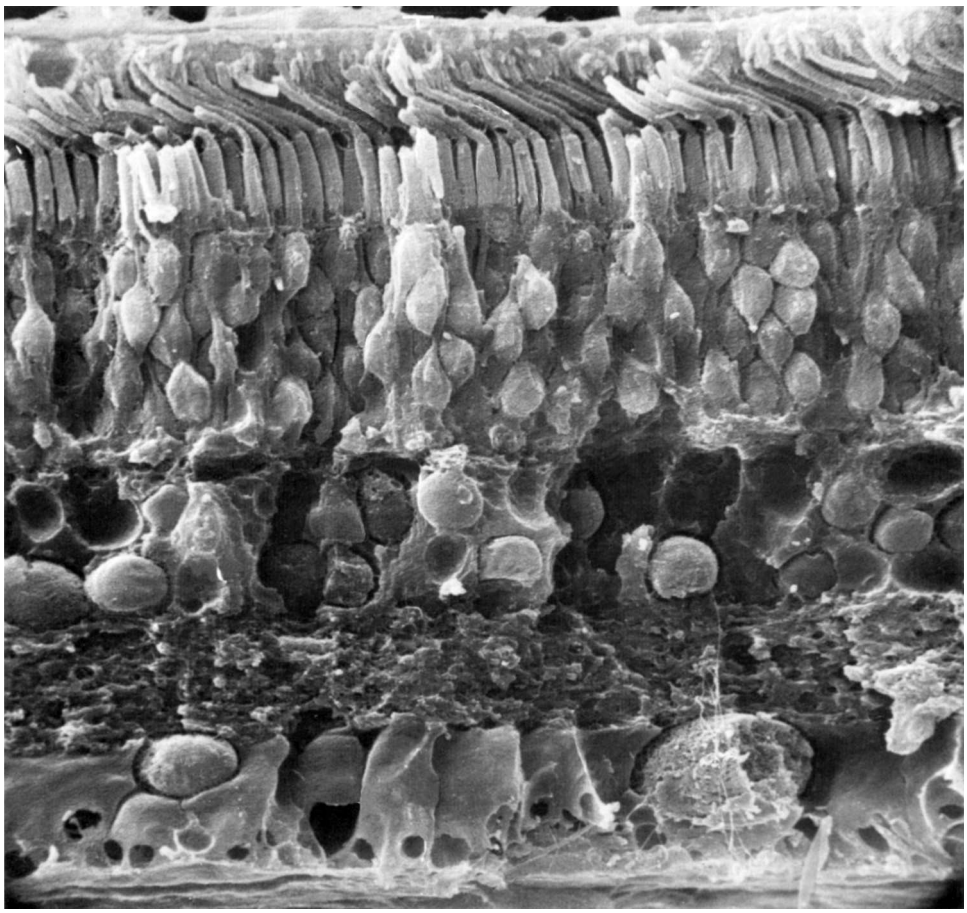

Figure 3.1: A Cross-Section of the Retina
Light arrives from below and passes through the entire retina (of a rabbit) before it gives rise to a photochemical reaction in the photoreceptors (at the top). The visual information, converted into changes in the electrical potential across the membrane, percolates in the reverse direction through the various cellular layers until it gives rise to all-or-none action potentials in ganglion cells (at the bottom). More than one million ganglion cell axons make up the optic nerve along which the spikes travel to more central processing stages. Modified from Kessel and Kardon (1979).

Retinal neurons enhance spatial and temporal contrasts and evaluate color information by evaluating the differential photon catch in distinct photoreceptor populations. The details of this processing are irrelevant to our quest.[2] The sole output of the retina are the 1.5 million axons of the ganglion cells, constituting the *optic nerve*.

[2]For an account of the biophysical and computational processes occurring in the retina, see Dowling (1987), Wandell (1995), and the lovingly illustrated textbook by Rodieck (1998).

Inspecting a state-of-the-art video camera under a microscope reveals millions of identical circuit elements tiling the image plane. Like one of the big new housing developments in the American West, a few basic designs are endlessly replicated. Eyes follow a different design strategy.

Two classes of photoreceptors are spread unevenly across the retina. About one hundred million rods work best under dim light conditions, while five million cones, responding more rapidly than rods, mediate daylight vision. For most day-to-day activities (including reading), the output of the rods is saturated and only the cones provide a reliable signal.

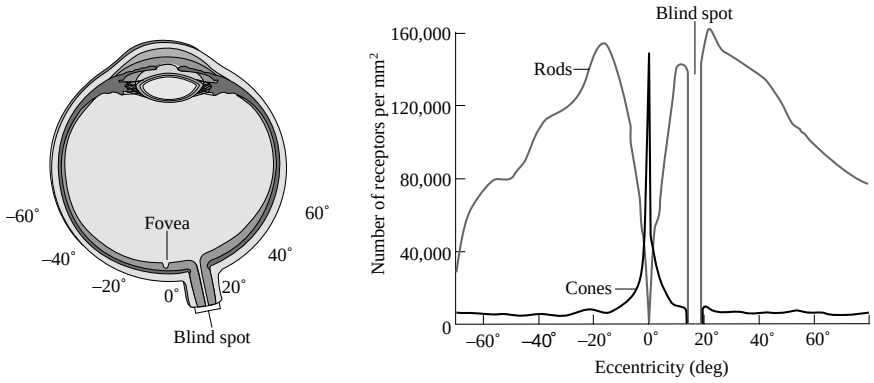

Figure 3.2: Photoreceptors Are Unevenly Distributed
A schematic cross-section of the eye is on the left. The angle relative to the point of sharpest seeing, the fovea, is referred to as *eccentricity*. The density of cones drops off dramatically outside the fovea. Conversely, night vision, mediated by the much more common rods, is optimal some distance away. No receptors sample the incoming brightness at the blind spot. Here, ganglion cell axons form the optic nerve that connects the eye to the brain proper. Modified from Wandell (1995).

