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PHYSIOLOGY AND PSYCHOLOGY OF COLOR VISION -- a review

by

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Abstract: This paper is a review of the anatomy, physiology, and psychology of human color vision.

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Introduction

Color vision is an interesting and controversial area of research, or as Cornsweet put it: "few if any areas of scientific endeavor include more miscellaneous facts and more confused theoretical discussions than the area of color vision" (Cornsweet p. 205). In this paper I will present an introduction to this problem and and a brief review of the anatomy, physiology, and psychology of color vision.

Color vision is the ability to discriminate between lights of different wavelengths in a manner that is independent of their intensities. Color is defined as a sensation, and therefore it makes sense to talk about the color of a point in an image only in terms of *perceived color*, or the sensation we feel when presented with the same stimulus. One of the major difficulties in color vision is that the perceived color of any point in an image is not simply a function of the amount of light at various wavelengths at that point. If this were true, our perceptions of color would change with variations in illumination. Instead, the colors we see usually correspond to the surface properties of the objects around us. This is obviously a useful feature from the standpoint of survival, since it might prove dangerous to have our visual perception change drastically every time a cloud moved in front of the sun. If, on the other hand, our skin contained chlorophyll and our survival depended on absorption of light, perhaps our visual systems would be more sensitive to illumination than reflectances.

We do not know how to compute perceived color for generalized scenes. One reasonable approach to this problem is to examine one of the few devices that is

capable of accomplishing this task: the human brain. Unfortunately, most of our information in this area concerns the first dozen cells in the human visual processing system, which is normally called the "visual pathway". The next chapter of this paper will provide a brief introduction to the anatomy and physiology of this part of the brain. In the third chapter, I will discuss the human color processing system from an overall behavioral point of view.

Color vision in computers does not have to be a model of the human brain. It is quite possible that the most efficient way of processing color information in a machine is completely different from the algorithms used in human beings. The most important requirement is that the computer vision system should be roughly equivalent to the human one in terms of behavior. The computer should "see" the same colors as a human being who is confronted with the same stimulus. I believe, however, that an understanding of the human visual system is an important first step towards achieving the goal of a general computer vision system.

The Basic Physiology of Color Vision

In this chapter I would like to present a brief review of what is known at this time about the anatomy and physiology of the human visual system. The most detailed information in this area concerns the connections of the eye to the brain, which are called the *central pathways*. The optic nerves from the two eyes enter the cranium and then come together to form the region known as the *optic chiasm*. In the higher vertebrates, the fibers from the medial (nasal) half of the eye cross over in the chiasm to connect to the opposite half of the brain (see figure 2.1). This means that since the lens in the eye inverts the image, the left half of the brain receives sensory information from the right half of the visual field (see Gassaniga).

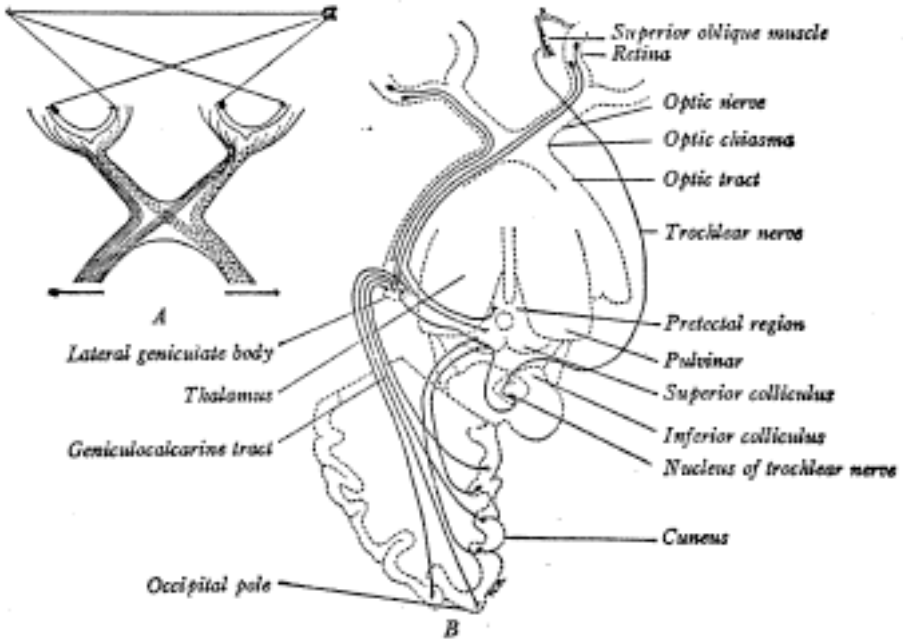

Figure 2.1 The Anatomy of the Visual Pathway

The optic nerve fibers then form the *optic tract* which (in higher

vertebrates) projects to various areas of the brain. Most of the fibers (about 70 to 80 percent) connect to the *lateral genticulate nucleus* (LGN) in the thalmus, which sends fibers to the *striate cortex* (or "primary visual cortex"). The majority of the remaining fibers go to the *superior collticulus* and the *pretectal region*. There are nerve fibers which run from the superior colliculus to the muscles of the eye, and it is believed that this is one of the areas of the brain which control eye movements. The pretectal region sends fibers to the eye muscles which control the size of the pupils.

The available physiological evidence indicates that color information is transfered through the LGN to the cortex. In the next section of this chapter, I will describe the properties of the nerve cell, or *neuron*. The remaining sections will deal with the three parts of the visual pathway: the retina, the LGN and the striate cortex.

The Neuron

The nervous system is made up of a special set of cells called nerve cells, or neurons. Although there are many different kinds of neurons, they normally have three parts: a *cell body* which contains the nucleus, a long fiber or *axon* attached to the cell body, and a set of short fibers or *dendrites* (see Figure 2.2). The neurons interact at the junction of the axon of one neuron and the dendrites of another neuron which is called a *synapse*. The axon is normally longer than the dendrites and may be up to 3 feet in length. At its end, the axon splits into many fine branches which end in *terminal buttons*. These branches may connect to the dendrites of different neurons, and there may be multiple synapses with

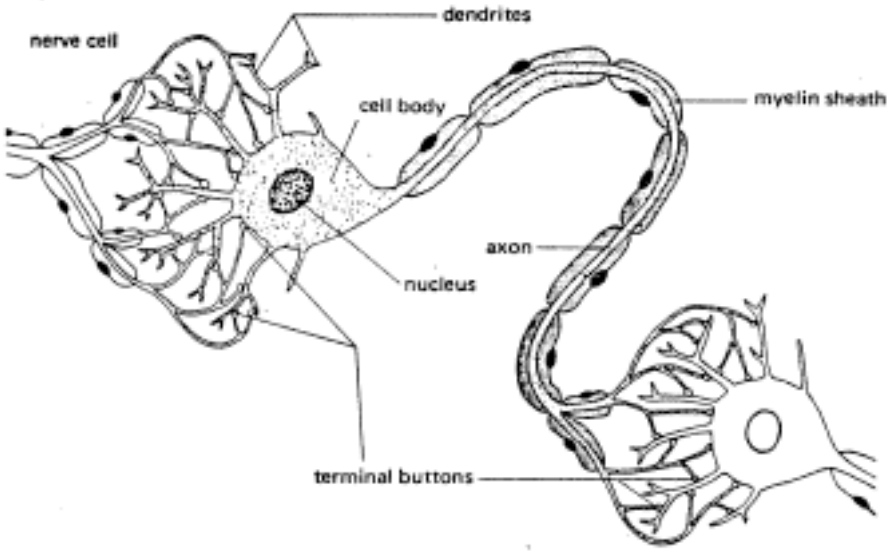

Figure 2.2 The neuron (from Blakemore)

a particular neuron.

All cells in an organism are made up of a nucleus, cytoplasm and a membrane which encloses the whole structure. The entire surface membrane of the neuron is electrically polarized; the inside of the cell being some 70 to 90 millivolts negative with respect to the outside. This potential is called the *membrane potential* or *resting potential* of the cell. A decrease in the membrane potential is called a *depolarization* and an increase is called a *hyperpolarization*. One of the basic properties of the neuron is its ability to propagate local membrane depolarizations which are called *nerve impulses* or *action potentials*. In any particular nerve fiber, the impulses have essentially the same magnitude and form and they travel with the same speed. Thus the strength of a nervous signal is transmitted by the frequency of the nerve impulses, and not by their amplitudes.

The local depolarization lasts for only about one millisecond at any point in the nerve fiber, but propagates at a rate of 1 to 100 meters per second. The speed of the

impulse is related to the diameter of the axon. In general, large fibers conduct at higher velocities than small fibers. Some axons are enclosed by a sheath of *myelin* which has a high electrical resistance. These fibers can propagate action potentials at higher speeds than bare fibers of the same diameter. The myelin sheath has gaps in it called *nodes of Ranvier*. The action potential does not propagate down the entire axon, but is formed only at the nodes. This is called *saltatory conduction* (from the Latin "saltare" which means to leap). The axons of the retinal ganglion cells, which form the optic nerve, are myelinated and very small in vertebrates (about 1 to 15 microns). This makes it very difficult to record the electrical activity of individual fibers in the primate optic nerve.

