PROFESSOR: So today, we are going to talk about the midget and parasol channels. I will begin by reminding you of what these two channels are that I very briefly talked about before. Namely, if you look at the Golgi stain of cross sections of the retina, when you look at just the ganglion cells, it was discovered initially by the inventors, if you will, of the Golgi stain, namely, Golgi himself and Cajal.

Cajal did this, initially. And then, subsequently, a fellow whose name is down here, Polyak, has used the same technique to study the retina. And what he discovered was-- not Polyak initially, but Cajal initially discovered, and then was verified by Polyak using these anatomical techniques, namely, the Golgi stain-- that there's one class of cells that have very small dendritic arbors, as you can see here, and another set that has much, much larger arbors.

Now, once this was discovered, subsequently when it became possible to record from individual cells and to study their receptive fields and their basic organization, and also to look at them from a different point of view, it was, first of all, shown that these are two very distinct classes of cells in terms of the dendritic arbors they create. And as I mentioned to you before, these cells are called parasol cells because the dendritic arbors look like an umbrella. And at comparable eccentricities, the midget cells are three times smaller in diameter than are the so-called parasol cells. And then I showed you some data the last time indicating that these two types of cells conduct at different velocities to the central nervous system, with these bigger cells having bigger axons conducting significantly faster than the midget cells, and that you get a distribution when you record from all the axons in the retinal geniculate pathway that they form separate populations because, initially, the argument was that maybe this is just a continuous population of cells.

But it turns out these are indeed two very distinct classes on the anatomical level as well as the conduction velocity level. Then when it became possible to record from single neurons and to study their receptive fields, it was shown-- I showed you this picture before as well-- that you have, first of all, center-surround organization as discovered by Kuffler; and secondly, that the midget and parasol cells are very different in size as far as the receptive fields are concerned as well as the dendritic arbors; and that the midget cells in central retina receive an input from just a single cone, whereas the parasol cells got a multiple input.

And another distinction that was, at that time, made is that the neuronal responses are such that midget cells respond in a much more sustained fashion when you activate the center mechanism than do the parasol cells. So these respond much more transiently.

So this, then, were initial cues as to what might be involved as to why we have these two systems. And there were all kinds of hypotheses. And then, eventually, in the '60s and '70s and especially in the '80s and '90s, all kinds of experiments were carried out to try to establish just why these two separate systems have evolved.

So now, when the anatomical work was further progressing, it was found, as I have mentioned also the last time to you, that each cone in the retina-- certainly true for the red and the green cones, which would be equivalent to the midget system-- gives rise to an ON and an OFF bipolar cell. Those are the ones we discussed the last time. What I did not discuss the last time, and I'm not going to discuss today either but will the next time we talk about color perception, is that the blue cones-- how many of you remember what is the frequency of blue cones in the retina?

AUDIENCE: 1 in 8.

PROFESSOR: 1 in 8. Very good. Excellent.

So that being the case, that already makes them different because the numerosity's so much lower. And then people have done all kinds of recordings, and to this day, it's not quite clear what the connection or pattern is. But as I said, next time, we will

talk about it in some detail when we talk about color.

But here, don't get frightened, we will discuss this the next time. But at any rate, we have the blue cones. And when we talk about color opponency that I will explain the next time, we think of the opponency to blue being yellow, and the opponency to red being green.

And so the assumption here was made that the connections in this case, somehow, must take place to create color opponency. And since we have only three kinds of cones in the retina, in the primate-- red, green, and blue-- the opponency here must, somehow, involve both the red and green cones. So that's the complication that we will discuss the next time.

And so the assumption was made that we have some so-called yellow/blue cells and blue/yellow cells. And so I just want to keep you puzzled as to what this means, especially what it means with respect to how we can see colors. And that's what we will talk about the next time.

So now, here we have, by contrast-- and we go back here-- here, this is, then, the midget system. And I showed you the two extremes, which are very clear, and in between, which would be somehow creating the blue system. So that's the midget system with very small receptive fields and very small cells.

Now, by contrast, when you look at the parasol system, what you see here is, as indicated by the picture I showed you just a minute ago, is that the receptive fields are bigger. The ganglion cell's a lot bigger. And the dendritic arbors are much more extensive. Now, that means, by the way-- I shouldn't even say "by the way"-- that means, very importantly, that the ON and OFF bipolar cells that connect with the ON and OFF cells of the parasol system are also much bigger and have much more extensive arbors, dendritic arbors, because they sample many cells instead of just one.

But overall, what this means is that when you look at the bipolar cells in the retina, there are about three times as many bipolar cells than there are photoreceptors.

Now, if you think about this-- somebody remembered the last time-- that in the retina, we have about 50 million cones, and we have about 120 to 150 rods. And so, then, if you think about the fact, at least, for the cone system, we have three times as many bipolar cells. This only applies fully to the cone system.

