Objectives

1. Explain how neurons within the visual cortex become tuned to a visual edge of a particular orientation.
2. Predict the long-term deficit that can result from a cloudy lens in one eye of a newborn. Specify how this is different from the deficit in a newborn who exhibits strabismus.
3. Specify where in the visual pathway the signal from the two eyes first comes together. Specify what these neurons do that the preceding neurons cannot.
4. List the feature channels that the primary visual cortex signals to the higher-order areas. Specify from where in the primary visual cortex these channels arise.
Introduction

In Figure 2.1 we see that visual stimuli activate a structure called primary visual cortex. The activation is around an important fold in the cortex called the calcarine sulcus.

This activation, on the right side of the brain, only occurs when a visual stimulus appears on the subject's left.

Also, when this stimulus is above the subject, an area below the calcarine is activated. Conversely, when the stimulus is below the subject, an area above the calcarine is activated.

In this session we will learn what cells in this area do with the signals they receive from the eye.

But first let’s see how the signal gets there from the eye.

Figure 2.1  The Primary Visual Cortex (VI). V1 is centered on the calcarine sulcus (red dashed). A visual target above and to the left activate an area below the calcarine in the right cortex.
The Ganglion Cells’ Projections

Images seen in the right or left visual field are processed by the opposite side of the visual cortex. To do this, the ganglion cells on the medial side of each eye, from the middle of the fovea on, cross at the optic chiasm (green lines in Figure 2.2).

**P (small) ganglion cells,** primarily from the fovea, code the object’s details and project to a part of the thalamus called the **lateral geniculate nucleus (LGN).**

**M (large) ganglion cells,** primarily from the peripheral retina, code where objects are and project both to the LGN and several structures in the brainstem, including the **superior colliculus (SC).**

The SC causes the eye and head to turn to the location of a visual object that draws your attention: the **visual grasp reflex**. This in turn points the fovea at the object. Now foveal P cells can inform the cortex about the object’s details.

In the LGN, the two eyes maintain their own separate representations in different layers. The LGN sends information to the visual cortex: information as to **what** an object is (P cells) and **where** it is (M cells).

### How does a ganglion cell connect to the correct place in the LGN?

Ganglion cell axons grow to specific locations within each of the LGN’s 5 layers. Neighbouring cells grow to neighbouring locations in the LGN.

**Step 1:** The eye develops a chemical gradient based on its location in the head (e.g. nasal vs. temporal)

**Step 2:** Ganglion cells are given a location identity by the position specific chemical gradient in the eye.

**Step 3:** A similar gradient is set up in the LGN.

**Step 4:** Axons from the medial (nasal) retina are guided by this “scent” to the correct location in the contralateral LGN.

**Step 5:** Some time later, axons from the lateral (temporal)
retina are guided to the ipsilateral LGN.

Thus each part of the retina projects by way of its own ganglion cells to a specific location in the LGN. A similar process maps the LGN onto the visual cortex.

**What don’t we know about the LGN?**

**Things We Don’t Know**

1. Why there are so many layers in the LGN? Two of the P cell layers appear to be redundant.
2. 80 to 90% of the input is not from the retina but from the primary visual cortex and surrounding areas. What does this input do?
3. The receptive fields of LGN neurons are the same as those of ganglion cells. Thus the LGN does not appear to further process visual information. If so, why have the synapse? Why not send information from the ganglion cells directly to the cortex?

Understanding the LGN is important because the LGN is part of the thalamus and the other sensory modalities also synapse in the thalamus prior to projecting to the cortex. But what the thalamus does is also largely a mystery.
Primary Visual Cortex (area V1)

The LGN projects to the primary visual cortex located at the back of the head, mostly on the medial (inside) side. The primary visual cortex has many names: V1, area 17, and striate cortex. V1 is called the striate cortex because of a very thick layer 4c produced by the massive input from the LGN.

V1, like every cortical area, is made up of a thin sheet of grey matter at the cortical surface and contains 6 layers. To pack lots of grey matter into a skull, this sheet is folded. Having lots of grey matter is important because this is where the neurons and their connections are and thus where memories are formed. About half of V1 represents the details from the fovea.

Below the grey matter lies the white matter. White matter contains the nerve fibers that interconnect the cells in the grey matter.

V1 contains several types of cells:

1. **Layer 4c cells** have receptive fields that are the same as that of LGN and ganglion cell.

2. **Simple cells** with elongated receptive fields which make them maximally sensitive to a line or edge of a particular orientation at a particular location of the retina.