The transmission of the nerve impulse across the synaptic gap between neurons is in most cases chemical, not electrical. The endings of the axon release a transmitter chemical which produces changes in the post-synaptic membrane. This change can take two forms: depolarization or hyperpolarization, which are called *excitation* and *inhibition*. The dendrites of a particular neuron may be connected to the axons of many other neurons, some in an excitatory and some in an inhibitory fashion. The post-synaptic neuron must reach a certain threshold level of depolarization, called the *generator potential* before it will fire a nerve impulse. The release of transmitter chemical from the endings of an axon is to some extent proportional to the frequency of impulses arriving at the synapse (DeValois 1966) and the change in post-synaptic membrane potential is proportional to the amount of chemical released. The chemicals released by a single impulse are generally insufficient to fire the second neuron, so there must be a summation of many different effects at the synapse and thus some analysis of the information coming in from the

presynaptic neurons.

This analysis can take on several forms. If two neurons synapse on a third neuron, and both are excitatory, the output will roughly be the sum of the outputs of the two neurons. An unstimulated neuron is never really at rest. There is always spontaneous firing, so that inhibition can be seen as a decrease in firing rate. Thus if two neurons synapse on a third in an inhibitory fashion, there will be a decrease in firing rate proportional to the sum of the firing rates of the two neurons. If one axon is excitatory and one inhibitory, the effect may be to subtract one from the other. It is also possible for an axon to synapse on another axon. An inhibitory synapse on an excitatory axon is called *presynaptic inhibition*, and the effect may be closer to division than subtraction (DeValois 1966).

This combination of excitation and inhibition is very important in understanding the physiology of our visual information processing. As we shall see in later sections, it has led to plausible explanations of some seemingly complex effects, such as the hypercomplex cells in the visual cortex which respond to certain types of lines at specific orientations which move in a specific direction.

The Retina

Light enters the eye through the cornea, passes through the pupil and lens, and then falls on a photosensitive layer at the back of the eye, which is called the *retina* (see figure 2.3). The retina is actually a part of the brain which pushes out in the

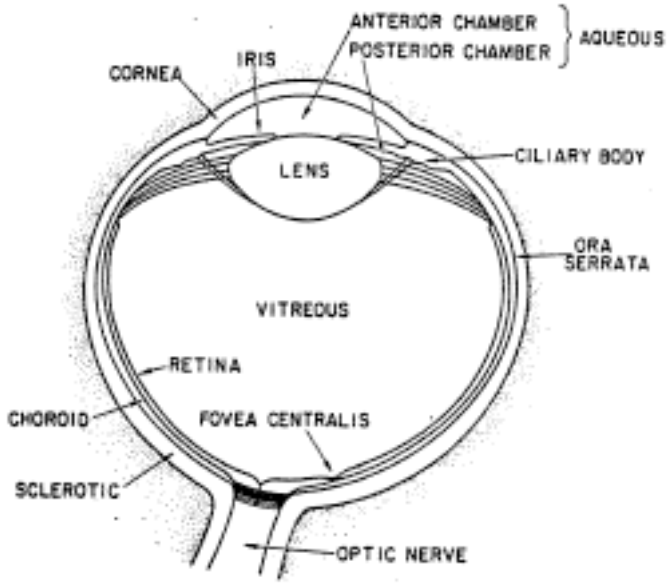

Figure 2.3 The anatomy of the eye (from Eyzaguirre)

development of the embryo to meet the specialized skin tissues that will form the eyeball and lens (Blakemore). There are five kinds of nerve cells in the retina: the *receptors*, *bipolar*, and *ganglion* cells which form the visual pathway in the retina, and the *horizontal* and *amacrine* cells which provide lateral interconnections. The receptors synapse with the horizontal and bipolar cells, and the axons of the bipolar cells synapse with the amacrine and ganglion cells (see figure 2.4). There is also some evidence for direct receptor connections in the primate retina. The axons of the ganglion cells form the *optic nerve* which makes up 38 percent of all nerve fibers entering or leaving the central nervous system (Ranson and Clark).

One surprising fact about the anatomy of the eye is that the first cells in the visual pathway, the receptors, are at the very back of the retina. Light must pass through the optic nerve fibers, blood vessels, and all the other cells before reaching the light receptive cells. There are two different kinds of receptors: the *rods* which are responsible

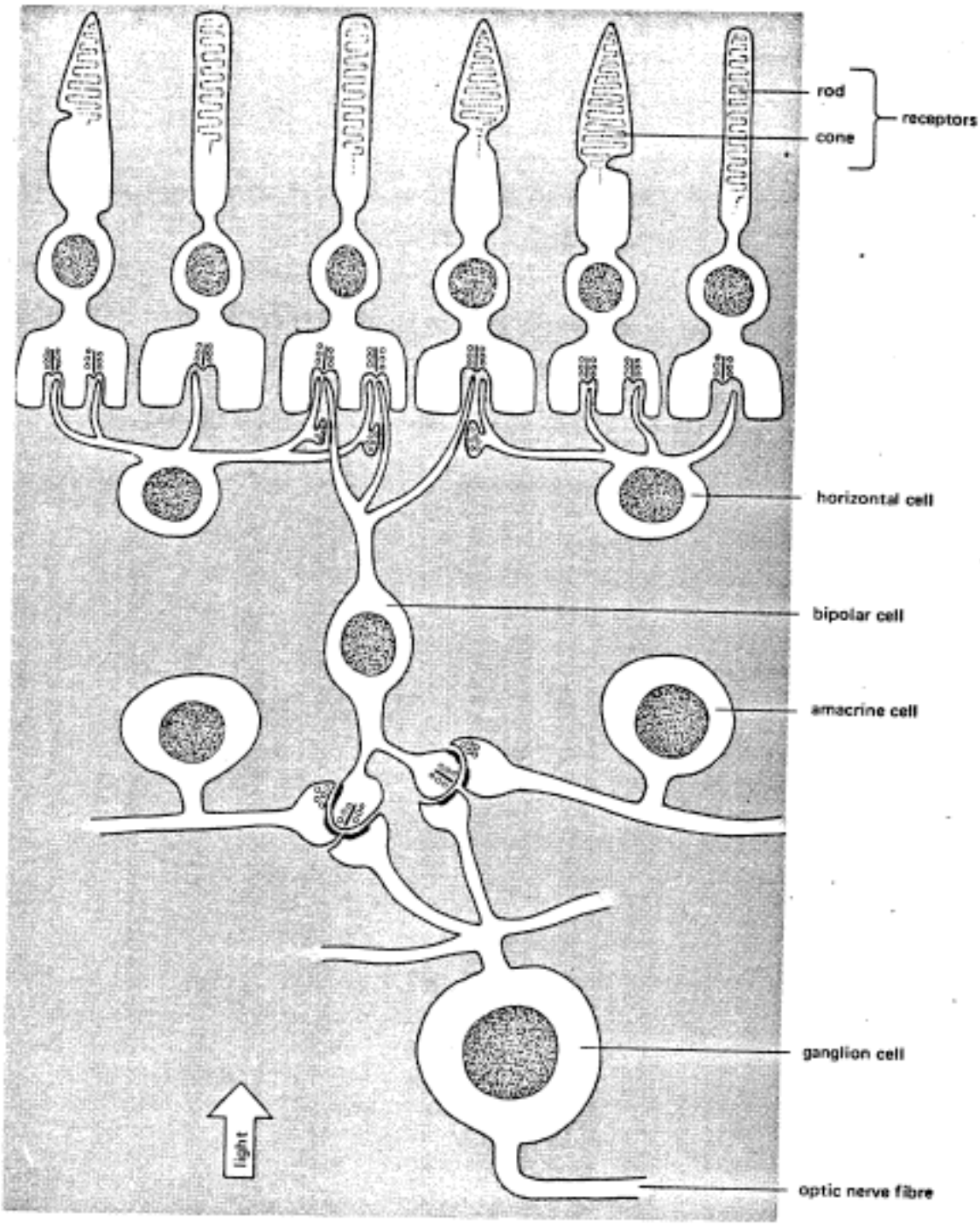

Figure 2.4 Schematic diagram of the retina (from Blakemore)

for low light vision, and the *cones* which are used for bright light and color vision. In the

primates (and many other animals) there is a special region of the retina, called the *fovea*, which is specially adapted for detailed vision. The human fovea is in the center of the visual field and contains only cone receptors. There are also no blood vessels over this area to interfere with the light. Although the human fovea is only the size of the head of a pin, and is only two degrees of the visual field, all of our high detail vision originates there. For example, the words you are now reading are being focussed on your fovea. Human foveal vision is very small in terms of the whole visual field, and is about the size of a quarter at an arm's length (70 cm.) away from the eye. On the nasal side of the eye, about 5 mm (or 16 degrees of the visual field) from the fovea, is an area where there are no receptors, called the *blind spot*. This is where the optic nerve fibers leave the eye.