The point of highest resolution is found in the central part of the fovea (Figure 3.2). Vision is sharpest here. The effective density of cone receptors drops rapidly with increasing distance from the fovea, to level off beyond 12° of visual angle, or *eccentricity*, away from the fovea. The central one degree of vision is heavily overrepresented by both photoreceptors and ganglion cells at the expense of the rest of the visual field.[3]

Because of the uneven receptor distribution—many in the center and few in the periphery—humans constantly move their eyes to bring the fovea to bear on those portions of the environment that are of interest. This movement permits the retinal neurons to sample that region with the highest possible resolution. Subjectively, the uneven distribution of photoreceptors goes largely unnoticed. Vision appears everywhere sharp and clear—an illusion, but a compelling one. Even a cursory inspection reveals that you can't see all that well "out

[3]The central part of the fovea, about 1° of visual angle in size—the width of your thumb at arm's length is around 1.5 to 2°—is specialized to ensure that vision there is as good as possible.

of the corner of your eyes." Fixate on the central • in the line below and try to identify as many letters as possible without moving your eyes:

txet siht fo tsom • daer t'nac uoy

You won't be able to read more than two or three characters on either side. In order to comfortably see each letter, their size needs to increase as their distance from the central spot increases.

3.2 Color Vision Uses Three Types of Cones

The much-cherished sense of color is a construct of the nervous system, computed by comparing activity in the different cone classes. There are no "red" or "blue" objects in the world. Light sources, such as the sun, emit electromagnetic waves over a broad wavelength spectrum. Surfaces reflect this radiation over a continuous range and the brightness incident to the eyes is continuous as well. Nevertheless, all of us persist in labeling objects as red, blue, violet, purple, magenta, and so on. Color is not a direct physical quantity, as is depth or wavelength, but a synthetic one. Different species have fewer or more cone types, and therefore experience quite different colors for the same objects. For example, some shrimp have eleven cone classes. Their world must be a riot of colors!

Most mammals make do with two types of cone receptors. The exceptions are humans, apes, and old world monkeys, which possess three types. Distinguished by the portion of the spectrum of light to which each is most sensitive, they are referred to as *short-*, *middle-*, and *long-wavelength* cones, or, more succinctly, as S, M, and L cones. Due to the overlap in receptor sensitivity, any one photon can be absorbed by the photopigments in the different receptor types. Collectively, each cone class signals the number of photons it absorbs but nothing explicitly about the spectral composition of the light. In this early stage, color is thus implicitly encoded by three numbers, the relative activation of the three cone types, the basis for Thomas Young's and Hermann von Helmholtz's celebrated *trichromacy theory* of color vision. Now that more is understood about the genetic variation in photopigments in the general human population, trichromacy needs to be enlarged to accommodate the color perception of women with four cone photoreceptor classes and men with only two.[4]

At any one eccentricity, the three classes of cones are not distributed evenly. S cones are absent from the central part of the fovea. Given that this is the point of sharpest seeing, you would think that this deficit would be obvious to anybody who can see. It can't be seen directly, however, so it must be inferred. The way it is done is to ask observers to

[4]The retinae of some women express two forms of the L photopigment that differ by 4 to 7 nm in the long-wavelength portion of the spectrum (Nathans, 1999). Sensitive psychophysical tests have been designed to evaluate the color perception of these "extraordinary" women (Jordan and Mollon, 1993; Jameson, Highnote, and Wasserman, 2001). If the visual cortex learned to process advantageously the additional wavelength information, these *tetrachromat* women would experience subtle hue variations forever unavailable to the rest of humanity. In particular, they should be able to distinguish two colors that look identical to trichromats.

look at a violet annulus with a central hole. As long as subjects are accurately fixating the center of the annulus, thereby placing the hole over the portion of the retina devoid of S cones, the brain assumes that the surrounding violet stimulus extends into the center. As a consequence, a complete disk, rather than an annulus is seen. I mentioned this already in Section 2.1, that inferring missing data based on information from neighboring areas is something the brain engages in all the time.[5]

Even outside the fovea, S cones are much less common than M and L cones. Furthermore, patches of retina dominated by M receptors are intermingled with patches where L cones dominate. This irregular appearance does not show up, however, when looking at evenly colored surfaces, probably because of pervasive filling-in mechanisms that operate throughout the visual field, all part of the great con job called perception.[6]

3.3 A Hole in the Eye: The Blind Spot

At some distance from the fovea, the axons of all ganglion cells are bundled together and exit the eye (Figure 3.2). No photoreceptors are in this area, so there is no direct information about this part of the image, either. This is the *blind spot*.[7]

Normally, input from one eye makes up for the blind spot in the other eye. Yet even if you close one eye, you still won't see a hole in your visual field. A single bad pixel in your home video camera, however, manifests itself as an ugly black spot in the image. What, then, is the difference?