3. **Complex cells** whose receptive fields are similar to those of simple cells, except the line can lie over a larger area of the retina (positional invariance). Some are more sensitive to a moving stimulus.

How are the different receptive fields produced?

**Simple cells**: Several ganglion cells, whose receptive fields lie along a common line, converge by way of the LGN onto a simple cell.

**Complex cells**: Several simple cells of the same orientation converge onto a complex cell.

Other more complex types have also been found. One is the **end stopped complex cell** (also called the hyper-complex cell). Their receptive fields are similar to complex cells except that they are maximally activated by lines of a particular length. The activity is less for both longer and shorter lines. Still other end stopped complex cells fire when a line ends in their receptive field.
Why is this important and clinically relevant?

These studies provide a hint of how the visual system might construct complex representations, of for example a face, using the brain's building block, the receptive field. The simple cell is tuned to a very particular stimulus, a line of a particular orientation. The complex cell then generalizes this over a larger area. Similarly, we will see that cells in the inferior temporal lobe respond only to particular faces that are generalized over the whole of the retina.

The discovery of simple and complex cells is the work of David Hubel, a medical student from McGill who grew up in Windsor, Canada and who together with Torsten Wiesel was awarded the 1981 Nobel Prize in Physiology or Medicine. As we will see in a moment, they showed how changes in the organization of these cells can lead to a form of blindness called amblyopia.

Blind Spots

Patients with damage to their fovea often experience the following. When viewing a face against a striped background, they do not see the face (because it is in their blind area) but see stripes where the face should be. How can one explain this perception of stripes in terms of what you know of visual cortical cells?

No cells in the cortex are activated within the eye’s blind spot because there is no input from the retina here. But end stopped complex cells outside the blind spot become activated by the end of lines. The firing of these cells elicits the percept of a line even within the blind spot.

Your visual system is producing a similar filling in even as you read this. You are not aware of a blank black area in your vision about the size of that cast by the moon. The blind spot is where the optic nerve leaves the back of the eye. Your thumb nail at arm’s length is the size of your fovea. Your blind spot is twice the size of your fovea.

To find your blind spot close your left eye and look at some object on a blank wall. Hold a pencil, preferably with a red tip, at arm’s length and at eye level. Move the pencil to the right. At about 20 deg. the tip should disappear. Now raise the pencil. The tip of the pencil should reappear and there is no empty gap in the pencil where the blind spot was. Your visual system fills in the image of the pencil.
You can also look at the red x while paying attention to the dot on the right. Remember to close your left eye. It may be difficult at first to keep from taking a peek at the dot. In the first row when you move the page/screen between 1 and 2 feet away from you the dot should disappear. In the second row, the gap in the line should disappear. In the third, the face should disappear but the lines should be visible where the face was. You should see what the patient with the blind spot in the retina saw.

Figure 2.8  What you can and cannot see because of your blind spot. Close your left eye and the stare at the x. Place the screen or the page at about arm length. Adjust this distance until the dot disappears. **Top:** When the dot is in your blind spot you should see only white, no dot. **Middle:** The break in the line should disappear. **Bottom:** The face should disappear but the lines behind it will be visible in your blind spot illustrating the impotence of end stopped complex cells.
More Neat Examples of the Brain “Making Things Up”

Figure 2. 9  The brain even fills in colors and complex textures. Top: When the blue center is in the blind spot and the surround is yellow, the brain fills your blind spot with yellow. Bottom: When the blue center is in the blind spot and the surround is a pattern, the brain fills your blind spot with the pattern. This illustrates an important principle: The brain often fills in information that is missing. It makes things up the best it can. So don’t believe all that you see! This filling in property of the brain also explains why damage to the peripheral retina in visual cortex often goes unnoticed.
The visual cortex actually improves on what the eye sees.

Figure 2. 10 How the visual cortex improves vision. Step back until all the dot pairs in the top row are seen as single dots. Look down at the row of lines. You should see no break in the line for the one on the far left. Pick the line to your left that first appears as a line pair. The difference between which rightmost pair of dots appears as one and the same for the line pairs is a measure of how much the simple cells in your primary visual cortex have improved your vision.

To see this for yourself look at the row of dots. Step back until the top row appears as a single dot. Look down at the corresponding line pair. You should still be able to detect the break in the lines. Ganglion cells see dots. Simple cells in the cortex see lines. By analysing line segments that encompass many ganglion cells, the visual cortex improves acuity. This is called hyper-acuity. The smallest line offset that you can detect is smaller than that of the smallest dot offset. Clinical letter charts test the acuity of both the eye and the hyper-acuity of the visual cortex. A problem with either causes impaired vision.