The receptors in the human retina are a truly amazing feat of nature. They are able to discriminate brightness differences over a range of a billion to one, and yet, as Hecht demonstrated in 1942, the rod cell can be activated by the absorption of only one photon of light (see Cornsweet). It has been known for over one hundred years that the eyes of a frog (and many other animals) contain a pigment which is red in dim light and that the color gradually fades, or *bleaches* when the eye was exposed to light. It is now known that this pigment, called *rhodopsin* or *visual purple*, is contained in the outer segments of the rod receptors. The cone receptors, as a class, contain three other pigments which are chemically similar to rhodopsin but differing in some important characteristics.

The receptors are composed of a inner segment, much like an ordinary neuron, and a rod or cone shaped extension which is sensitive to light. The discrimination between rods and cones on the basis of the shape of this segment is good in some animals,

such as the frog, but inadequate in the human retina where the cones in the center of the fovea look more like rods. Therefore, the differences between the two classes of photoreceptive cells are normally based on two other criteria: that the rods are the cells which contain rhodopsin, and that the rods are the cells which are active when the retina has been dark adapted for ten minutes and is then exposed to dim light.

A quantum of light can be absorbed by, or in general activate, only one molecule or atom (Wald 1965). This means that the absorption of one photon by one rhodopsin molecule in a rod (which contains about 4 million such molecules) is enough to activate the receptor cell. Exactly how this happens is unknown, but there first part of the process, the photochemical reactions of rhodopsin are well documented. It has been discovered through the use of the electron microscope that the pigment-bearing section of the rod cell is filled by a membrane which is arranged in layers. Since adjacent layers of the membrane are in contact with each other, it is evident that the pigment molecules are actually imbedded in the membrane (see Cornsweet). Rhodopsin is made up of a protein (an *opsin*) and the aldehyde of vitamin A, called *retinal*. Retinal has a double carbon bond and therefore has two geometric configurations, or isomers. The only action of the light is to change the geometric configuration of the retinal molecule from the 11-*cis* to the *all-trans* isomer. All other changes are *dark* reactions, which correspond to changes in the protein molecule, and the final product is a mixture of opsin and free all-trans retinal. The molecule will lose its color during this process, which of course corresponds to the bleaching of the pigment. Molecules in the final state spontaneously

regenerate to form rhodopsin, but this part of the process is slow. The first part (the photon changing the geometry of the retinal) is virtually instantaneous. The rest of the process which leads to the separation of the retinal and opsin are believed to take about 1 second. The regeneration of rhodopsin, however, requires an average of 5 minutes (Cornsweet pp. 91-96).

The geometry of this process is very important. In the rhodopsin molecule, the retinal covers a pocket in the opsin (the protein). The cis-trans isomerization straightens out the retinal and causes one end to come off the protein. The pocket in the protein then opens up, until finally the trans-retinal is separated from the opsin (see Figure 2.5). Most of the speculation about how this process triggers the electrical activity of the rod

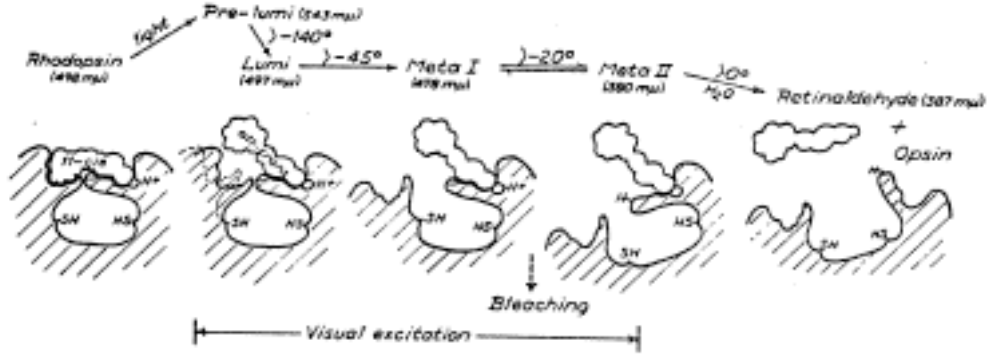

Figure 2.5 The rhodopsin cycle (from Wald and Brown)

are concerned with the opening of this pocket (see Cornsweet). One theory states that the opening of the pocket changes the permeability of the membrane, permitting the flow of ions as long as the pocket is open. This could cause a change in the membrane potential which might propagate to other parts of the cell. Another theory speculates that the pocket contains an active site of the protein that generates a chemical transmitter, and that

opening the pocket releases this chemical which causes a depolarization or hyperpolarization of the membrane.

The pigments of the eye absorb light in only a small range of wavelengths, from about 400 to 700 nanometers (nm), which is called the *visible spectrum*. Each of the pigments in the eye absorb different amounts of light at different wavelengths in this range, which is known as the *spectral sensitivity* of the pigments. The spectral sensitivity curve for rhodopsin has been determined from both psychophysical experiments and spectroscopic analysis of rhodopsin which has been extracted from the eye (see Figure 2.6). Both of these methods produce a bell-shaped curve with a peak value near 500 nm (in

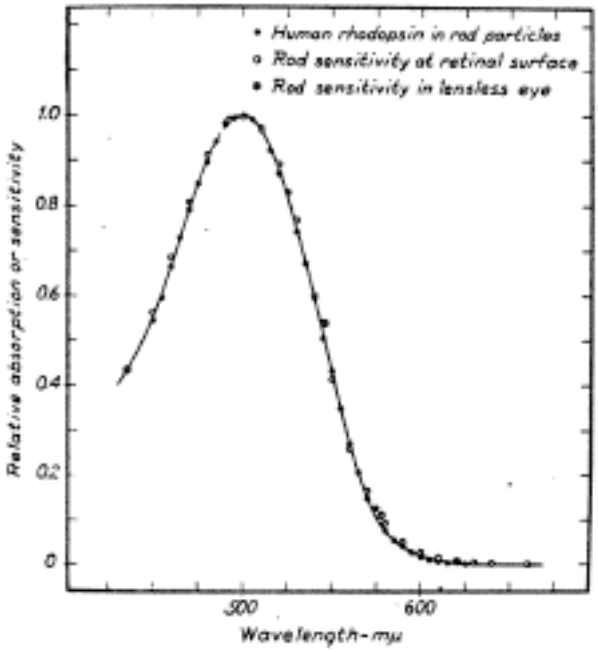

Figure 2.6 Spectral sensitivity curve for human rhodopsin (from Wald and Brown)

the blue-green part of the spectrum). The psychophysical experiments consist of measuring the spectral sensitivity of the eye in dim light. This is why the two criteria mentioned

earlier for distinuishing between rods and cones are equivalent. In other words, in dim light the eye is absorbing light in only one of its pigments, rhodopsin, and there can be no color perception. The fovea, which has no rods, is therefore completely blind at night.

There are three cone pigments which have their peak absorptions in the regions of 445, 540 and 570 nm (see Figure 2.7). There is a great deal of overlap in these

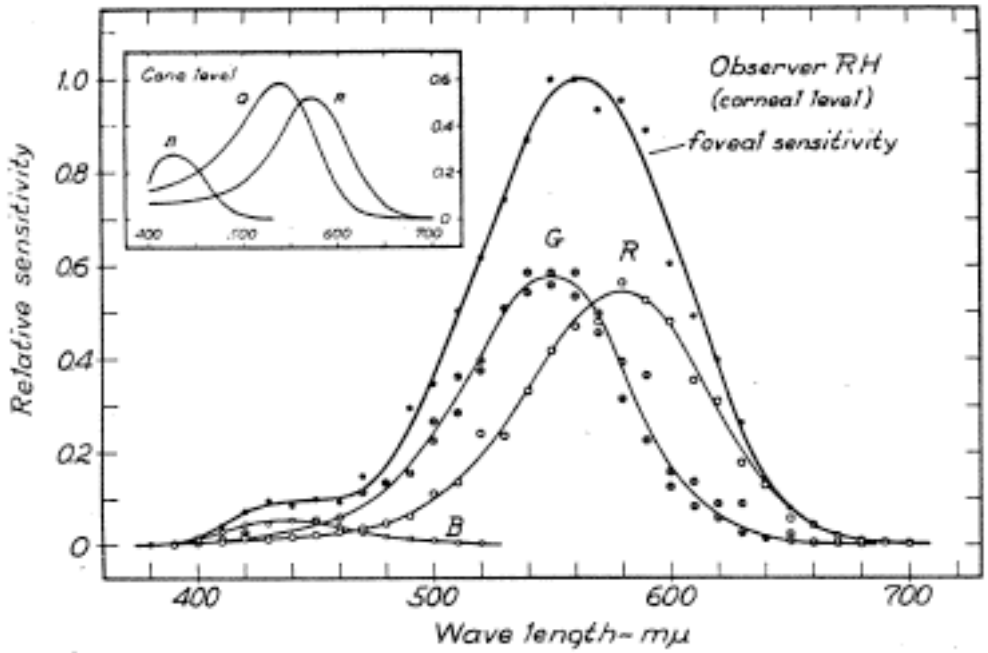

Figure 2.7 Cone sensitivity curves (from Wald and Brown)

absorption curves, and at least the latter two cone pigments absorb some light over the entire visible spectrum. Each cone in the retina of a normal person contains one of these pigments. There are therefore three types of cones, which are often called the *blue*, *green* and *red* cones. This, however, can be misleading. The 570 nm cone, which is called "red", is not limited in spectral absorption to the region we see as red (600-700 nm), and does not even have its peak absorption in this area. This type of cone is in fact more sensitive to the

part of the spectrum we perceive as pure green (510 nm) than to pure red (650 nm). I will therefore use the notation recommended by DeValois (Devalois 1973): *L cone* (long wavelength cone), *M cone* (medium), and *S cone* (short) to denote the cone types with peak absorptions at 570, 540 and 445 nm, respectively. This seemingly arbitrary decision is important because the cones are not "color receptors", but "light receptors". Each cone absorbs light from the whole visible spectrum, and therefore color perception must be based on cells that analyze the information from more than one cone type.