Unlike electronic imaging systems, the brain does not simply neglect the blind spot, it paints in properties at this location using one or more *active processes* such as completion (as in Figure 2.5), interpolation (Section 3.2) and filling-in (Section 2.1). Cortical neurons fill-in on the basis of the—usually sensible—assumption that the visual properties of one patch in the world are similar to those of neighboring locations (in terms of their color, motion, orientation of edges, and so on). Accordingly, if you place a pencil across the blind

[5]The original psychophysical experiments are described in Williams, MacLeod, and Hayhoe (1981) and Williams et al. (1991). Curcio et al. (1991) directly visualizes the distribution of S cones in the human retina.

[6]Local analysis of human retinae (Roorda and Williams, 1999) reveals patches of 5 arcmin extent or more that contain only L or M cones, limiting people's ability to perceive fine color variations.

[7]The blind spot is located around 15° along the horizontal meridian on the nasal side of the retina. Find it by closing the left eye (you won't see much with both eyes closed) and fixating the tip of your left thumb with the right, open eye. Now slowly move the index finger of the right hand, extended at arm's length, from the outside toward the thumb, while keeping your eye glued to the stationary thumb. You will discover that at some point (when the two fingers are somewhere between 15 and 25 cm apart) the tip of the index finger disappears. When the finger is farther out, however, you can still see it. You just discovered that you don't see anything in a patch of about 5° diameter. Remarkably enough, this simple observation, known to most school-aged children today, was not made until the second part of the 17th century by l'Abbé Edme Mariotte in France. He inferred the existence of the blind spot by careful anatomical examination of the retina (Finger, 1994, provides an historical account). The Greeks, Romans, and other ancient civilizations, despite their vast intellectual, artistic, and organizational achievements, failed to appreciate this basic fact of human vision.

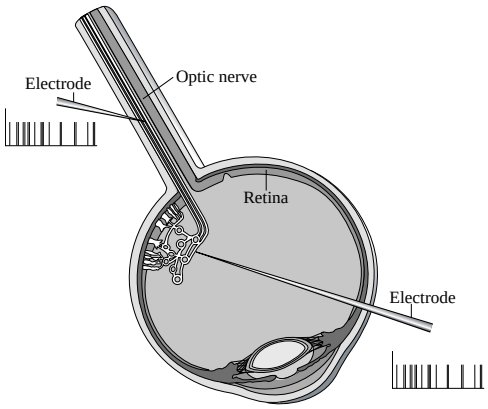

Figure 3.3: Recording the Activity of Ganglion Cells
Action potentials from ganglion cells can be picked up by either placing a microelectrode near their cell bodies in the retina or in the optic nerve outside the eye. Modified from Enroth-Cugell and Robson (1984).

spot, you'll see a single, uninterrupted pencil with no hole in its middle. Neurons above and below the blind spot signal the vertical edge; thus, the neurons responsible for the visual representation of the blind spot assume that the edge was also present at the blind spot, even though it can't be known for sure.[8]

The psychologist Vilayanur Ramachandran at the University of California in San Diego carried out numerous ingenious experiments to study filling-in. As in the foveal experiment just described, he placed a yellow annulus over the blind spot such that the center—devoid of yellow—falls entirely within the blind spot. Observers perceived an intact and complete yellow disk, even though they could vividly see the annulus when looking a little bit off to one side. The brain goes beyond the information given at the retina by making an 'educated' guess about what might be at the blind spot. As no retinal neurons respond to light patterns falling onto the blind spot, the NCC can't be in the retina, since you don't see any holes when looking at the world.[9]

[8]The activity of the V1 neurons that represent the blind spot in the monkey have been recorded. The cells have large binocular receptive fields that extend outside the blind spot and inform the rest of the brain of the presence of large surfaces covering the blind spot (Fiorani et al., 1992; Komatsu and Murakami, 1994; Komatsu, Kinoshita, and Murakami, 2000). For related physiological experiments probing interpolation using artificial blind spots, see Murakami, Komatso, and Kinoshita (1997) and DeWeerd et al. (1995).

[9]Ramachandran and Gregory (1991) and Ramachandran (1992). Kamitani and Shimojo (1999) induced artificial blind spots using transcranial magnetic stimulation. For an exhaustive compendium on filling-in, see Pessoa and DeWeerd (2003). Dennett (1991; see, also Churchland and Ramachandran, 1993) has rightfully emphasized that this does not imply a pixel-by-pixel rendering of the missing information on an internal screen. Instead, active neuronal mechanisms perpetrate the deceit that information is present where none should be visible.

3.4 Receptive Fields: A Key Concept for Vision

The all-or-none spiking activity of ganglion cells, the sole conduit for information leaving the retina, is relatively easy to detect with microelectrodes (Figure 3.3). Pioneered by Stephen Kuffler, at the time working at Johns Hopkins University in Baltimore, such experiments led to a refinement of the concept of the *receptive field*, introduced by Keffer Hartline at the Rockefeller University during his investigations of the visual system of the horseshoe crab, *Limulus*. Operationally, the receptive field of a neuron is defined as the region in the visual field in which an appropriate stimulus, here a flashing spot of light, modulates the cell's response.[10]

Electrophysiologists frequently connect the amplified output of their electrode to a loudspeaker to more easily locate the receptive field of a neuron by listening to its discharge. In the absence of any stimulus, many cells fire spontaneously one or a few spikes per second. If a small spot of light is placed in the cell's receptive field, however, the speaker erupts with a crackling that sounds like a machine gun firing away. This sound is the hallmark of an *on-center cell* (Figure 3.4A). When the spot of light is moved a bit outside the *center* of the receptive field, it has a suppressive effect. This is true for a light stimulus anywhere within a small region surrounding the central area. Removing the light spot from this inhibitory *surround*, by turning it off, provokes the cell into an off-response. Thus, for an on-center cell, a spot of light surrounded by a ring of darkness yields the strongest response.