That still means that we have 150 million bipolar cells in the retina. So it's absolutely incredible, then, this tiny little thing that you have in your head, which is less than an inch in diameter, you have these millions and millions and millions of cells. Amazing. So this is then the arrangement for the parasol system.

And now, we can progress and make another point about how clever the wiring became in the course of evolution. The cleverness here is that, as you know, we only have a single layer of photoreceptors, which, outside the fovea, has a mix of rods and cones. But then if you look at the receptive fields of ganglion cells, what you find is that they have overlapping receptive fields for many of the attributes. So you have overlapping for ON and OFF, of course. And that's obvious from the wiring.

But you also have overlap for the midget and the parasol cells. And lastly, there's an overlap-- even if you just look at the rod input-- which results in realizing that the receptive fields become larger during dark adaptation by virtue of the connections of the rod photoreceptors to the rod bipolars than to the A, to amacrine cells, which then connect with the same ganglion cells as do the cones. So this is, then, an incredibly clever arrangement which enables you to see things extremely well. And it's an incredible feat of wiring that accomplish this incredible arrangement.

So now, another important thing to consider, in addition to what I've said so far about the midget and the parasol cells, is that they have distribution over space. If you look at it from, say, from the center of the eye, meaning the fovea, going to the periphery, the ratio of midget and parasol cells changes dramatically. In the center here, in the fovea, you have a huge difference in number of cells. But as you get to the periphery, these two types of cells eventually become equally numerous.

Now, to show this in relationship to the lateral geniculate nucleus-- again, to repeat,

in the fovea, you have an 8 to 1 ratio; in the periphery, they have 1 to 1 ratio. And this is directly reflected in the lateral geniculate nucleus. Everything I've shown you so far about the geniculate was a six-layered structure.

But those six layers exist only to about an 18-degree eccentricity from the fovea. And after that, the lateral geniculate nucleus becomes a four-layered structure. And as done here in a schematic fashion, the four layers for the left and right eyes are pretty much equal in numerosity. And that creates, then, the 1 to 1 ratio. So that makes one think, why do we have this shift?

There must be something very important in central vision for which the midget system is good for. And in peripheral retina, the parasol system becomes more important. So we are going to pay attention to that as we examine what the functions are of these two systems. So how do we go about finding out the functions of these two systems?

Well, there are several methods. The first one I'm going to tell you about is to record from individual neurons-- say, in the cortex-- and determine what kinds of inputs they get from the midget and the parasol cells. So to do that, you first want to look at the exact projections of the retinal ganglion cells again. Just to remind you, here's the lateral geniculate nucleus, again, in the region where there are six layers.

I already showed you this picture before. And I told you that the parvocellular layers project to 4C alpha; the magnocellular layers to 4C beta; and those other cells, which we'll talk about eventually, its lateral layers project to the upper parts of the cortex. So now, we have a good idea that even if the inputs to the visual cortex-- meaning, in this case, of course, the V1-- the two systems are separate in the input layers, in 4C alpha and beta.

And then the question comes up, well, what happens when you look at cells above and below the input layers? Do they converge? Or what's going on? So that's the kind of questions we're going to ask. And we are going to look at this more carefully here to find out just how do you do an experiment like that.

Well, one thing you can do here, you can use a reversible agent that you can inject either in the parvocellular or magnocellular layers of the geniculate. And if you do that, if in the parvocellular layers, this would be a blocking agent. Xylocaine is something that's used frequently. There are several other agents.

And so if you inject that substance in here, you render the cells in the geniculate unresponsive. It's to serve the same idea as what we talked about with APB in the last time, except this is not quite as neat because it's not a pure chemical treatment like you did with APB. Secondly, you can do it in the magnocellular layers as well and block that region.

Now, if you do this kind of experiment, let's first talk about what happens in area of V1. Now, this is a very difficult and complicated experiment. Sometimes, you can spend days recording from a single animal because, first of all, what you have to do is you have to put in electrodes into the lateral geniculate nucleus, which is way down, preferably into both regions, but there are many experiments just to one or the other. And then put an electrode into V1.

Now, the next important task, of course, is that you've got to record from V1 in the region to which these cells or these cells project. So to find that overlap in the receptive fields that you need to do when you record here, here, and here is that you have to take many, many electrode penetrations until, finally, you have an overlap. And once you have that overlap, you can be sure that when you inactivate this region by injecting the Xylocaine, you can assess what the responses are in the V1 before, during, and after the injection. And most importantly, before and during, naturally. So let's look at that.

This has been done studying many cells to get an overview of what's going on. And I'm going to show you, initially, just one example of a cell to give you a feel for what that's like. So here we have the cell respond.

What you do is you take a bar of light and move it across the receptive field-- brrp, brrp-- for each edge. So those are the two cumulative responses. [INAUDIBLE]. That's the normal.

Now, you inject into the parvocellular portions of the geniculate. And lo and behold, the cell keeps responding. Then you inject into the magno portions. And then lo and behold, the cell still keeps responding.