There are about 120 million rods and about 1 million cones, but only about 1 million ganglion cells (or fibers in the optic nerve), so some processing of the visual information must be carried out by the retina. The interconnections between the cells in the retina are very complex, and the theories about the roles that the different cell types play in retinal processing are speculative at best. There are is a popular misconception that since there are so many rods, each ganglion cell must receive input from a large number of rod receptors, while each cone is connected to only one ganglion cell. Therefore the rods have "party lines" to the brain, while the cones get "private lines". This is simply not true. There is good evidence that each ganglion is connected to many rods, but this is also true for cones. The activity of each ganglion cell is affected by the actions of a large number of cones, and each cone influences many ganglion cells.

Because of the complexity of the primate retina, many of early electrical recordings of visual responses were from the horseshoe crab, *Limulus* (see Miller, Ratliff and Hartline). Although the compound eye of the Limulus is much simpler than the eyes of vertebrates, it still clearly shows the effect of neural interaction. The eye of the Limulus is

made up of 1000 ommatidia ("little eyes"), or facets. The output of these receptors feed into eccentric cells whose axons form the optic nerve without intermediate cells. There are mutual interactions among the eccentric cells, and these interactions are always inhibitory. This means that when light falls on neighboring ommatidia, the output of each is less than it would be if the same amount of light fell on that ommatidia alone. The degree of inhibition is proportional to the firing rate of the inhibiting neuron, and tends to decrease with the distance between the eccentric cells. This type of neural interaction, which is called *lateral reciprocal inhibition* seems quite simple and reasonable in terms of our understanding of the neuron and synapse, and yet it leads to some seemingly complex effects. If there is a sharp contour in the visual field so that half of the receptors are strongly illuminated and half are dimly illuminated, the response will tend to emphasize the border area. In the bright area of the field, there will be some decrease in response due to mutual inhibition, but the cells near the border will be less inhibited since some of their neighbors are receiving less light. This produces an increase in response on the bright side of the border. Similarly, the cells on the dark side of the border will have more inhibition than dark cells far from the contour, which produces a decrease in response on the dark side of the edge. This phenomenon is often deceptively called "edge enhancement", which implies that the contrast across contours is increased. The difference between the maximum and minimum response across the edge is actually about the same (see figure 2.8). The net effect of this phenomenon is that the information in the visual system is transfered in terms of the changes across boundaries in the visual field.

In recent years, experimenters have been able to record the electrical

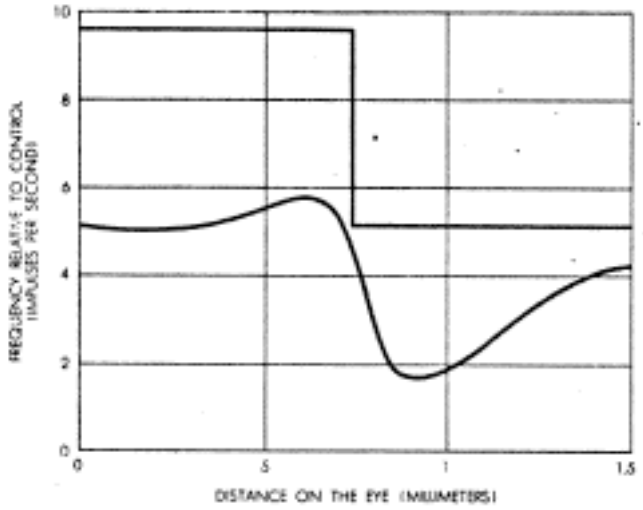

Figure 2.8 Limulus response to step pattern (from Miller, Ratliff and Hartline)

activity of the cells in vertebrate retinas. The absorption of light produces a *hyperpolarization* of the receptor cell. The changes in membrane potential are not impulses, but a graded change which consists of an initial transient that decays to a steady level. There is a also a small *off* transient when the light is removed. There are no impulses in electrical potential of the receptors, bipolars, or horizontal cells, which may be due to their close proximity to each other. The hyperpolarization seen in the receptors is surprising since the release of chemical transmitter at most synapses is triggered by depolarization. The resting potential of the receptors is about 30 mV, and the change in potential is about 5 mV. The receptor response seems to follow the relation:

$$V/V_{max} = I / (I + K)$$

where K is about 800 quanta/rod/sec for humans (see e.g. Marr 1974). This is consistent with the data that shows the receptors to be linear (in the log domain) over a range of two log units (see Cornsweet pp. 251-253). In other words, the output of the receptors seems to be

approximately the logarithm of the intensity within a certain range. The importance of this relation will be seen later in the discussion of the incremental amount of brightness necessary to discriminate between two areas in the visual field.

The dendrites of the horizontal cells synapse with about 7 to 12 cone cells. The horizontal cell axon synapses with a large number of rod cells. Each cone is connected to about 6 different horizontal cells, and each rod synapses with exactly two different horizontal cells. The electrical response of the horizontal cell is also a graded hyperpolarization. It is linear over 3 log units and saturates at a higher intensity when diffuse rather than spot illumination is used, which may be caused by a weighted summation of many receptors (Marr 1974).

There are several types of bipolar cells. Each type of bipolar cell synapses with either rods or cones. Each rod synapses with about two bipolars, and each rod bipolar is connected to between 14 and 45 rods. The receptive fields of bipolar cells are divided into two concentric antagonistic zones. About half of the bipolars hyperpolarize to central illumination, and half depolarize. Peripheral illumination can reduce the response from the central region, but is not capable of producing a signal in the opposite direction. The magnitude of the response is constant over a wide range of absolute intensities for a fixed ratio of center to surround illumination.

The amacrine and ganglion cells are the first cells in the visual path that produce nerve impulses instead of graded potentials. There are several kinds of amacrine cells, which differ in the size of their receptive fields. These cells respond best to sudden changes in the level of illumination, and give little response to gradual changes.

The receptive fields of the ganglion cells are circular and divided into two concentric antagonistic regions (see Figure 2.9). The centers of the fields range in size

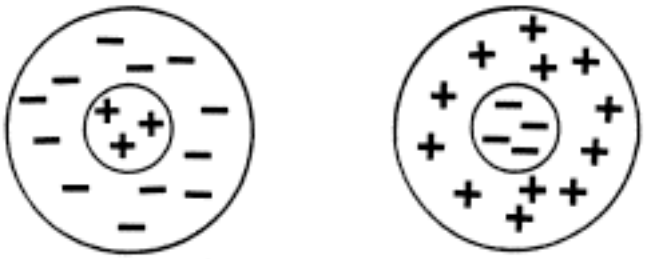

Figure 2.9 Center surround receptive field organization

from 0.1 to 2 degrees of visual angle, with the surround being somewhat larger. The size of the receptive fields of ganglion cells are smallest in the center of the fovea, and gradually increase toward the periphery of the retina. The ganglion cells have been traditionally separated into two groups: *on-center* cells that respond to illumination in the center of their field, and *off-center* cells that respond best to a dark region on a light background. More recent work has shown that there are three distinct types of ganglion cells: the *W-cell*, *X-cell* and the *Y-cell*. Little is known about the W-cells, except that they seem to be motion sensitive. The axons of the W-cells project to the superior colliculus and therefore these cells are probably not an important factor in color perception. The response of the X-cells in bright light correspond to a linear summation of inputs from two concentric antagonistic fields (as described above). They are relatively insensitive to flicker over a wide range of frequencies. The Y-cells do not respond well to stationary contrast, but are very sensitive to movement and flicker. The axons of the X-cells and Y-cells project to the LGN, and their importance will be seen later in the discussion on the cells in the visual cortex.

In bright light, the X-cells and Y-cells exhibit center-surround fields of

both the on-center and off-center type. This effect, however, is not present in dim light. This change can not be explained in terms of a "switch-over" from cones to rods, since it occurs at light levels where the cones are active (Brindley p. 87).

