PROFESSOR: OK, I just wanted to go back over a few things to make sure you're understanding it right. When I talked about this anatomical plasticity in the hippocampus, you just have to realize that every one of these rectangles represents the very same slice, just showing different things in the same little bit of the-- as you see here. It shows a slice in the dentate gyrus but just through the dendrites of the dentate gyrus from the peel surface down to the cell bodies.

So here it's showing one kind of connection, here it's showing another kind of connection, and it shows what happens if you remove one of them. You get [? sprouting ?] of the other one. Yes?

AUDIENCE: [INAUDIBLE]

PROFESSOR: It means that this particular projection doesn't go there, but the projection here from the entorhinal cortex doesn't terminate in the inner part of the dendrite near the cell body. It terminates in the outer 2/3 whereas the association fibers coming from other parts of the hippocampus, they terminate in that area. So in that rectangle is white, the upper 2/3 is white.

So obviously there's some kind of specificity in the way those terminals grow in. Maybe in development they do overlap, but in the adult, they don't. But then if you remove one of them, the other one will sprout. And I'm only showing true projections. There are other projections like the acetylcholine axons coming from the septum. They change too after you make one or another lesions of these [INAUDIBLE].

OK and then the point about this more modulatory states and how it changes with different stages of sleep postulated to really change the flow of information between the hippocampus and the neocortex. I just wanted to point out that we know that these are only three of the modulators that can change the state of the brain. So if this theory is correct, and probably is at least partly correct, it just indicates that when brain state changes, can pretty drastically change the way connections are working where the information is flowing in the cortex.

And remember when we-- in chapter 17, I talked about how many different possible brain states there are. It means this kind-- this is just the beginning of this kind of model to represent information flow in neocortex.

AUDIENCE: [INAUDIBLE]

PROFESSOR: Sure.

AUDIENCE: [INAUDIBLE]

PROFESSOR: That's right.

AUDIENCE: [INAUDIBLE]

PROFESSOR: Yes, in fact, in REM sleep, it doesn't mean there's nothing important going on. We know that dreaming is occurring. You know, people argue all the time about what the function of that might be. We also know that if you're in dream sleep, when you're awakened, you tend not to remember. You can train yourself to remember, because if you keep it in mind, you keep rehearsing after you get up, you can report a dream. And that's why in studies of dreams, it's not very reliable to talk about somebody a half hour later after they get up, because they will forget most of it.

So that has something to do with these states too. But there's really a lot that we just don't understand, but we do know that there's different states of our consciousness where we don't-- there's a lot of dissociation between. And in abnormal cases, you can get more than one personality existing in the same person, the same central nervous system. That has to involve these different kinds of space, but we don't understand the brain mechanisms behind that kind of dissociation. Yes?

AUDIENCE: [INAUDIBLE]

PROFESSOR: You would like to know what the neurochemical coeruleus are, and I can't tell you. You can look in the lecture to see if anybody's even tried. And since you can get REM sleep in animals, you can do these REM sleep deprivation studies in animals, it seems to me that you could certainly look for things like that. There might even be changes in the cerebral spinal fluid.

As we know, you can inject cerebral spinal fluid from one animal that's sleeping to an animal that's not, and the animal that's not will go to sleep. So we know there are effects even there, but that kind of research is just not done. One of the things that neuroscience like to-- people work on what's popular. It takes an enterprising graduate student to dive into something like that.

These pictures of the human brain are just to help orient you to these same structures in our own brains. You can begin to see why we use these rodents. They're much more like the embryonic human brain, because the human brains have grown so big and the cortex, especially, has grown so big that it distorts the whole appearance.

This here shows the hippocampal formation in fornix, and the part of the hypothalamus memory body where the fornix terminates. And you can't tell from a picture like that, but this is definitely the brain stem, the upper brain stem and this is up in the hemisphere, but in fact, that's what it represents. And I just sketched here the hippocampus anterior, posterior, and the fibers of the fimbria in the fornix coming out of the hippocampus and becoming the columns of fornix here. I don't show the ones that go into the septal area which would be right there. I do show the commissure [INAUDIBLE].

OK and then this is a little more like a rodent, but this picture was a reconstruction published in a human neuroanatomy book by Nolte which tries to depict the human hippocampus. And it's not too hard to see the close relationship, but the same structures we see in the rodent we can find in a human.

I want to talk about the limbic-- other parts of the limbic forebrain other than hippocampus in the hypothalamus, especially the amygdala and the basal forebrain. They're closely related to each other.

This is Chapter 29 in the book. Let's just review a couple terms to start with. Where does that term rhinencephalon come from? What does it mean? It means nose brain. Well that was an old term used for all these structures that we now say are the limbric endbrain.

The term limbic system didn't start to be used until after Papez's publication and then interest that that engendered from other anatomists. The neuron anatomists began specifically studying these connections, especially with Nauta introducing a more sensitive method for studying connections in animals. We talked about that in chapter-- way back in the beginning, Chapter 2.