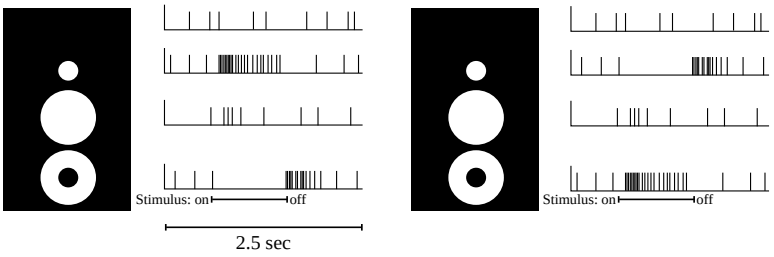

Figure 3.4: On-Center and Off-Center Cells
Firing response of an *on-center* (left) and an *off-center* ganglion cell (right) in the cat's retina in the dark (top row), to a small spot of light covering the receptive field (second row), a much larger spot of light (third row) and an annulus (bottom row). These neurons respond best to round patches of light or darkness. Modified from Hubel (1988).

Off-center cells show the same concentric *center-surround* organization, but with an inverted sign (Figure 3.4B); that is, a central region of darkness surrounded by an annulus of light maximally excites the neuron.

The receptive field of most retinal ganglion cells possesses a spatial antagonistic structure, with responses from the central patch opposing the responses from the peripheral region.

[10] Kuffler (1952).

This can best be demonstrated by a large spot of light that covers both center and surround; typically, cells will only weakly respond, if at all (Figure 3.4).[11]

As emphasized in Section 2.1, the notion of a neuron's receptive field constitutes the cornerstone of perceptual neuroscience and is not restricted to its spatial layout (that is, its center-surround organization). It includes the wavelength of the light to which the cell is maximally sensitive, the direction of motion of the stimulus the neuron prefers, and so on. This concept has been extended to other sensory modalities as well. For instance, the receptive field of an auditory neuron includes the pitch to which it is optimally sensitive to and whether it is excited by sound delivered to one or the other ear.

Two, usually unarticulated, assumptions underlie this concept. Foremost is the belief that the analysis of a complex scene by the entire organism can be broken down, in a highly atomistic fashion, into the response of individual nerve cells. This is likely to be oversimplified and groups of two or more cells, firing in concert, are likely to encode stimulus attributes that are not represented at the level of individual neurons.[12] Furthermore, any quantitative receptive field analysis relies on a choice of what feature of the neuronal response is critical to the rest of the brain. Is it simply the number of spikes or the peak discharge rate, two commonly used measures that assume a rate code (Section 2.3), or is there something about the temporal patterning of spikes that conveys information? For reasons of biological robustness and methodological convenience, most people simply count spikes within some meaningful interval.

I am now in a position to succinctly summarize one research strategy for discovering the NCC. It is to quantitatively correlate the receptive field properties of individual neurons with the subject's perception. If the structure of conscious perception does not map to the receptive field properties of the cell population under consideration, it is unlikely that these neurons are sufficient for that conscious percept. In the presence of a correlation between perceptual experience and receptive field properties, the next step is to determine whether the cells are, by themselves, sufficient for that conscious percept or whether both are only incidentally linked. To prove causation, many additional experiments are needed to untangle the exact relationship between neurons and perception.

A simple example will have to suffice at this point. Rather surprisingly, people don't know whether they see an image with their left or their right eye! If a small light is projected from dead-ahead into either one of the eyes, an observer can only guess which eye was stimulated (provided cheating, by blinking or moving the head, is prevented). The neurons that underlie visual consciousness do not encode *origin-of-eye* information in an explicit manner.[13]

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[11] Formally, on- and off-center cells encode the positive and negative half-wave rectified local image contrast. If the contrast is positive, on-cells respond and off-cells are silent; the converse is true if the contrast is negative.

[12] For the conflicting claims on how much information is found in correlated retinal ganglion cells spikes see Meister (1996), Warland, Reinagel, and Meister (1997) and Nirenberg et al. (2001).

[13] This does not imply that origin-of-eye information isn't exploited in binocular stereo or vergence eye movements. Humans just don't have conscious access to this information (von Helmholtz, 1962; Ono and Barbeito, 1985; Kolb and Braun, 1995).