The spectral sensitivities of the ganglion cells in a light-adapted eye are complex, and do not seem to fall into simple classes that correspond to the spectral response curves of the receptors. The spectral sensitivity can be altered by adaptation with monochromatic light, which indicates that the ganglion cells receive input from more than one type of cone cell. Most of the data on the color properties of the primate visual cells comes from the LGN of monkeys, which will be discussed in detail in the next section. Most of the evidence, however, indicates that the spectral sensitivities of the primate ganglion cells are similar to those found in the LGN.

The LGN

The lateral geniculate nucleus (LGN) contains the fourth neuron in the visual pathway, and acts as a relay station between the retina and the visual cortex. During the last fifteen years, DeValois and his associates have made extensive recordings of the spectral responses of the LGN cells of the macaque monkey, which has a visual system that is extremely similar, if not identical, to that of man (DeValois 1965). DeValois has compared the physiological properties of the macaque LGN cells to the psychophysical studies of human color perception, which has produced some very interesting results.

The cells in the LGN fire spontaneously in the absence of light. Flashes

of light of various wavelengths will tend to increase or decrease the rate of firing. DeValois classified the LGN cells according to whether the effect is excitatory, inhibitory, or both. Some cells showed excitation (or inhibition) to light of all visible wavelengths. These cells, which show uniform responses to all frequencies, are called the *non-opponent cells*. Other cells, which are called *spectrally opponent*, show excitation to some wavelengths and inhibition to others. This opponent nature of response is maintained over a large range of illumination, and therefore these cells are responsive to differences in color, not illumination. About 30 percent of the cells studied were of the non-opponent type. Of these, approximately half were excitatory and half inhibitory.

There are four types of spectrally opponent cells, which are classified by the wavelengths that produce maximum excitation and inhibition. One type of opponent cell shows peak excitation to light of about 630 nm and maximum inhibition to 500 nm light. Since 630 nm is normally perceived as red and 500 nm green, this cell is called (+R -G). About the same number of cells show the mirror response (maximum excitation at 500 nm and maximum inhibition at 630 nm) and are called the (+G -R) cells. The other two types of cells have maximum excitation and inhibition to light of 600 nm (yellow) and 400 nm (blue), and are called the (+B -Y) and (+Y -B) cells (see figure 2.10).

There is a large amount of interest (and very little agreement) on the matter of which cone types provide input to the various LGN cells. Most of the evidence indicates that the RG cells (+R -G and +G -R) receive inputs from the L (570 nm) and S (540 nm) cones, which were discussed earlier. One of these cone types is excitatory and the other inhibitory. For example, the (+R -G) cell receives excitatory input from the L cone and

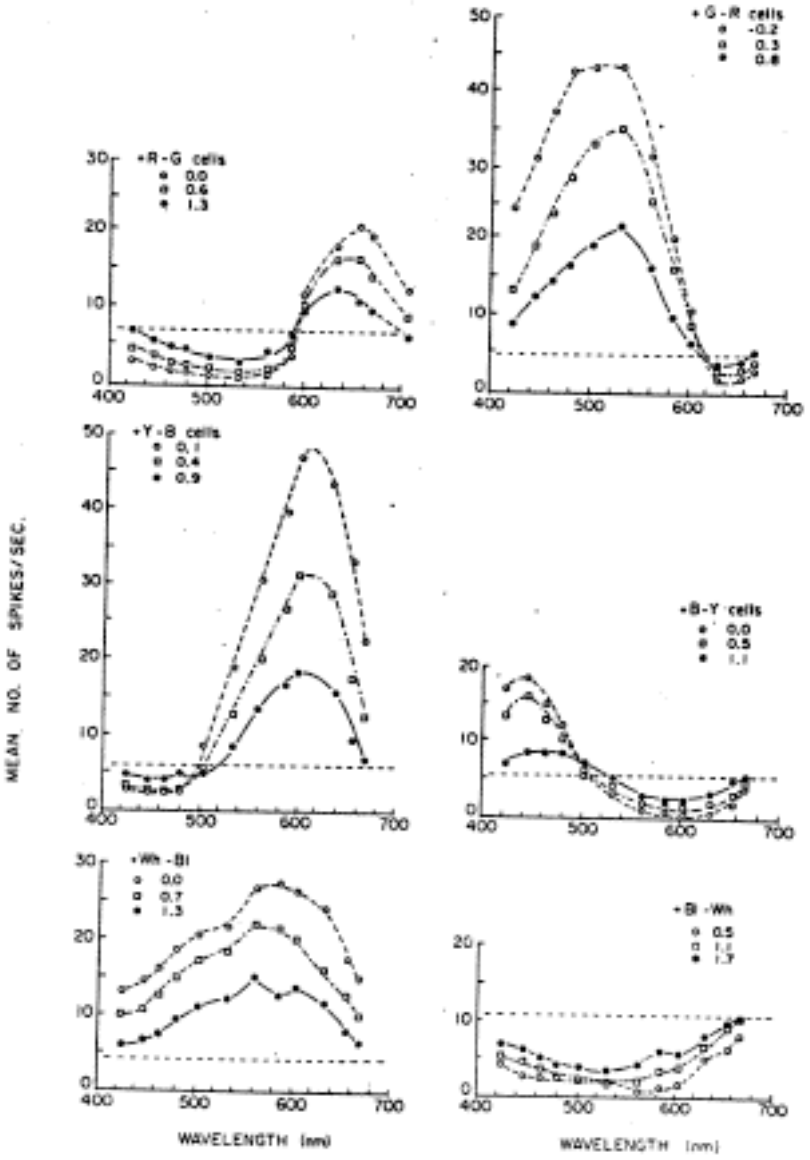

Figure 2.10 LGN cell responses (from DeValois 1973)

inhibitory input from the M cone. The response of the (+R -G) cell is thus dependent on the difference between the excitation from the L cone and the inhibition from the M cone. This means that the maximum excitation of the (+R -G) cell is not at 570 nm, where the L cone produces maximal excitation, but at 640 nm where the difference between the excitation of the L cone and the inhibition of the M cone is the greatest. Similarly, the peak inhibition of the (+R -G) opponent cell is not at the peak value of the M cone (540 nm), but

at 520 nm where the difference between the two cones is maximal. The effect of this opponent organization is to separate the points of maximal response of the cell, compared to those of the receptors.

There is general agreement that the YB cells (+Y -B and +B -Y) get input from the S cone. This leads to three different theories about the inputs to this cell: the S and M cones, the S and L cones, and all three cones. There is some evidence to support each of these theories, and it is not clear at this time which is correct. It is not immediately obvious that the non-opponent LGN cells must have multiple cone inputs. Evidence from the shape of the spectral sensitivity curve and chromatic adaptation experiments, however, indicate that these cells receive input from both the L and M cones (DeValois 1973).

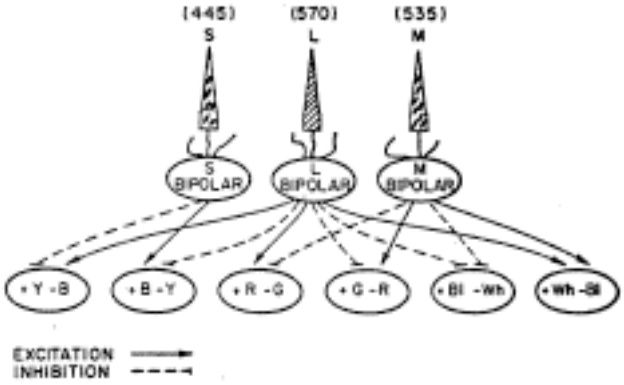

Figure 2.11 Cone inputs to the LGN cells (from DeValois 1973)

When the eye is dark adapted, many of the cells in the LGN, both opponent and non-opponent, have spectral sensitivity curves similar to that of rhodopsin. This indicates that the LGN cells related to the non-foveal part of the retina receive inputs from both rods and cones. This finding is consistent with the anatomical and physiological evidence about the retina, which was discussed in an earlier section of this chapter. This

rod-cone interaction could lead to tetrachromatic (four color) vision, and there is indeed some evidence that this happens in the periphery of the eye. The exact interactions between the rod and cone inputs to a LGN cell is not well understood at this time, and it is believed that the ganglion cell tends to minimize the extent to which rod and cone messages are simultaneously transmitted down the same channel (DeValois 1973).

The spatial organization of the receptive fields of the LGN cells is a center-surround field similar to those found in the retinal ganglion cells. The non-opponent LGN cells have either an excitatory center and inhibitory surround, or an inhibitory center and an excitatory surround, with the center dominant in both cases when the whole field is stimulated. The receptive fields of the opponent cells fall into two categories. The majority of the cells (77 percent) are of *Type 1*, which have excitatory input from one cone type in the central region and inhibition from another cone type in the surround, or vise versa. Most of these are RG cells, and all four possible combinations of field organization are found: red excitatory center and green inhibitory surround, etc. Only a few cells were found with blue excitatory center and yellow surround, and none with the reverse, or blue surround.