And now, of course, we have many, even more sensitive, methods involving various tracer substances.

But anyway, this is the Papez story, and I just point out here that some anatomy books, including one that I've often made use of, [INAUDIBLE], he-- in an earlier version of this book he had this nice description of Papez's work, and then in the third edition he eliminated. The question is why? Well he was trying to get rid of a lot of the history, because he didn't want the book to be too long. So lend it to what medical students will read.

But there are other reasons too. There's different kinds of grandmasters. Some of them are more interested in the structures, where they live, how you can separate them. But they don't focus on the connections as much. Neurologists too. We don't know enough about functions, and they don't want to try to group things.

So it's really the people, the Nauta type of people-- and there's a lot of us, because now they train a number of people, and we've trained additional people-- that had led me and other people to sort of talk more about [? Papez ?] and the limbic system in general.

Remember that [? Papez ?] took that term, rhinencephalon, and analyzed it and said a lot of these structures aren't actually involved in olfaction from the human clinical cases that he was familiar with. This was back in the 1920s and 30s.

But he did find that all of these structures were involved in seizures that affected the motion and that autonomic nervous system. So he said it seemed to be an interconnected-- he could define what he called an interconnected serious of cell groups that were concerned with feelings and emotional expressions. He emphasized that aspect of it. He could have added autonomic changes.

But what it led to was new thinking about our ability to group structures according to relationships in their connections, and that led to a colleague of Nauta's, Paul [? McCrane ?] at NIH giving the system a name, the limbic system. He based that on Broca's term from the 19th century. We talked about the great limbic lobe. And we've seen that in the medial views of the human brain.

And more recently, the reason for a lot of revival of interest, of course, is because we know it extends-- the functions of this interconnected system extends beyond mood and emotion plays a major role in spatial cognition and the formations of specific memory for places and events in our lives, not just in humans of course but in animals too.

This is the Papez circuit, slide we saw before. Papez described these structures that you see in red here and the network diagram that I made up where I was trying to include more recent findings of how these structures are related to each other. Some of them are exactly as Papez described, mammary bodies to anterior nuclei to the thalamus to the cingulate cortex.

Now in Papez's time, cingulate included some of the more caudal areas that now are often named differently, the retrosplenial cortex and other paralimbic areas that extend right down to the entorhinal cortex.

He, of course, separated the entorhinal area from the cingulate, but what looks like posterior cingulate, posterior, part of a singular gyrus we now call retrosplenial cortex. And there's some other names too used for components of it.

OK and then I'm showing how from the hippocampus itself, there are connections, back to the mammary bodies that Papez knew about. Remember, he didn't have the methods to know what was direct and what was indirect, but he did see that bundle. You could even trace them into sections, and you could follow that bundle all the way to the mammary bundles. But now we know there are many other connections of those axons as I've shown here.

So what's dominant input if it's not olfaction as Papez realized? You can't just say, well emotions are the dominant input. Feelings are the dominant input. That's crossing realms here from behavior to anatomy. I want to know anatomically, what's the dominant input?

Well in mammals the dominant input is here. It comes from the neocortex, because these association areas, particularly the multimodal association areas, the areas that have grown the most in the more recent human evolution, and in that period our brains have almost doubled in size. They are closely connected to singular areas, retrosplenial cortex and other paralimbic areas and to the entorhinal cortex.

And often you will see in textbooks that this projection of entorhinal area into the dentate is the dominant input. I point out here that there's connections to the subiculum as well. But the dominant input, by way of these paralimbic structures comes from neocortex. The

So now how do we get from neocortex to the hypothalamus? I'm asking here, what are the most direct routes, monosynaptic and disynaptic from neocortex to hypothalamus. Well you can look at the page circuit diagram and see their connections, but not so direct between neocortex and hypothalamus seen those diagrams.

But I've taken Mycelium's box diagrams where he's depicting transcortical connections from the most specialized cortical areas, the primary sensory and lower areas, to association areas which get connections from those primary areas. You have then the modality-specific association areas and the multimodal areas. Those are the areas closely connected-- adjacent boxes here are interconnected, two-way connections between those areas. So for every one of these you could draw arrows like this, and sometimes when I show this I do show that kind of arrow.

And then you have the paralimbic areas and then finally the limbic areas which are the other cortex and some subcortical regions, he calls corticoid because they have some layering. But I've drawn in there in red arrows direct connections or disynaptic connections to the hypothalamus.

So, for example, we know the amygdala which is directly connected to the hypothalamus as well as through other areas of the limbic system, the amygdala gets direct input from association areas, multimodal especially, but even unimodal visual areas project onto the amygdala as discovered by Nauta.

And then we also know that cingulate gyrus, parahippocampal areas, they project to hippocampus and to other of these limbic system structures which are all closely-- we know by definition, these are the areas closely connected to the hypothalamus.