3.5 Multiple Parallel Pathways Exit the Eye

Let me return to a more mundane, but critical, aspect of the eye—the axons of ganglion cells. It was the patron saint of neuroscience, the Spaniard Santiago Ramòn y Cajal who, at the end of the 19th century, first stained and identified the basic cell types of the vertebrate retina. Neurons are commonly classified like postage stamps—by their looks; that is, by the morphology, position, and size of their cell bodies, dendrites, and axonal terminations. Today, this information is often supplemented by identifying unique molecular constitutive elements, for instance, the presence of particular calcium-binding proteins.[14]

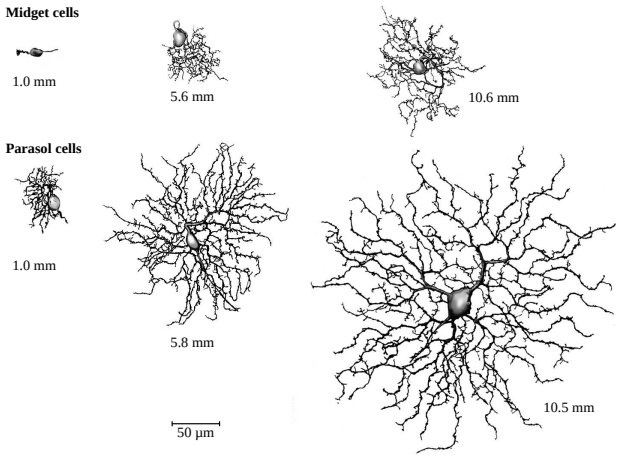

Figure 3.5: Retinal Ganglion Cells
Two cell classes dominate retinal output to the thalamus. At a given distance from the fovea, *midget cells* have small, compact dendritic trees and are much more common than *parasol neurons*, which possess larger dendritic trees. The size of these dendritic tree increases steadily when moving away from the fovea (that is, with eccentricity, indicated in millimeter distance from the fovea). Modified from Watanabe and Rodieck (1989).

By far the most numerous ganglion cells are midget neurons (Figure 3.5). In the fovea, a single cone provides, via an intermediary, the only input to a pair of on- and off-midget cells. While the on-cell increases its firing rate when stimulated by a spot of light, the off-cell does the opposite. It will, however, fire more vigorously when light is turned off inside the central region of its receptive field. Given the one-to-two connectivity between individual cone photoreceptors and midget cells, they serve as the conduit for signaling fine image details.

[14]For a translation of Ramòn y Cajal's best-known work, including extensive comments, see Ramòn y Cajal (1991). The modern study of the primate retina was inaugurated by Stephen Polyak (Polyak, 1941; Zrenner, 1983; Kaplan, 1991). For a masterful exposé of today's knowledge of retinal anatomy and physiology, consult Rodieck (1998).

About one in ten ganglion cells is of the parasol type. At any fixed distance from the fovea, parasol cells have larger dendritic trees and cell bodies than midget cells (Figure 3.5). A parasol neuron collects information from many cones and expresses this as either an increase (on) or a decrease (off) in firing rate when a light is turned on within its receptive field's center. The spatial extent of its dendritic tree increases with retinal eccentricity, as does the size of the associated receptive field.

The Lateral Geniculate Nucleus: Midway between the Retina and Cortex

When injecting a cell body with a chemical tracer, the substance is transported all the way to the axon terminals, staining the entire axonal process on the way, and allowing neuroanatomists to visualize the projection patterns of a cell population. Conversely, in retrograde transport the tracer moves back along the axon toward the cell body.

Applied to ganglion cells, these techniques reveal that at least nine out of ten project to a central, thalamic structure known as the *lateral geniculate nucleus* or LGN (Figure 3.6). It is the best known of several thalamic nuclei that process visual information.

The LGN is strategically positioned between the retina and the cortex. Incoming retinal information is switched over onto a geniculate projection neuron that sends this data onward to the primary visual cortex. The receptive field of the projection cell is nearly identical to that of its input fibers, so much so that it is usually assumed that no significant transformation of the retinal input occurs in the LGN.

Yet that assumption is unlikely to be true. The forward projection from the LGN to the primary visual cortex (V1) is paralleled by a massive cortical feedback. In the cat, about ten times more fibers project back from the deeper layers of V1 to LGN than project forward. Think of a video camera hooked up to a computer with a much thicker cable snaking back from the computer to the camera. About half of all synapses in the LGN originate in the cortex and many other synapses come from diffuse projections in the brainstem. What are they doing? At the least, it is likely that the cortex selectively enhances or suppresses retinal input passing through the LGN. Yet the function of this massive feedback pathway, characteristic of all thalamic nuclei, remains baffling.[15]

The LGN resembles a six-layered, warped cake. The lower two layers contain large cell bodies, called *magnocellular* neurons, while the upper four layers are characterized by small cell bodies termed *parvocellular* neurons. Closer inspection reveals a further substructure sandwiched between these layers: small, cone-shaped *koniocellular neurons*. The visual environment is mapped in a continuous manner onto all geniculate layers.

[15]For the anatomy of the forward and feedback pathways, see Sherman and Koch (1998), the monograph by Sherman and Guillery (2001), and Section 7.3. Many investigators believe that the cortico-geniculate or, more generally, the cortical feedback to all thalamic nuclei, helps to predict the presence of stimuli. This is known as *predictive coding* (Koch, 1987; Mumford, 1991, 1994; Rao and Ballard, 1999). Przybyszewski et al. (2000) cooled V1 in the cat, thereby turning it off, and showed that this affected the visual contrast-response curve of geniculate neurons.