In the other kind of receptive field organization, *Type 2*, the inputs from the opposing cone types have identical spatial distributions. This means that at every point in the field, there is a balance between the excitatory influence of one cone type and the inhibitory influence of the other cone type. It is not clear whether the cones that project to a Type I cell actually lie in discrete areas of the retina. By chromatically adapting the eye, it has been demonstrated that the cone type in the surround is also present in the center, and is actually most sensitive in the central region of the field. One explanation of this

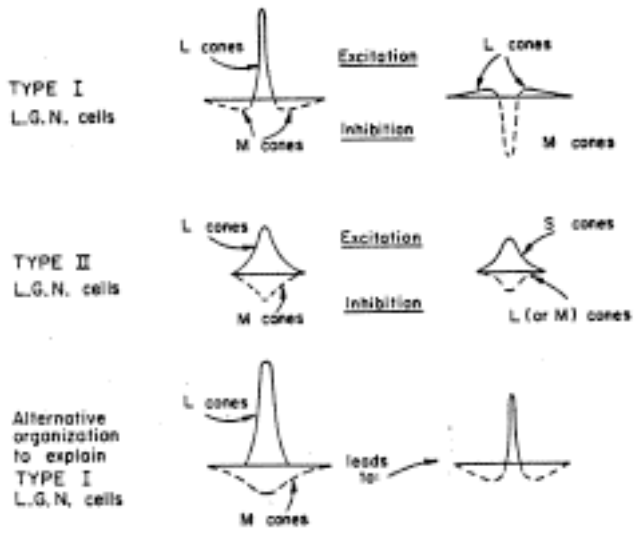

Figure 2.12 Field organization of LGN cells (from DeValois 1973)

phenomenon (shown in figure 2.12) is that both types of cones have their maximal sensitivity in the center, but that the central type cone has a much higher peak. This means that the receptive fields of both Type 1 and Type 2 receive inputs from cones over the entire field, but in the Type 1 field one of the cone types dominates the central region.

The Striate Cortex

The striate or visual cortex is the area of the brain that receives most of the visual information from the retina. It is composed of a large thin plate of gray matter which is arranged in a large number of folds. The plate is made up of a number of distinct layers. Fibers from the optic nerve project to areas of the cortex in a systematic topographical manner. The axons from the LGN synapse with the cells of the fourth layer of the striate cortex. The cells of the third and fifth layers send out fibers to other parts of

the brain. Most of the connections between cortical cells are in a direction perpendicular to the surface, and lateral connections are usually quite short (Hubel). This means that the retinal projection of an optic nerve fiber will affect only a small area of the cortex, and the cortical cells will normally have small retinal receptive fields.

Hubel and Wiesel have made extensive recording of cells in the striate cortex of cats. They have classified these cells into three groups: *simple*, *complex*, and *hypercomplex* (see Hubel). The simple cells react best to line stimuli (bright lines, dark lines, or boundaries) at particular orientations and positions in the visual field. For example, a particular simple cell may respond only to a dark line on a light background. It will fail to respond if the orientation of the line or its position is changed. The simple cells are distinguished from the other types of cells by the fact that their receptive fields can be divided into distinct regions of excitation and inhibition whose inputs are summed in a linear fashion (see figure 2.13). In other words, a bright spot in the excitatory region of the

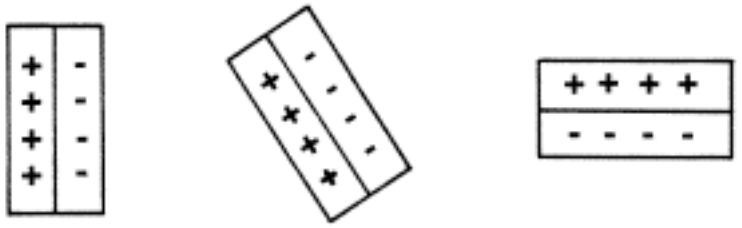

Figure 2.13 Simple cell receptive field organization

receptive field will produce the same responce as a similar spot in another part of the same region. Two spots of light in the same region will produce twice the response, and if one spot is placed in each region, there will be almost no response. The complex cells also respond to lines, bars and edges at particular orientations. Unlike simple cells they are not

as sensitive about the exact position of the line, and also respond with sustained firing to moving lines. The hypercomplex cells are similar to the complex cells, but respond only if the line is terminated in their receptive fields.

The cortical cells are functionally arranged in *columns* perpendicular to the surface. There are no visible separations between columns, but this term is useful because all the cells in a column respond to lines at the same orientation (see figure 2.14). It

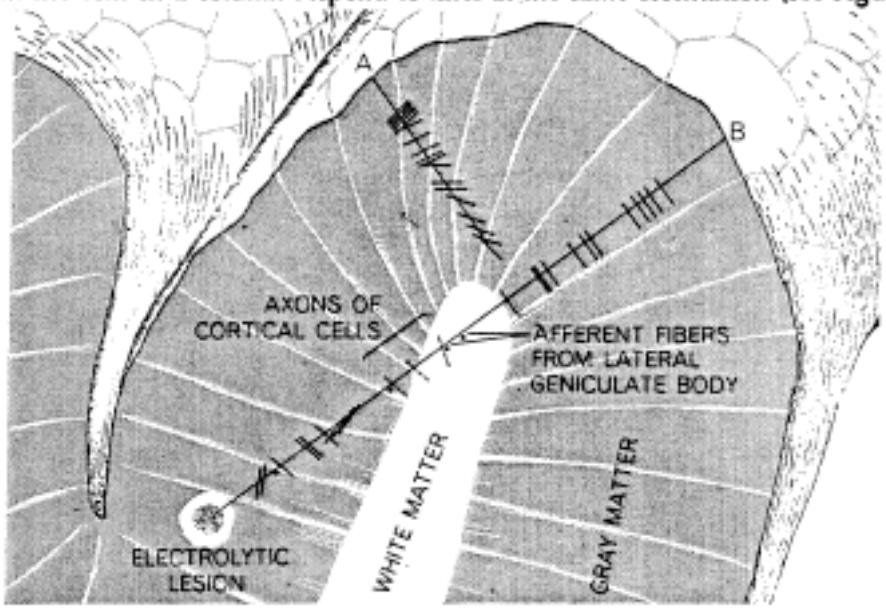

Figure 2.14 Columns (from Hubel)

is speculated that the inputs to the simple cells come from LGN cells whose receptive fields are aligned. This combination might form the line detector mechanism of this cell (see figure 2.15). It is further speculated that the complex cells of the cortex synapse with the simple cells in one column, which would explain their orientation sensitivity and independence of exact visual position (Hubel). It is also possible that the inputs to the simple and complex cells come from the projections of the two different kind of ganglion

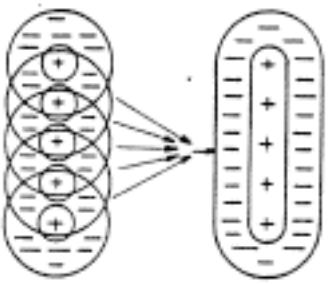

Figure 2.15 Proposed ganglion cell inputs to simple cells (from DeValois 1966)

cells that connect to the LGN: X-cells projecting to the simple cortical cells, and Y-cells to the complex ones. This would explain the difference in the response of these cells to moving stimuli.

The original work in this area was done on cats, and was not concerned with the spectral response characteristics of these cells. More recent studies with monkeys have found some cells of both the simple and complex type that are color sensitive (Zeki 1973). Hubel and Wiesel found that only about a quarter of the simple cells and 10 percent of the complex cells they studied in the macaque monkey seem to be concerned with color (DeValois 1973). The best stimuli for these cells were also lines, bars and borders, but the regions in these fields must be have specific colors. Mapping the receptive fields of cortical cells is a long and difficult process which often takes hours for each cell. Because of these technical problems, there has not yet been enough research done on the color properties of cortical cells to get a clear understanding of their nature, and the functions that color plays in their processing.

The Psychology of Color Vision

This chapter will deal with the behavioral studies of color vision and how they relate to the physiological data presented in the last chapter. These experiments use three general techniques. In some tests, the subject is a "null detector", and responds when various stimuli look the same. Color matching experiments fall into this category. In other tests, the subject is asked to respond to "just noticeable differences" (JND's). Wavelength discrimination experiments are of this type. In the third type of test, the subject is asked to describe his (or her) perceptions of various stimuli. This is how data on color naming is obtained.

Psychologists think of color in terms of *hue, saturation*, and *brightness*. Hue refers to the color name associated with a visual sensation. Saturation is a measure of the purity of a color, or its difference from white. Brightness refers to the perceived intensity of an object, and is often called *lightness*. All three of these factors vary with changes in the wavelength of a stimulus, and changes in the spatial organization of objects in the visual field.

I will begin this chapter with a discussion of trichromacy, color blindness, and some of the different theories which attempt to explain these phenomena. The following sections will deal with the three factors (hue, saturation, and brightness) defined above, and how they relate to some physiological data from monkey LGN cells.