But when you inject into both-- bango! There's no response. So that means that this particular cell gets a convergent input from the midget and the parasol cells as they pass through the lateral geniculate nucleus.

Now, if one then does a systematic study and then records from many cells, what has been found here is that some cells do get an exclusive input from the midget cells. Some get an exclusive input from the parasol cells. And some are just like the one I've shown you here-- namely, they get a convergent input.

So that tells us, then, what the very, very basic nature is of the input to the visual cortex, you, to some degree, keep separate the two systems. And to some degree, you also have a condition where they are united. So that's what happens in area V1.

So now, the next thing you are going to ask is, well, what happens in other cortical areas? So here, then, I'm showing you the same method picture. But now, you have recording in V4 and MT, doing the similar kinds of injections that I just described in the lateral geniculate nucleus.

So when one does this, some interesting results had been obtained. And as always, before this kind of experiment that actually tested this question, there were, of course, hypotheses. Some people hypothesized that area of V4, which had at one time been proclaimed to be a color area-- and we'll come back to that shortly-- it was claimed, therefore, that this area gets input only from the midget system. And this area-- since it has motion-selective cells, if you remember-- only gets input from the parasol cells.

So, therefore, we can now have the acid test, thanks to some remarkable work that had been done, much of it by John Maunsell over at Harvard, who had actually worked here at MIT before then. And he did an experiment like that. And here's an example of a single cell recording from V4.

This is a magno block, and this is the parvo block. This is before the block, and this is after the block. So what you can see here, this cell, again, response to a bar moving across-- brrp, brrp-- like that. Here are the two responses shown here as to how they come across the receptive field.

And you can see a vigorous response after you do this and before you inject Xylocaine into the geniculate to block, in this case, the magno system; and in this case, the parvo system. This is backwards from the way I usually presented. I usually like to put parvo first. Preference-- it's the bias.

At any rate, you can see what happens is dramatic for this particular cell after you injected, you blocked the magno system. The cell-- we said has spontaneous activity-- but it no longer responds to the edges. By contrast, when you do a parvo block, you only get a small effect. There is a reduction for this particular cell, but it's still responding.

So that is the example of a recording in area of V4. Now, let us go and ask the question, what about MT? Now, here's an example. Same arrangement-- magno first, parvo second, before the block, after the block.

In this case, in MT, you get a cell that totally stopped responding after magno block to this moving stimulus. And here, what you see is after parvo block, the response continues. So now, this is just two cells.

Now, to be sure that these two cells are generally representative of these observations, what you need to do is to collect this kind of information from many cells, and then come up with a qualitative statement. So what you can do is you can, for each cell, when you do this experiment, you can't determine how much the cell fired here to the stimulus and how much it fired here. And then you can get a ratio of that response. You can turn it into, like, a percentage or maybe just to score them as 0 to 1 number. And then if you do that for a whole bunch of cells, what you find is shown here.

This is a bunch of cells in V4. This is a bunch of cells in MT. And what you can see

here is that in V4, first of all, you have a medium degree of blockage, nothing major, but some blockage.

But most importantly, you get blockage both for parvocellular and magnocellular inactivation. By contrast, in MT, what you find is that when you block magnocellular geniculate, most of the cells are dramatically affected. Only a few cells are blocked as a result of a parvocellular block. So the outcome of this is that the hypothesis pertaining to MT, that it gets mostly an input from the parasol system, is correct. But the idea that V4 gets an input only from the midget system is obviously incorrect, showing that it gets an input from both.

So to then summarize the wiring diagram here, what you have here is the eye, of course. And then you have the midget and parasol cells that project, respectively, to the parvocellular and magnocellular layers of the lateral geniculate nucleus, then they go off to the cortex. And if you remember, in the cortex, they terminated 4C alpha and beta.

And then, also, as I told you, up here, you already have many cells above and below the input layers that get a convergent input from the midget and parasol cells. So what happens then, in terms of the projections to higher cortical areas-- to talk about V2, for example-- you get some cells which are purely driven by the midget system, some cells purely driven by the parasol, and many cells that are driven by both. So V2 becomes very complicated because they have different subdivisions.

Maybe in V2-- we talked about it a little bit-- that may be receiving different input from area of V1 in terms of whether they are driven by the midget or the parasol cells. Now, most notably, when from V2 and from V1, you look at the projections to area MT, the middle temporal area, what you find is that this is heavily dominated by the input from the parasol cells. Then if you go beyond that, this continues to the parietal lobe from MT. But then, when you go from V2 to V4, then temporal, then the frontal lobe, what you find that there's a mix of inputs from both of these systems.

So it highlights the fact that, indeed, these things are quite complicated. And so we

need to now turn to a different method to try to ferret out beyond just establishing the connections as to what on earth these two systems are for. And needless to say, hypotheses were rampant about this. And what I'm going to do now, I'm going to tell you about how one can go about and how many investigators have gone about trying to determine what the functions are of these two systems in processing visual information. So how do you do that?