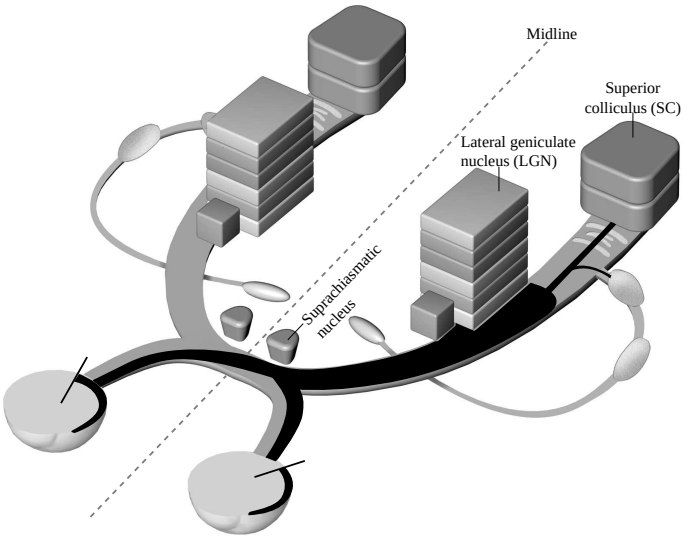

Figure 3.6: What Happens to the Retinal Output?
About 90% of fibers in the optic nerve project to the *lateral geniculate nucleus* (LGN) of the thalamus and from there onto primary visual cortex. This pathway dominates conscious visual perception. About 100,000 ganglion cells project to the *superior colliculus* (SC) at the top of the midbrain. These cells mediate relatively automatic opto-motor behaviors. Smaller subpopulations project to minor nuclei involved in housekeeping tasks outside the pale of consciousness. This is a schematic drawing. Panel **C** in the front endpages provides absolute scale. Modified from Rodieck (1998).

The Geniculo-Cortical Pathway Dominates Retinal Output

Each midget ganglion cell sends its output to one of the four parvocellular layers in the LGN. These, in turn, project into a sharply defined sublayer in V1, cryptically labeled 4cβ, a fraction of a millimeter thick (see Figure 4.1). The entire population of midget ganglion cells at all eccentricities, their geniculate targets, and their cortical recipients are known as the *parvocellular stream*, *channel*, or *pathway*. Similarly, each parasol ganglion cell projects to one of two magnocellular layers. These geniculate cells innervate sublayers 4cα and 6 of the primary visual cortex. Collectively, this brotherhood of cells is referred to as the *magnocellular pathway*. The koniocellular neurons have their own distinct termination zone in V1.

In biology, function and structure are tightly linked. Thus, the distinct anatomy of retinal ganglion cells and their separate termination patterns in the LGN and V1 are associated with profound differences in their behavior and function (Table 3.1).

Parvocellular neurons respond in a *sustained* manner to the on- or offset of light—that is, they keep on firing (albeit at a reduced rate) for as long as the stimulating light pattern

is present, while magnocellular neurons respond in a much more *transient* manner. In general, magnocellular neurons prefer rapidly changing stimuli, as occur during motion, while parvocellular neurons prefer sustained or slowly changing input.

There are many more parvocellular cells than magnocellular ones. Parvocellular cells represent the world at a fine grain. They also care about color. *Red-green opponent cells* receive inputs from L cones in the central, excitatory part of their receptive field, and opposing input from M cones in their surround. Complementary cells are driven by a greenish spot of light directed at their center and are inhibited by a reddish annulus. These populations correspond to the *red-green opponency channel* that had been inferred from sensory measurements as early as the 18th century. Magnocellular neurons are much less sensitive to wavelength and have no color opponency organization to speak of. They carry a signal related to the intensity or luminosity (with L, M, and S cone contributions).

Table 3.1 Conscious Vision is Mediated to a Significant Extent by Two Pathways that Originate in the Retina and Project into V1.

Property	Parvo cells	Magno cells
Color opponency	Yes	No
Receptive field size	Smaller	Larger
Cell size	Smaller	Larger
Response to light step	Sustained	Transient
Low-light pattern vision	No	Yes
Percentage of ganglion cells	70%	10%

A striking feature of these pathways is their anatomical independence. This makes it possible to selectively and deliberately destroy one or the other pathway by multiple injections of a chemical poison that destroys all the cell bodies in the appropriate layers of the LGN of monkeys. After complete interruption of either channel, the animal is trained to recognize color or patterns that require low or high acuity to properly resolve, similar to the way your optometrist tests your vision.

Eliminating the parvocellular layers profoundly affects color and high fidelity, spatial vision. The monkey has great trouble detecting either fine details or faint patterns and completely loses its ability to find a target purely on the basis of color. The animal's sensitivity to patterns that change rapidly with time remains intact. Destruction of the magnocellular pathway has no discernible effect on the monkey's sensitivity to fine details, yet severely limits its ability to detect rapid temporal changes.[16]

A third pathway, parallel to the magno- and the parvocellular stream, is the koniocellular one. Koniocellular neurons lack a pronounced, spatial center-surround organization,

[16]The system is redundant: the sense of visual motion partially survives destruction of the magnocellular layers; likewise, depth perception can be subserved by either system (Schiller and Logothetis, 1990; Merigan and Maunsell, 1993).

signaling instead chromatic opponency. That is, they respond to the difference between S cones and the sum of L and M cones. They are thought to be involved in the *blue-yellow opponency channel* inferred by Ewald Hering on the basis of perceptual color experiments.[17]

As long as the eyes are open, these pathways, with over one million fibers, carry more than 10 million bits per second of visual information. This is a lot. It is ironic to learn then, as I shall have occasion to explain in Chapter 9, the vast majority of this data stream is discarded by the conscious mind.