There are many more cells in the visual cortex than in the LGN. Why?

Cell A in the LGN shares its information with many simple cells in the visual cortex. By grouping different LGN neurons in simple cells, sensitivity to a variety of orientations can be achieved using only a small number of LGN neurons.

Each LGN neuron, e.g. A, sends a signal to many simple cells, each with different orientations. In this figure, cell A shares its information with 3 simple cells. If there were a simple cell for each 5 deg. change in orientation, the same cell A would provide information to 36 simple cells (180 deg./5 deg. = 36).

Figure 2. 11 A single cell in the thalamus, A, projects to many cells in the visual cortex. Top: cell A projects to a cell with a horizontal orientation. Middle: cell A projects to another complex cell with a vertical orientation. Bottom: cell A projects to another complex cell with an oblique orientation.
The Columnar Organization of V1

V1 is composed of a grid (1 mm by 1mm) of what are called hypercolumns. Each hypercolumn analyses information from one small region of retina. V1 has a retinotopic representation. It forms a map of eye in your brain, with adjacent areas in the eye mapped to adjacent hypercolumns in the brain. But the map is distorted with the fovea having a very large representation. There are as many columns devoted to the fovea as there are to the rest of the retina.

Input from the left and right eyes (via the LGN) enters at layer 4c. Here, one finds monocular cells with circular surround receptive fields. As one moves to higher or lower layers, one finds binocular cells, first simple, and then complex.

Each hypercolumn extracts the following features:

1) **Stereopsis**: In each half, one or the other eye dominates. One sees in stereo by combining information from the two eyes in binocular cells located above and below the input layer 4c. These binocular cells detect disparity.

2) **Color**: In the center of each cube one finds a column, called a blob, running through all 6 layers except layer 4. The blob contains color sensitive double opponent cells with circular surround receptive fields. Thus each hypercolumn contains two blobs; one right eye dominant, the other left. These color sensitive cells in the blobs make up only 10% of the cells in the column and yet color seems to dominate vision.

3) **Orientation of line segments**: Radiating from the blobs, like spokes from the centre of a wheel, one finds simple and complex cells ordered into pinwheels of the same orientation. These cells are edge sensitive, but not color sensitive. The pinwheel arrangement allows cells with similar orientation sensitivities to be grouped together. This is an important organizing principle shared by the entire cortex. Neurons like to be near their own kind. It minimizes the length and number of axons.
The Effect of Visual Deprivation

At birth most simple and complex cells above and below layer 4c receive equal binocular input.

As V1 matures connections are lost and one side of the column becomes dominated by the left eye and the other by the right eye.

In newborns, each eye competes for representation in V1. If, at birth, vision is impaired in one eye, the impaired eye loses its connections while the good eye expands. This eye thus has a competitive advantage. Collaterals from this eye take over cortical representation normally occupied by the impaired eye.

This is called deprivation amblyopia: permanent cortical blindness in the impaired eye, which persists even if this eye regains normal function.

A cataract at birth, which is not removed until after one year, has a profound effect. A similar deprivation as an adult has little effect.

This early sensitivity in infants to thalamic competition from each eye for cortical representation is called the critical period.

Studies in kittens raised in the dark suggest that equal deprivation keeps the critical period dormant. When only one eye is deprived, a large shrinkage of the eye’s cortical representation can occur after only a week of deprivation. These effects are impossible to reverse.

The visual cortex however retains the capacity for other forms of plasticity, such as learning and recovery from damage throughout one’s lifetime.

Figure 2. 13 The Plasticity of the Visual Cortex Top: at birth cells above and below layer 4c are binocular. Middle: in the normal mature adult each side of the column has an ocular dominance. Bottom: if the right eye is deprived, the columnar representation of the good left eye expands.
Synaptic Mechanisms for Neural Plasticity

Molecular basis of the synaptic plasticity is synchronous activity. Synchronous activation of two or more synapses causes a strong depolarization. The key is the NMDA receptor (colored in blue in Figure 2.14) which opens only after the cell is strongly depolarized.

If two synapses fire synchronously, they strengthen each other at the expense of others that fire asynchronously.

**Cells that fire together wire together.**

This model has been used as a basis for plasticity or learning throughout the central nervous system.

The steps are:
1. Synchronous activation causes a strong depolarisation.
2. The NMDA receptor is activated allowing calcium (Ca) to enter the cell.
3. Postsynaptic nerve growth factor is released and taken up only by recently active presynaptic terminals.
4. These particular terminals enlarge at the expense of others.