Well, the way you do that is you can use what is called lesion studies. What you can do is you can selectively block either the parvocellular or magnocellular systems at the level of lateral geniculate nucleus because, by lucky happenstance, the parvocellular layer is getting input from midget, and the magnocellular layer is from the from the parasol cells. So you can make lesions, then. How do you make lesions?

There are variety of ways of making lesions. That's a huge field. It applies not only to vision, to many, many other areas, to try to make carefully selective lesions in various parts of the brain. Now, it's not easy to do that in the geniculate. So what you have to do is, again, you go in to the lateral geniculate nucleus with a microelectrode.

Once you do that-- you already know the layout of the geniculate-- you can find out where the receptive fields are located, and you can determine whether you are recording from the parvocellular or magnocellular layers. You're going to adjust the depth of the electrode to be either in the parvocellular or magnocellular layers. And once you have established the receptive field location and were certain about in what parts of the geniculate you are recording, you can then proceed to make a lesion.

Now, there are a number of ways of making lesions. One of them is called heat lesions. You should take a metal microelectrode. You take it down there, and then you pass some current to make the tip of the electrode hot, you can affect, as you know, maybe a millimeter area or something like that in the lateral geniculate nucleus. I'm talking about small, small areas.

Another alternative that you can use is to inject a chemical. One of those commonly used is called Ibotenic acid, which is a very nice attribute, that when it causes a lesion, the borders are nicely clearly defined. So no matter how you do this, then once it is done in a monkey, then you can study a monkey for months on end to see what the vision is, of how the vision is affected. And then after you've done that, you then process the brain and look at the lateral geniculate nucleus to see what was the size and location of the lesion.

Now then, in some cases, it's "Oh, my god, you did both. Nyah, nyah, nyah." You screwed up. So months and months of work goes down the toilet. But in some cases, you get a good effect. And that, eventually, if you do it several times on several monkeys can result in a solid publication.

So that is the basic process for making the lesions. Now, let me then move on and broach the next important topic-- namely, what are you going to study? So what you have to study since you want to have an open mind and you don't want to say, "oh, yeah, everything in the brain does color" or something like that, and so instead of just studying color, you have to study many other aspects.

So I'm going to tell you about several kinds of tests that have the newest. And I will explain each of those to you. But first, I will list them.

The behavioral task that is used-- very important-- is that you've got to be able to confine the crucial stimulus either to the area that you had blocked or the area that is intact. So one way to do that, which is the easiest way, is to use a detection task. We already talked about that briefly.

The monkey first fixates. That confines his looking to that location. And then you can present a stimulus like this. And if he makes a saccade to it, he gets a drop of apple juice for a reward.

Now, that is a procedure. And then, of course, our next trial, it appears someplace else. On each trial, it's in different locations.

And on some trials, you can present the stimulus in the area that had been blocked. You get it from by magnocellular or parvocellular lesions or infusions or in an intact area. And that, then, enables you to compare performance in regions where the monkey's performance is normal because he's intact and in those regions where he lacks either an input from the magnocellular or from the parvocellular layers of the lateral geniculate nucleus, meaning from the midget and parasol cells.

So now, another task which is used to be able to carry out a more thorough examination of visual capacities is his discrimination. In which case, after fixation, you have a whole bunch of stimuli coming on. This is very effective for studying color.

And in this case, this discrimination task often is called the so-called oddities task. That's an easy way to remember it. "Oddity" because this is odd. All the others, the distractors, if you will, are the same. Only one stimulus is different.

And, of course, the monkey has to make a direct saccade to this stimulus to get a drop of apple juice for reward. So that would be a so-called discrimination task. So these are the two basic tasks that can be used. And now, we can proceed and ask the question of what exactly, what kinds of visual capacities should be studied?

So let me just say one more thing, by the way. The way this is done in a laboratory is that you have a performance monitor, so-called, where the computer puts a square around each of the dots that will have appeared-- in this case, this should be discrimination-- or where they will appear singly in the detection task. And if the monkey makes a saccade that lends the saccadic eye movement, which you are recording, into the square area there, then automatically a drop of apple juice is discharged. But if he were to make a similar saccade to here, he will not get rewarded because this is the only correct position.

And on each trial, that's going to be someplace else. Everybody understands the method? It's fairly straightforward.

So now, we can move on and look at an example of the kinds of lesions that this

can create. Here is a lateral geniculate nucleus again, the six-layered portion. And you can see this area here, that this is a lesion.

There are no cells functional here, which affects only the parvocellular layers. You see here the two magnocellular layers are normal. So this is the lesion area which affects a few degrees of visual angle-- in this case, in the lower part of the visual field.

Now, here we have a magno lesion. If you just look at this quickly, you say, what lesion are you talking about? But if you look closely, can you see this here?