Although the magno-, parvo- and koniocellular neurons dominate retinal output, they are not the only ones. besides a large projection to the superior colliculus, discussed next, numerous minor classes of ganglion cells relay visual information to a motley collection of small nuclei that mediate blinking, gaze and pupilary control, diurnal rhythms, and other regulatory functions (Figure 3.6). None of these contain a map of the visual world. They are unlikely to play a role in conscious vision.

3.6 The Superior Colliculus: Another Visual Brain

About 100,000 ganglion cell axons run from the retina to the *superior colliculus* (SC) on top of the midbrain. The two SCs are the most important visual regions in fish, amphibians, and reptiles. In primates, much of their function has been taken over and extended by the cortex. Nevertheless, the superior colliculi retain a number of important visual functions underlying orienting responses as well as eye and head movements.

Patients who have lost part or all of V1 and adjacent cortical regions are blind in their affected visual field, even though their retino-colliculi pathways are intact.[18] Thus, SC activity probably is insufficient for conscious vision.

The colliculi are critically involved in the fast eye movements called *saccades* that primates incessantly engage in (more on these in the next few pages). The SC signals the difference between where the eye is pointed to now and where it is next supposed to go. This information is relayed directly to brainstem oculomotor areas controlling eye muscles as well as to the pulvinar nuclei of the thalamus.

The SC can be divided conveniently into superficial, intermediate, and deep layers. The superficial layer receives direct input from retinal ganglion cells in a topographic manner. Neurons in the deeper layers have been linked to behavior by direct injection of electric current. If the amplitude is strong enough, a saccade is triggered.

Eye Movements: Visual Saccades Are Ubiquitous

The eyes and their distinct patterns of movements are a fascinating source of information—not only for poets but also for scientists. Six eye muscles are responsible for rotating the

[17]Koniocellular geniculate neurons are driven by specific types of retinal ganglion cells. For their properties, see (Dacey, 1996; Nathans, 1999; Calkins, 2000; Chatterjee and Callaway, 2002).

[18]The clinical evidence is documented by Brindley, Gautier-Smith, and Lewin, 1969; Aldrich et al., 1987; and Celesia et al., 1991.

eyeball in several distinct patterns.

A *saccade* is a rapid movement of both eyes yoked together. Evolution minimized the duration for which the eyes are in transit to less than a tenth of a second. Saccades are said to be *ballistic*—like a V-2 rocket from World War II. The brain aims to reach a particular spot; once the eyeball is launched, no visual control is exerted until the eye comes to rest again. When the eye movement is off-target, a *corrective* saccade of small amplitude brings the target right onto the fovea.

You move your eyes all the time. You read by skipping with a series of small saccades across the text. You look at a face by constantly glancing at its eyes, mouth, ears, and so on. At a couple of saccades per second, your eyes move more than 100,000 times a day, about as often as your heart beats. Still, almost none of this furious activity makes it into consciousness (this claim will be buttressed in Section 12.1).

The intervals between saccades are brief, as short as 120-130 msec. This corresponds to the minimum time needed to process visual information during the fixation periods.

Rapidly displacing the eyes appears effortless, but requires fine coordination among a large cast of players dispersed throughout the brain. Drawn in bold strokes, two parallel pathways mediate saccades. Reflexive, orienting eye movements (as when something appears off to one side) are mediated by the superior colliculus. Planned, voluntary saccades are the responsibility of posterior parietal and prefrontal cortical regions. If one system is impaired, the other one compensates to a limited extent.[19]

When you track a target, say a bird in flight, your eyes move in a pattern known as *smooth pursuit*. This movement involves quite distinct parts of the brain.

Vision Fades When the Image Is Stabilized

If eye movements are prevented (for instance, by artificially stabilizing an image at the same retinal location), vision rapidly fades. If you are ever a subject in a visual functional brain imaging experiment, you will be instructed to keep your eyes as still as possible to minimize motion artifacts that cause a reduction in the signal amplitude. You will lie in the magnet and studiously stare at the fixation mark. This can lead to a gradual fading of the entire visual field—a sort of blackout—that can be counteracted by blinking.[20]

It is often assumed that fading is a purely retinal phenomenon, caused by a temporal derivative-like operation implemented by retinal neurons. They follow the motto: if nothing changes, don't bother to report anything. This cannot be the complete story, however, because experiments carried out in the late 1950s showed that the fading of line drawings depended on a variety of global figural qualities not expressed in the retina.

Unfortunately, little is known about the neuronal basis of fading. The excitability of the neurons expressing visual consciousness should mirror the perceptual fading. That is, as an

[19]The neurophysiology of eye movements is summarized by Schall (1991) and Corbetta (1998).

[20]It can take anywhere from a fraction of a second to a minute or more for an image to fade (Tulunay-Keesey, 1982; Coppola and Purves, 1996). Fading depends on whether the subject attends to the figure, whether the figure is meaningful and so on (Pritchard, Heron, and Hebb, 1960).

image fades from consciousness, so should the relevant activity of the NCC.