Trichromacy and General Color Theories

The first major psychological experiment on color perception was performed three hundred years ago by Sir Isaac Newton. He discovered that projecting white light through a prism produces a spectrum of colors, which may be then combined to form white light by using a second prism. He also noticed that a color can be produced by combining various other colors together, and that there are an infinite number of such combinations, or *metamers*, which produce the same sensation. White light may be produced by combining certain pairs of wavelengths, called *complementary colors*, and there are an infinite number of such pairs. Newton also discovered that any color could be matched by adding together three other colors, which is known as the tri-color, or *trichromatic* theory of color vision. This theory was refined and put in essentially its present form by Young (1820) and Helmholtz (1867).

This means that if color perception is defined as "wavelength discrimination" in the complete sense of the word, we are all color blind. The existence of metamers is actually quite useful, since without it we would not have color printing or color television sets.

The retinas of some people contain only rods, and they therefore have only one visual pigment, rhodopsin. The spectral sensitivity curve for rhodopsin is roughly bell shaped with a peak around 500 nm. Given two lights of different wavelengths, it will always be possible to adjust their intensities to produce the same sensation. This conclusion is based on the assumption that the wavelength of a photon will determine the probability

that it will be absorbed by a pigment molecule, but that once absorbed it will produce the same effect on the receptive cell as any other photon. People with this type of vision are called *monochromats*, and are completely color blind. This is also why people with normal color vision can not discriminate colors in dim light.

If a person can match a light of any wavelength to a combination of two other light sources, he has *dichromatic* vision. Most color blind people fall into this category. The presence of two visual pigments is a necessary, but not sufficient condition for this type of vision. The spectral absorption curves of the two pigments must overlap, or it will be possible to find two primary wavelengths of light that will not match an arbitrary third wavelength (see figure 5.1). Also, if the spectral absorption curves of the two pigments are

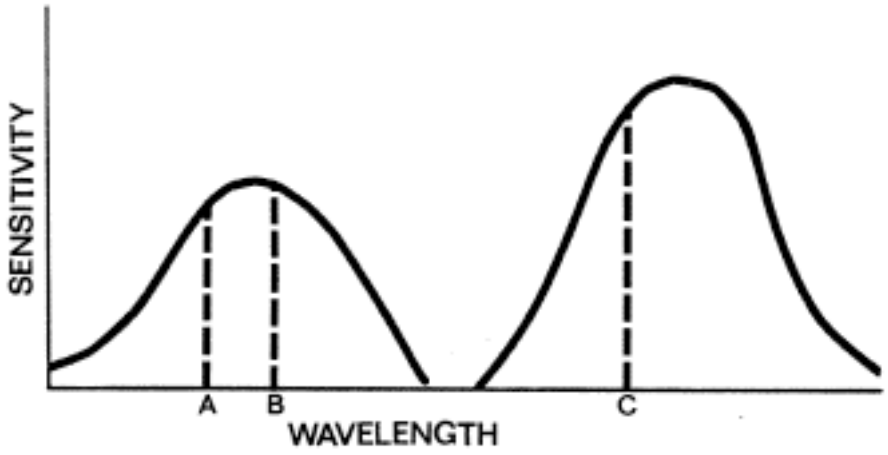

Figure 5.1 Hypothetical spectral absorption curves for a two pigment monochromat. Notice that no combination of A and B will produce the same response as C. If a person had these pigments, he would be able to distinguish between two lights if each one stimulated one of the pigments. There are, however, no known cases of this type of vision.

multiples of each other, the person will have monochromatic, not dichromatic vision.

Assuming that these conditions are met, the determination of the intensities necessary to

match two lights of wavelengths A and B to a third light of wavelength C, reduces to finding the solution to two equations in two unknowns (the intensities of the two lights). This solution may have a negative value. For example, if the intensity of light B is negative, this means that adding that amount of light to C will produce a match with light A.

A person with normal color vision is able to discriminate the difference between two patches of light when one of them contains one wavelength and the other a mixture of two wavelengths, regardless of their intensities (assuming the lights are bright enough to stimulate the cones). Given four light sources of various wavelengths which can be mixed in two patches, a person with normal color vision will be able to adjust three of the intensities to produce a perfect match between the patches. This is called *trichromatic* vision. This is the basis of the trichromatic theory of color vision which states that there are three different visual pigments and three "color channels" involved in the perception of color.

The theory in its simplest form states that the color of an object is determined by comparing the outputs of these three channels. The advantages of the trichromatic theory are that it is simple and agrees with our present understanding of the pigments in the retina. It also provides a plausible explanation of the matching experiments described above. The disadvantages of this theory are that it fails to explain color constancy, color contrast and several other major perceptual phenomena involved in color vision. Color constancy refers to the fact that the colors we perceive remain roughly constant over a large variety of illuminations. In other words, the colors we see tend to

correspond to the surface properties of objects and not the light falling on them. Color contrast refers to changes in the perceived color of an area which are caused by the effects of its surround. These effects will be discussed in detail in the other sections of this chapter.

The other traditional theory of color perception is called the *opponent-process* theory, which was originally stated by Hering in 1875. In this theory there are also three types of receptors and three channels. The difference between the two theories, in their present form, lies in the nature of the channels. In the opponent-process theory, the channels convey information on three types of opponent activity: red-green, blue-yellow, and white-black (see Hurvich and Jameson). This agrees with the evidence from the LGN cells, and will be developed further in the next section.

Both of the traditional theories assume that the color that is perceived at any point in the visual field is based on the spectral properties of the light striking the retinal image of that point. Color constancy and contrast effects are treated as anomalies. The colors we see correspond to the reflectance of the surface of an object, and yet there is no way for the eye to sense this directly. The light incident on the eye is the product of the intensity of the light illuminating an object and the reflectance of the surface of the object. These two components of image intensity differ in their spatial distributions. The incident illumination will normally vary smoothly over an image, and the reflectance will change sharply at the boundaries between objects and remain relatively constant between such edges. This means that the contrast across an edge in the visual field is proportional to the ratio of the reflectances of the objects adjacent to the edge.

Edwin Land has proposed another theory of color perception, the *retinex model*, which is based on a process of removing the effects of variations in the illumination component of the intensity (Land 1959). He proposes three sets of sensors and three channels (or retinexes). The output of each set of sensors is processed independently to remove the component of intensity due to illumination gradients. The result of this processing, which is called *lightness*, is conjectured to correspond closely to the reflectance of the objects in the visual field. The color of an area is found by comparing the output of the three channels, and is therefore no longer dependent of the spectral properties of light incident on the retina. There have been several methods proposed for the computation of lightness (Land 1971, Horn 1973b), but they are all based on the assumption that the major discontinuities in reflectance occur at the edges between objects in the visual field. These methods are designed to work on a special type of image, called a *Mondrian* (named after the Dutch painter Pieter Cornelis Mondrian), which has large areas of uniform matte color and sharp boundaries between the areas. This computation of lightness seems plausible, since (as we saw in the last chapter) the information in the later stages of the visual pathway seems to be in the form of effects across edges, and not absolute values at each point in the visual field.

Hue

In this section I will discuss three types of experimental phenomena that deal with the relationship between hue and wavelength: color constancy, color contrast and hue shifts. Perhaps the most amazing feature of the human visual system is its ability to

deal with large variations in illumination. The hue of an object remains relatively constant with large changes in the spatial and spectral qualities of the light illuminating it, which is known as color constancy. Land developed a very simple experiment for testing this phenomenon (Land and McCann). He illuminated a picture made of pieces of colored paper with a set of chromatic light sources. The light reflected from each piece of paper is just a combination of light at the wavelengths of the light sources, where the amount of light at each wavelength is determined by the pigments on the surface of the paper. In other words, if the only light source is "red" light, the only light reflected off the paper will be some amount of red light. This means that by varying the intensities of the light sources, he was able to change the spectral properties of the reflected light. The observed hue of each of the pieces of paper remains almost constant even with large changes in the intensities of each of the light sources. By using a photometer and filters, Land was able to measure the amount of light at any wavelength reflected off any part of the picture. The filters used in these experiments have the same spectral properties as the cone pigments in the human retina. He measured the light reflected from a white piece of paper, and was then able to change the intensities of the light sources so that a piece of paper that was perceived as red had the same photometer readings through the filters. The hue of the paper was still red, even though it was reflecting the same light that looked "white" earlier. This contradicts the simple form of the trichromatic color theory, which says that white light is white light, and should never look red.

Land then set up a red light at one side of the picture and a green light at the other side. By using this side lighting, he was able to produce large intensity

gradients of each type of light across the picture. There now was a red area (on the side of the picture near the green light) which produced the same photometer readings through the filters as a green area (on the red side). This also contradicts the trichromatic theory since these two areas are producing the same local stimulus to the receptors in the eye and should therefore look the same. The opponent-process theory provides no help since is also assumes an equivalence between color and local spectral properties. The retinex theory explains this phenomenon since it removes the effects of the intensity gradients of the red and green lights.