There are no cells here. So this is the region where both layers, magnocellular layers that get the input from the parasol cells, had been blocked. So that is the block there.

So this is, then, an example of a successful lesion. And once you do these kinds of experiments-- some of these experiments can take a couple of years, maybe longer-- you end up having some monkeys who have neat lesions like this, then it's a control. Let me just add that, which I'm not going to show any pictures of.

But you can also make, on purpose, a lesion where you block both of them-- both the magno and the parvocellular layers. You just block out a portion of the lateral geniculate completely. Now, that's an important control. For all kinds of experiments, controls are essential.

Now, let me tell you-- this is available in the published material that you have been asked to read-- that when you block both magno and parvo, maybe a quarter of the lateral geniculate nucleus, whenever you present stimuli in the area that has a topographic correspondence to the lesion's region, the monkey cannot see a thing. He cannot perform any of those tasks at all. So clearly, whatever effect one gets here with these selective lesions can then be ascribed-- if you see deficit after this one or deficit after that one-- can be ascribed to what the midget and the parasol cell systems do.

So that, then, is the procedure. And now, we can move on and list the perceptual

functions that one wants to test. Now, think about it for a minute, just quickly. What kinds of functions would I want to test if I'm running this experiment? How can we break down the multitude of things that we have to process into some basic functions?

Well, first of all, very important function, even though it may have now [INAUDIBLE] is called contrast sensitivity. Because everything you look at, the light reflects just about from everything that you look at, and so the contrast of the stimuli that's on a white sheet of paper or a gray sheet of paper or when you look at photographs is highly varied. And the question is, how well can you see the different levels of contrast?

Another one, of course, that's obvious is color. How well can you process color information? And then another one is pattern. We often talk about basic patterns. And the one I'm going to tell you about mostly would be checkerboards or something like that.

Then we talk about texture. Most of the things that we encounter in the world are textured. And so it's important for us to be able to see texture.

Then, of course, shape, that's obvious. Then stereopsis. We will talk about stereopsis in much more detail in a bit. What stereopsis involves, as I've mentioned to you already, are the difference of the input in the two eyes. And that difference is called disparity, which the brain then interprets as depth.

Another one, very important because you often just see things appearing quickly in very brief times, is to study flicker. And yet another one is to study motion because that's so central to our existence. And then to study brightness. And one thing I haven't mentioned yet is scotopic vision. How well can you see on the photopic and scotopic conditions?

So those are, then, the procedures. And so let us now look at the first one of these-- contrast sensitivity. This has been extensively studied, hundreds and hundreds of papers, many of them in humans. And most commonly in these papers, what they

did was they used sinusoidal gratings.

And the way you do that, then, in similar experiments as a detection experiment, you present sinusoidal gratings with spatial frequency and whose contrast you systematically vary. And when you do that, you get what is called a contrast sensitivity function. And so the most common one that you read in many papers is plotted this way.

This is spatial frequency low, high. And this is contrast. Low, high. And then if you do that, then you systematically studied this in humans, interestingly enough, you get a function like that-- meaning that in between levels of spatial frequency, you see the best. And extreme levels, you don't see quite as well.

So one could use sinusoidal gratings in animals. But sinusoidal gratings aren't essential. What you can do instead is you can present a checkerboard.

And so here's an example, same procedure as before. The monkey sees this and makes a saccade to it. It gets you water. And then you vary the spatial frequency and the contrast of it. So here is one.

You can hardly see that because the contrast is low. So now, to see this overall, here's an example. Here, we vary the contrast. And here, we vary the spatial frequency.

And if you look at that, you can see that in this region, depending on how far back you are, this region, you can see the best. Here, it will be less so. And here, of course, it drops off dramatically, just like that curve I have drawn there.

So this, then, enables one to generate a so-called "contrast sensitivity" function in a monkey in those regions of the visual field that are intact and in those regions of visual field in which you have either magnocellular or parvocellular lesion that selectively blocks the parasol and the midget systems. So that's the experiment, then, for just studying contrast sensitivity. And if you do that, this is the kind of result you get.

This is the monkey's normal performance. In this case, of four spatial frequency levels. And you go up and down with contrast, just like that curve I drew there. So this is your contrast sensitivity function. And it shows that under normal conditions and after magnocellular lesion that blocks the parasol system, there is no effect, meaning that the parasol system doesn't seem to be too important for contrast sensitivity.

By contrast, there's a huge effect, especially at high spatial frequencies, after a parvocellular lesion that blocks the midget system. So that, in essence then, is what happens with contrast sensitivity. Now, let us move on and say, well, what about color vision?

So how do you do the color vision? I already told you that before. What you do is you present, in this case, eight stimuli, one of which is different from the others, and just have red and green ones.

This is the odd one. The monkey makes a saccade to it. He gets a drop of apple juice for a reward.

Now, if you want to be systematic about it, you can vary the degree of color contrast. But the effect is so dramatic that it was not necessary to do that. So let me show you what the effect was.