Saccadic Suppression, or Why You Can't See Your Eyes Move

What effects do eye movements have on the rest of the system? When playing around for the first time with a video camera, you quickly discover that filming your toddler by following her around the house is likely to induce nausea when viewing the result. Sudden camera movements and turns give rise to an uncomfortable sense of induced motion. Why, then, don't you experience such feelings each and every time you move your eyes? Subjectively, the outside world looks remarkably steady. How come?[21]

Another expected detrimental effect of rapid eye movements is image blurring, as would occur, for instance, while trying to capture a moving car on a photograph using a slow shutter speed. During the 30 to 70 msec duration of the saccade, the visual field should be horribly smeared out, yet it continues to look sharp. What is going on?

The stability and sharpness of the visual world during eye movements is a consequence of numerous processes, including *saccadic suppression*, a mechanism that interferes with vision during eye movements. You can experience saccadic suppression by looking in a mirror and fixating first your left and then your right eye, back and forth. You will never catch your eyes in transition. Your eyes are not moving too fast, because you can see perfectly well the saccades a friend is making. During the time your eye is in transition, vision is partially shut down. This eliminates blur and the feeling that the world out there is jerked around every fraction of a second.[22]

Why, then, isn't everyday vision characterized by annoying blank periods? This must be prevented by some clever, *trans-saccadic integration* mechanism that fills in these intervals with a "fictive" movie that needs to be a composite of the image just before and just after the saccade. The mechanisms and neuronal sites of this integration remain largely unknown.[23]

[21]The world doesn't have to appear stable, as the neurological patient R.W. knows only too well (Haarmeier et al., 1997). His world spins in the opposite direction every time he moves his eyes or head. His visual acuity and ability to judge motion are normal. Bilateral lesions in his parieto-occipital cortex destroyed motion compensation.

[22]How this occurs remains a hotly debated question. One school asserts that eye movements actively suppress intrasaccadic motion processing, while the opposing camp asserts that visual factors such as forward and backward masking cause the suppression (Burr, Morrone, and Ross, 1994; Castet and Masson, 2000). More likely than not, multiple processes contribute. Note that saccades don't completely prevent vision. That something can be seen during saccades can be ascertained by looking at the crossties of railroad tracks from a moving train while making a saccade against the direction of motion of the railroad car. The artist Bill Bell has exploited this in his "Lightsticks" art pieces. Viewed on a dark background, these vertical bars of blinking LEDs paint a picture of an animal, flag, or face onto the retina of the viewer who rapidly saccades across them. When fixating them directly, however, only a flickering bar of red light is seen.

[23]McConkie and Currie (1996).

Blinks

The eye cleans itself by blinking the lids and lubricating the front of the cornea with tear glands. Typically, you'll make a few blinks while reading this paragraph. Each one briefly blocks the pupil, causing an almost complete loss of vision for a tenth of a second or so. Yet, while you are exquisitely sensitive to a brief flickering of the room lights, you are almost totally oblivious to blinks.[24]

Accordingly, I expect the NCC neurons to be indifferent to blinks. That is, while retinal neurons should stop firing during a blink, the NCC neurons should remain firing during this temporary shutdown of vision.

Adding up all of the little snippets of the running movie that constitutes daily life and that are "lost" due to saccadic and blink suppression amounts to a staggering 60 to 90 minutes each day! An hour or more during which sight should be compromised, yet is not. And, until the advent of modern science, no one was aware of this.

3.7 Recapitulation

The retina is an amazing piece of highly laminated neural processors, thinner than a credit card, with more than fifty specialist cell types. Ganglion cell axons make up the optic nerve that leaves the eye. Think of them as wires that convey messages encoded as a temporal sequence of electrical pulses, organized along multiple parallel channels. A loose analogy can be made with a set of dozens of cameras, one transmitting black-and-white information, one red-green, and another one blue-yellow color opponency information, one channel emphasizing locations whose intensity is changing in time, and so on.

The best-studied of these are the magno-, parvo- and koniocellular pathways that project, via the lateral geniculate nucleus into the primary visual cortex. Magnocellular neurons signal luminosity and temporal change, as occur during motion, while parvocellular neurons transmit red-green information and fine spatial details. The koniocellular pathway cares about blue-yellow opponency and even less well understood image features. All of these are critical to conscious, visual experience.

The second largest retinal tract projects to the superior colliculus and is involved in automatic forms of rapid eye movements. A great deal of further machinery is dedicated to subserving saccades and other eye movements that are rapid, accurate, and adaptive. Minor sets of ganglion cells project to odd places in the brainstem. These control gaze, the pupilary diameter, and other important house-hold functions. Yet the vast majority of all this information is likely to be inaccessible to consciousness.

You do *not* see with the eyes but with the cortex. Discrepancies between what ganglion cells encode and what you consciously perceive includes the dramatic decrease in spatial acuity away from the fovea, the existence of a mere two photoreceptor types at the point of sharpest seeing, the paucity of color representation in the periphery, the blind spot, image blur during eye movements, and transient loss of visual input during blinks.

[24] Volkmann, Riggs, and Morre (1980); and Skoyles (1997).

Neuronal structures in the forebrain read out the optic nerve signals and generate a stable, homogeneous, and compelling view of the world. While the eyes are necessary for normal forms of seeing, the NCC are most certainly not to be found in the retinae. So let me now introduce the visual cortex.