There are two types of color contrast effects: *simultaneous* and *successive* contrast. Simultaneous contrast refers to changes in the hue of an area caused by the effects of the region surrounding it. For example, a small gray spot on a red background will be perceived as green. The change in hue due to simultaneous contrast is in the direction of the color which is complementary to the hue of the surround. If the surrounding region is small in relation to the central area, the hue change may be in the direction of the color of the surround, which is known as *simultaneous similitude*. One of the standard demonstrations of simultaneous contrast is the *colored shadows* experiment. Two projectors are aimed at a white wall. A filter is placed in front of one, so that one projector emits white light and one red light. If an object is placed in front of the wall so that it casts two shadows, one shadow will be red and the other *green*. In the area of the wall corresponding to the green shadow the object is blocking out the light from the red projector, thus this area of the white wall must be reflecting only the light from the white projector. White light reflecting off a white wall should look white according to the traditional theories, but

the effect of the surrounding red light makes it look green.

Land modified this experiment by placing black and white negatives in front of the two projectors. If the negatives are properly made and aligned, a whole gamut of colors appears, which is known as the *Land effect*. Each area of the picture is reflecting a combination of red and white light, so according to the traditional theories the only hues seen should be shades of red. The intensities of each projector may be varied over a large range without affecting the perceived hues. This effect can be produced by various combinations of wavelengths. In fact, Land was able to produce the whole range of hues with two monochromatic light sources that are normally considered yellow: 579 nm and 599 nm (see Land 1959). That means that the only wavelengths of light reflected off the objects were 579 and 599 nm, but all the hues appeared. Traditional color theorists have tried to explain this effect in terms of simultaneous color contrast and color mixing, ie: red produces green from contrast, and red and green mix to form yellow, which can produce blue via contrast, etc.

Successive contrast is what is normally known as color afterimages. If you stare at a red light for a minute or so and then look at a white piece of paper, a green afterimage will appear. A familiar demonstration of this phenomenon is the green, black and yellow picture which produces an afterimage of the American flag. The traditional explanation of this effect is in terms of chromatic adaptation. In other words the red light changes the state of the red cone system, making it less efficient in the local area that receives the red light. When you look at the white piece of paper, that area will appear green because the output from the green system will be stronger than the red one. In terms

of the trichromatic theory, it is difficult to explain why the color seen is always the complementary one. The opponent-process model is perhaps better since it says that the adaptation will effect the opponent color channels, so that red changes to green and blue changes to yellow. The color seen will often change back to the original color after a while, which is called *successive similitude*, and it is possible to see several such successive reversals.

The perceived hue of a monochromatic light will change with variations in its intensity, which is known as the *Bezold-Brucke* phenomenon. Long wavelength light, for example, appears red at low intensities and appears more yellowish as the intensity of the light is increased. Lights of wavelengths greater than 577 nm or between 475 and 508 nm appear to decrease in wavelength with increases in intensity; and those between 508 and 577 nm or less than 475 nm appear to increase in wavelength (Brindley p. 145). In other words, the hue of a monochromatic light will appear to go towards the hue of 475 or 575 nm light. These two wavelengths correspond to the crossover points between the inhibition and excitation of the RG and BY cells in the LGN (DeValois 1973).

DeValois has made extensive comparisons between the electrical responses of the LGN cells and psychological tests on hue discrimination. A subject is shown a flash of monochromatic light and asked to describe its hue with one of four color names: red, yellow, green or blue. DeValois measured the relative activity rate of the four LGN opponent cells to flashes of light at various wavelengths, and compared that data to the color names chosen in the psychological experiment (see figure 5.2). The similarities in the response curves indicate that the color name used for this type of stimulus is closely related to the relative response of the opponent type cells in the LGN.

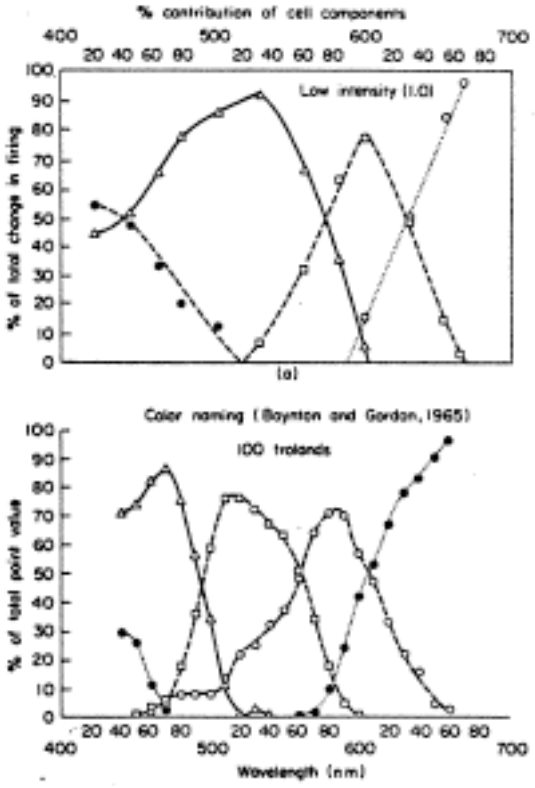

Figure 5.2 A comparison of the LGN cell responses to color naming (from DeValois 1973)

Saturation

The most saturated (or purest) light of any given hue is monochromatic light. Combinations of various wavelengths of monochromatic light will never produce a more saturated color than a monochromatic (or spectral) hue. The saturation of a spectral

color varies with the wavelength of the stimulus. Behavioral data on saturation sensitivity is based on matching experiments. These tests show that long and short wavelength lights appear to be much more saturated than light from the middle of visible spectrum. Yellow light (around 570 nm) appears least saturated. DeValois has speculated that saturation is measured by comparing the outputs of the opponent and non-opponent LGN cells. Adding white light to a stimulus will affect the non-opponent cells much more than the opponent cells. Figure 5.3 shows a comparison between the behavior saturation curve and the ratio of

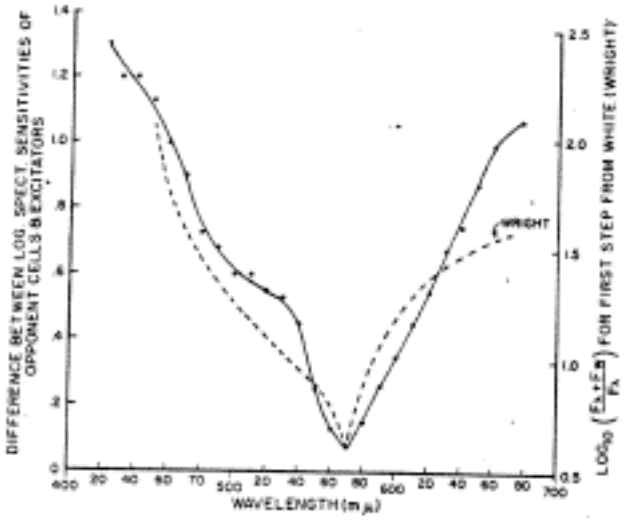

Figure 5.3 A comparison of the human saturation function (solid line) and the difference between the opponent and non-opponent LGN cell responses (from DeValois 1966)

activity of the opponent and non-opponent cells. The close correlation of these curves

indicates that the non-opponent cells are signalling brightness information and the opponent cells, color information.

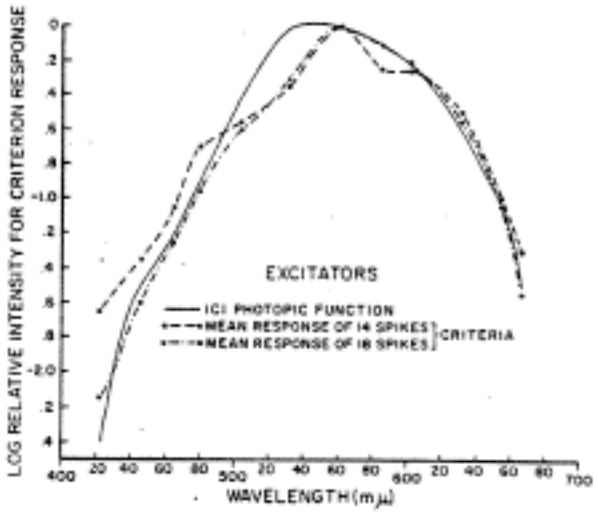

Figure 5.4 Spectral sensititivity of non-opponent LGN cells plotted against the CIE photopic luminosity curve (from DeValois 1965)

Brightness

The third factor in color perception, brightness, also varies with the wavelength of the stimulus. Flashes of long and short wavelength light do not appear as bright as light from the yellow-green part of the spectrum. There have been many

behavioral demonstrations of this phenomenon. The relationship between wavelength and apparent brightness of a flash (of bright light) is known as the photopic luminosity curve. DeValois compared this curve to the spectral sensitivity curve of the non-opponent cells in the LGN (see figure 5.4).

The close fit of these curves is another indication that these non-opponent cells signal brightness, not color.

The stimuli in these experiments were brief flashes of monochromatic light. The brightness of an area of the visual field depends not only on the wavelength and intensity of the light hitting the retina, but also to a large extent on the intensities of the regions around it. A dark background will make a region look lighter than the same region on a light background.

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