Here, we have a monkey's normal performance when the test element is blue, red, and green. This happens after a parvocellular lesion, meaning when you block the midget system, the monkey cannot see colors at all. Just a total loss. Whereas, after magnocellular lesion, his performance is indistinguishable from normal. So this, then, establishes the fact that color vision is controlled by the midget system.

Now, the fact that this is the case perhaps is not that surprising because I told you that when you look at the cells in the midget system in central vision, that most of them get an input from just-- at least, the red and green ones-- get an input from a single cone. So just looking at the receptive field organization tells you that that system is likely to be very important for color processing. So that, then, is confirmed

by this kind behavioral experiment.

So now, let us look at brightness perception. Now, how is that different from contrast sensitivity? It's different because, in this case, what to do then is you use a discrimination task like this.

And I think most of you can tell that this one is brighter than the others. Purposefully, I made that a small difference, so you can appreciate the fact that on each trial, we can vary the difference between the distractors and the target. And you can generate a curve seeing how much brightness difference do you need to be able to perceive a brightness difference. So if you do that kind of experiment, one is in for a big surprise.

This shows here what happens after a parvocellular lesion. And this shows here what happens after a magnocellular lesion. So when you block the midget system, the performance is unaffected as is the case with a magno lesion, meaning that if you, obviously, that both the midget system and the parasol system process information about brightness, at least, at these low spatial frequencies that we have used that I just showed you.

So now, the other surprise was that, then, the question became, well, what if you do this not under photopic conditions and do under scotopic conditions? And again, there's no effect, meaning that the unique inputs from the rods and the unique inputs from the cones must go into both of the systems. Now, the reason that was surprising is because a couple of papers have been published, maybe about 15 to 20 years ago, that claimed that the rods feed selectively into the parasol system, not into the midget system.

So this totally disprove that. And then subsequently, careful anatomical experiments also established that both the small and the large cells, magno ganglion cells, receive convergent input from the rods and the cones as I had diagrammed to you in the previous session. So then, let's go on and look at pattern and texture perception.

In this case, let me show you the kind of experiment that's done. This is when you look at patterns. One way to do it, go back to those same checkerboards, but make them high contrast and have one at a different spatial frequency than the others. And then you can systematically vary the degree of spatial frequency difference between the targets and the distractors.

So that would be the method that is used to study this kind of pattern perception. The other one is to look at textures. And in this case, those of you in the back probably can't see this. But those of you up front, can you see this little area here, where the diagonal lines are reversed?

So what happens is, first, you just present this whole thing. And then you present the reverse patterns and the surround. And when you do that, the monkey has to make a saccade to that location and will get a drop of apple juice again for that performance. And then on each trial, this appears somewhere else in one of the four to eight locations in the display. So that's the procedure.

And then if you do this, you, again, luckily get a very dramatic effect, apparently. Here it is. We have here normal performance. Here's a parvocellular lesion, magnocellular lesion. This is texture, and this is pattern.

Pattern tasks was, overall, much more difficult than the texture one in this case. Well, what you see here that is really dramatic is that when you block the midget system, there's a tremendous loss in your ability to see patterns and in your ability to see textures. So fine vision for detail seems to be central for the processing of the midget system. So that, then, is the effect that one gets with texture and with pattern.

So now, let's move on and talk about stereoscopic depth perception. It's a topic we are going to look at in more detail in a later session. Let me now introduce this, first of all, by telling you that stereoscopic depth perception resulted predominately by virtue of the fact that the eye is moving to the front so that there was a major binocular overlap.

And if you talk about a monkey, for example, or even many, many animals that do have stereoscopic vision, here's an example of looking at a tree when just about the only cue you have here would be based on stereopsis because all of them are equally dark. Which branch is in front of which one? And it's very hard to tell. If your monkey will jump from this branch to this branch, if he can't tell where they are relative to each other, the monkey is going to fall down and drop dread.

So it is very important for monkeys to have a highly functional stereoscopic system, which they do. And so, how do we study this? Well, the way we study this is to use what is called a random-dot stereogram.

Random-dot stereograms were created once computers became a reality many, many years back by Bela Julesz. And he came up with the idea that if you use this kinds of random-dot stereograms, there's no other depth cue. So, in other words, you can study just the stereoscopic depth perception aspect of it.

Now, why is this? Because what you do here is you look at this two displays, you present this to the left eye and this to the right eye. The way this is done is that you use what is called a stereoscope. I bet you most of you have seen a stereoscope. Everybody see a stereoscope? No?

So a stereoscope used to be something that was extremely popular starting in the 19th century. They created these handheld devices with the two lenses that you look through. And then they created a camera that had two lenses in it at the same distance, roughly, as your two eyes. And so it took a picture, thereby, creating two pieces, one by each lens.