Synapses are strengthened if the activity from several afferents occurs in the same time and is weakened if they occur at different times. Learning is the combination of forming memories and forgetting. The postsynaptic changes take time. The influx of Ca triggers a cascade of molecular processes some rapid but not long lasting, others taking days and are semi-permanent. If you have remembered these facts during your next test, a similar process must have occurred in some cortical area.

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**Figure 2.14** The Molecular Bases of Synaptic Plasticity
A synchronous input causes a large depolarization, opens NMDA channels, allows calcium to enter the cell, and releases a growth factor which strengthens recently activated synapses.

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**Figure 2.15** Synaptic Plasticity
A: synchronous activity from the left eye strengthens the synapses from the left side and weakens those from the right. B: synchronous activity from both eyes strengthens the synapses from both the left and the right eye.
How Competition Helps Align the Visual Maps of the Two Eyes

At birth many simple cells are activated by both eyes. However, this mapping is imprecise. Only those cells that are simultaneously activated by both eyes, that is, those that have the same retinal correspondence, will retain their connections. Thus the purpose of the critical period is not to produce amblyopia in some children, but to tune the acuity of the visual system.

It is amazing how well this is accomplished. A simple binocular cell is maximally activated by the same optimal line orientations in the two eyes. This means that a corresponding line of receptors on the retina of the two eyes must be wired to the same simple cell in the cortex.

Next let us look at what happens when this tuning does not work.

What happens in a child with strabismus?

When the two eyes are normal, but do not point in the same direction, one sees a double image.

Each eye is stimulated and thus retains its representation in the visual cortex. However, binocular cells are never activated simultaneously by the same stimulus. These cells eventually become monocularly driven and the child permanently loses stereopsis.

Because strabismus causes double vision, the image from one eye may be suppressed. This suppression may lead to amblyopia.
Stereopsis

Why are two eyes better than one? Look at Figure 2.18. Try to converge your eyes. At some point you should see three images. When this happens, the figure suddenly looks real with the bottom of the line appearing to come out of the page or screen. This is stereopsis. The right eye is centered on the image on the left and the left eye on the image on the right. Each eye sees a slightly different image of the line. The disparity of the images gives one the illusion of depth.

Figure 2.18  Stereo Vision or Stereopsis Try converging your eyes until you see three squares with one red line in them. In the middle figure, the line should appear to be coming out of the screen or page.

Figure 2.19 shows what the visual cortex “sees” when the images of the two eyes are combined. The bottom of the line appears in different places on the two eyes. This difference is called disparity. The disparity of these images gives one the illusion of depth.

If you looked at Figure 2.19 with a filter that only allowed red through the right eye and only blue through the left eye, the bottom should appear to be a line coming out of the page. The disparity of the top is the reverse and it should appear behind the page.

You can test this with a real line. Take a pencil and hold it by its middle about 10 cm on front of your face with its tip pointing straight up. Now tilt pencil so that its bottom tilts toward from your face and the top away from it. If you close one eye at a time, you should see the same tilt of the pencil as you see of the line in Figure 2.19. The bottom of the pencil should tilt to the right when viewed with the right eye and the opposite with the left eye.

Figure 2.19  A Binocular Cell’s View of a Line Which Tilts Out of the Page or Screen
This tilt produces a difference or disparity in each eye’s view.
At what level of the visual pathway is binocular disparity first analyzed?

Input from the two eyes first converges onto cells in V1 (above and below layer 4c) where about 70% are binocularly driven.

Each cell is only activated by a particular retinal disparity (Figure 2.19).

The 'far' neuron is activated when the images are displaced inward.

The 'in focus' cell is activated when there is no retinal disparity.

The 'close' neuron is activated when the images are displaced outward in the two retinas.

Figure 2.19 Activation of 3 Binocular Neurons in V1

A: The view from above of 3 objects and their projections on the two eyes. B: The view from behind on the two retinas. C: Each of the 3 neurons prefers a different retinal disparity.
In Summary

The eye flips the image of the world.

The retina distorts this image, magnifying the part falling on the fovea.

The images from the two eyes are combined in primary visual cortex. The left cortex codes images seen on one’s right side (by both eyes) and vice versa on the left. By comparing these two images, depth is computed.

Primary visual cortex separates the image into distinct feature channels. Different groups of cells work collectively to extract each feature.

1) Cells in the blobs extract color.

2) Binocular cells compute the retinal disparity and thus depth.

3) Simple and complex cell are activated by edges of particular orientations and their motion.

See problems and answers posted on

http://www.tutis.ca/Senses/L2VisualCortex/L2Problems.swf