And then, what you did, since the two have a slightly different perspective of what you're looking at, then the photographs are put into the stereoscope to look at. These two images are fused then. Looks like a single image.

And due to their very disparities, you see real depth. It's really dramatic. And when we talk about stereoscopic depth perception, I'm going to actually bring some stereoscopes here and some examples so you can see exactly what that is like.

But at any rate, in this case, what you can do is you can take a little area here just like before. And you can take the dots in this area. You move them a little bit this way, a few pixels. And you move this a bit this way, that way. And then if it's a square area, you're going to see a square sticking out.

And the monkey will then make a saccade to that. But with one eye, he cannot do anything. He can't see a thing because these random-dot stereograms only give you information about disparity.

So if you do that, you're going to have the monkey make a saccade to these. You can vary the degree of disparity. And when you do that, what you find is, quite dramatically, is that after a parvo lesion, when you block the midget system, there's a tremendous loss. The monkey essentially, especially at smaller disparities, has totally lost his ability to use stereoscopic depth information. So there is no deficit, however, after a magnocellular lesion.

So therefore, you can say safely that, especially at smaller disparities, what you have is the midget system that performs this remarkable task of seeing things in depth by virtue of disparity in the two eyes produced by stereoscopic vision. So that's really a very dramatic effect. Then the next thing we can look at is motion perception.

Again, this can be done in various ways, but I will just show you an example of it. So the monkey first fixates. Again, use random-dot stereograms here.

And then you set in motion a small square area. Ready? I hope. There we go.

Or you can do the high spatial frequency like that. And then, of course, on each trial, that appears someplace else. And you can vary the velocity or you can vary the contrast of the display and see how the monkey performs in the intact portions of visual field and how it performs in those portions of the visual field that are blocked either for the midget system or for the parasol system.

Clear? So let's think about it for a minute. What do you think you're going to get? Here we go.

Here we are-- motion detection. This is a parvo lesion. This is a magno lesion. And so what you get is a dramatic deficit, not an all-amount deficit, but a dramatic deficit in seeing motion after the magno lesion but not after parvo lesion.

So this says that, indeed, the parasol system plays a very important role in motion perception, not an exclusive role. There is still some performance there at very, very low contrast here. And there's a big effect at very high contrast, and monkey does better. So we can conclude that the parasol system is very important for motion perception.

So now, the next thing we can look at is flicker. And in this case, when you study flicker, instead of using a monitor, people used LEDs. Now, why do you think that is? Why don't they just use monitors?

Well, I mean, the reason for that is very straightforward, actually. When you use a regular monitor, what is the frequency of a monitor? It's the same as the frequency of the alternating current that you have, that shine the lights up here. But what is that in the United States? 60 hertz.

And so that's what happens on a regular monitor. And every 1 over 60 hertz is you shift the image. In Europe, the frequency is actually 50 hertz.

So at any rate, because of that, there's a rather limited range over which you can vary the ON and OFF activity of a flickering spot. So if you use any of this, however, you can use it in a huge, huge range of small steps. So, therefore, this is what the display looks like that people use to study this.

First, the monkey fixates here. And then one of these LEDs will start flickering. And I'll show you the flickering, which is not perfect here on this monitor, but it will give you a sense.

Everybody see that flicker? Now, the mean flicker value of that location is the same as the yellow lights that you see there. So if it flickers at a high rate, you can't tell that it's flickering because it's beyond the ability of the eye to resolve it, and then

you can't make a saccade to that location.

So if you look at that, what you find here, after parvo lesion and after magno lesion, you get a gigantic deficit after magno lesion. And that fits with what I told you in the beginning-- namely, that the midget cells respond in a fairly sustained fashion when the stimulus comes on. Whereas, the parasol cells respond transiently, which makes them much more readily available for motion and for flicker.

So those, then, are the major arrangements that we see with these various experiments. And so we can summarize what happens after parvocellular and magnocellular lesion and say what happens here. So this one here is when you block the midget system. This is where you block the parasol system.

And here are all the various test that had been used. And you can see that for the parasol system, the major deficit arises in motion perception and flicker perception. So those two are very important in the processing of the parasol system. Whereas, for the midget system, you get lots of deficits in color vision, texture perception, and fine pattern perception, fine shape perception, in contrast sensitivity, and in stereopsis.

So this, then, gives you a sense of what these two systems are important for in processing visual information. And so, what you can do next is to summarize what I just told you-- namely, that the midget system is important for color, texture, fine form, and fine stereo. The parasol system is important for fast flicker and fast, low contrast motion. Both systems are capable of doing brightness; coarse form; coarse stereo; slow flicker; slow, high contrast motion; and scotopic vision.

So it is not a simple arrangement. It's a complex arrangement. There is overlap in what both systems can do. So now, the big question comes up. Why was it so important to create both of these systems?

Well, the number of schemes that have evolved, certainly, the motion is obvious. But one thing one can come up with is a scheme of this sort that has been proposed-- namely, that what happened as a result of these two systems emerging,

your ability to process information has been extended. And for the midget system, you expanded the ability to see up to high spatial frequencies-- very, very important attribute. And I should add, also for the midget system, it became possible for you to see color.

Whereas, when you come to the parasol system, it expanded the range of your ability to process information in the temporal domain-- your ability to see very fast motion, to see flicker. Now, let me add here that's a very important attribute, by the way, for animals as well as humans because one of the common things that has emerged in the course of evolution is what is called camouflage. I think I may have mentioned that once before.

That is that when you or an animal, and the coloring of you, which is done in thousands of animals, is made similar to the background, then it's very difficult for you to see that particular animal. So it has an excellent camouflage. However, as soon as this animal begins to move, the ability to camouflage itself disappears.

And animals have to move. And that's because the parasol system is extremely sensitive to motion. And so whenever an animal begins to move, even though it has excellent camouflage, it is predominantly the parasol system that will destroy the camouflage effect. I'm not saying this is necessarily the only hypothesis, but this is one hypothesis that had been advanced to try to account for how sensible it is to have these two systems.

Now, of course, you could ask, how come this couldn't be just done in one system-- all of it? Well, there are several reasons for that-- is that to combine a situation where you have a sustained response and a transient response into one is next to impossible. And to have cells that are highly sensitive, or the parasol cells, you need to have a convergent input from several photoreceptors. But as soon as you have that, you lose your ability to see fine detail.

So these requirements to do this and this are antagonistic to each other. And it's not possible to do that in a single cell at the retinal level. So it was decided, inasmuch as they should decide, that it will be best just to create two separate systems however

complicated that might be.

So that, then, brings me to the overall summary of what I had covered today. First of all, I told you-- that's the obvious part-- are two major channels that originate in the retina are the midget and the parasol. I should add here that there are many other channels in the retina, but the cells that do that-- and we'll talk about some of those later-- are much less numerous.

The overwhelming majority of cells in the retina are either midget and parasol. Then in central retina, the receptive field center of the midget cells and parvocellular cells is comprised of just a single cone. The midget cells and the parvocellular cells-- meaning that the midget projects to the parvocellular layers. And I told you before that the midget cells of the retina have receptive fields which are quite similar to those, almost identical to those that you see in the parvocellular layers of the lateral geniculate nucleus.

Then the parasol cells have much larger receptive fields than do the midget cells. The cone input is mixed both in the center and the surround. And that is the reason why this system, the parasol system, cannot tell you anything about color, but it is very sensitive and can tell you about any change that occurs out there over time, irrespective of the kind of color it has.

Now, the midget and parasol cell ratio from center to periphery changes from 8 to 1 to 1 to 1. Now, I should reiterate that in that it is very important for us to be sensitive to motion in the periphery. That's if an animal is threatened by some predator, it is highly desirable to be able to very sensitive to any motion. And because of that, you have a higher number of parasol cells in the periphery than in the center. And so the ratio changes to become equal in the periphery.

Now, the midget and parasol systems converge on some cells in V1. And the example, the prime example, I've shown you when they were separately blocked at the level the geniculate, that the cell I showed you was one that did receive a convergent input. But many other cells receive single input.

V4 receives inputs from both the midget and the parasol cells. So it's not an area that only deals with color, obviously. And what it will deal with, we'll describe in more detail later. The major input to MT is from the parasol cells. And that, often, MT and MST have often been called the motion areas.

But they also play important role, by the way, in depth perception, as we shall see. And then the midget system extends the range of vision in the wavelength and high spatial frequency domains. The parasol system extends the range of vision in the high frequency domain.

And in the scheme that I showed you is what leads to this particular conclusion. So that, then, is the essence of what I wanted to cover today. And I am now certainly open to any questions that you might have about these two fascinating systems that have evolved in the retina over the millions and millions of years of evolution.

Well, I was so clear that there are questions, huh? Well, I hope that you sort of gotten a sense of what it is like to do these kinds of experiments and how luckily, at least, in some cases, these experiments can lead to nice discoveries as to the workings of the visual system. Now, last time when we talked about the APB, we did have a "magic bullet."

This approach here is not as neat, really, because you have to make lesions rather than specifically affecting certain neurotransmitters. And that could not be done because the neurotransmitters for the midget and the parasol cells are similar. So you can't use that kind of procedure that we were able to use miraculously for the ON and OFF channels. So that, in essence, is what we are going to cover today.

Did everybody sign? Any one of you not sign the attendance sheet? If not, please come up here and sign it.

And then let me just say again that next time, we are going to talk about color--another fascinating topic. And we are also going to talk about visual adaptation. And if you can, if you get a chance to do so, please try to read the preparatory material which should make it easier for you to not only comprehend but, especially, to be

able to memorize these facts.

And the memorization is a very important part of learning things in any course. And it will, of course, be essential when you get to the stage of having to take the exams. Well, thank you so much.