So let's focus mostly on the amygdala, and ask, what the heck is it? What does amygdala mean? Well it means almond. It's got an almond shape. Oh I shoot. I already deleted one of the other slides. I had a human brain there that shows-- I posted some slides somewhere online. I'll have to find them. You can see in a human dissection how apt a name that is for the amygdala.

It's an almond-shaped structure that sits right in front of the anterior pole of the hippocampal formation which in a rodent would be the ventral pole. You can think of it as kind of a modified part of the corpus striatum. In fact, we call, in the classical anatomy literature, we call the basal ganglia the corpus striatum and we the amygdala, al called basal ganglia.

Now actually, in embryological terms, only part of the amygdala is striatal in origin. Some of it's paleo in origin. In other words, it's like a cortex.

But there's a very good reason-- some people have argued though they're actually separate structures. We should talk separately about them, but I would say no, because connections are what determines functions. And the connections of the amygdala indicate that it's one structure. They are closely connected, and they have common outputs.

And as in the striatum, connections in the amygdala are [? elastic. ?] Remember I made a big deal out of that for striatum. It had originally olfactory heads were formed. It was the route through the ventral striatum, the oldest part of the striatum to influence movement which it did through the hypothalamus and midbrain. Well this part of the striatum is not concerned with the same kind of movements. It's concerned with motivation, emotion, feelings, if we talk subjectively about it.

So associations are formed between perceive objects and sounds on the one hand and apex and autonomic changes in the other. It gives objects in the world valences or affected tags. In other words, you can hang tags on things. I like that. I don't like that. That makes me happy. That upsets me. But we'll all learn things or most of it. There's argument about some of them like fear of snakes. They might be at least partly inherited.

This is the collection of structures that we lump together and call the amygdala in a ventral view of a rabbit brain which is very similar to rodent brains. The connections are all the same.

So here's the olfactory bulb way up at the front, but behind the olfactory bulb starts the olfactory cortex. The first part of it we call the olfactory peduncle, because it's sorts of a little stem connected to the rest of it. And you'll see they have these very widely projecting axons. They're very widely branching axons of the olfactory system, and they go back to this broadened part of that cortex which is why it gets the name pyriform cortex-- it's P- Y- R, not P- I- R. It's not the [? piricortex ?] It's the pear-shaped cortex, because this is the pear shape like the Bosc pears you buy in the supermarket. That's the shape it is.

And this little special part of the olfactory bulb the olfactory bulb projects directly to the cortical nucleus to the amydala. So that's in red here. And medial nucleus connects to it, also gets direct olfactory input. That part, the cortical and medial nuclei are often lumped together even though one of them is paleo in origin and one of them is striatal because of the similarity of connections and because they get, not only direct olfactory input, they also get some input from the brainstem from the taste system, directly from brainstems from the parabrachial nuclei, so you've got taste information. It also gets pain input from the brainstem.

And the basal [INAUDIBLE] nuclei, they get input from sensory systems like the visual system. Some of it comes directly from the thalamas, especially in the auditory system but there are some visual pathways too, but even more, especially for the visual system from cortical areas.

They also have projections to cortex, especially to the prefrontal area. So the prefrontal area recipricolly connected to this, but all of these nuclei are connected to the central nucleus and have some other connections with each other.

That central nucleus which is a striatal structure, striatal in origin, it gets input from all these different structures and it connects directly to basal forebrain and hypothalamus. So we want to look at that.

So here I've just shown paleo and striatal [? derivatives ?] separate. I don't think it's such a big deal, because [INAUDIBLE] connections. It's really difficult to separate them in that way.

So let's describe the stria terminalis Its origins, I just stated, comes from, mainly, the central nucleus and the amygdala. And then it's course in its major connections, and you'll see that its course is very much like the fornix fibers from the hippocampus. It has to go around the internal capsule fibers. And by going directly to the amygdala, it pretty directly influences motivational states.

I want to start talking about it, just by showing a section of the rat here. This is the amygdala. I put dotted lines around it. This is olfactory cortex out here. There's the rhinal fissure. We look in the hypothalamus. There's the fornix, and I pointed out the ventral medial nucleus, because this central nucleus, which is here, can you see my pointer here? This area projects directly to the cells, the neurons, of the ventral nucleus hypothalamus. And we want to follow those.

Here's the stria terminalis. I've outlined it for you. And you'll say, well how do you know that? Well If you look carefully here, you will see-- you'd have to blow it up a bit I guess to really see it, but let's do that. These are neurons. These little cells are oligodendro cells. The basic dye will stain the region of the nucleus in the oligodondron sites, and you'll see these little tiny cells all lined up along the myelinated axons. You see them here too, but here they're going differently.

These axons here are going like this and coming out of the thalamus or coming from cortex and going down like this into the cerebral peduncles. And this is the white matter of the cortex up here. So all these axons are connected there. We go right up here, and here they go. This is white matter of the cortex.

So here then are the fibers that are-- you can see them here coming out of-- here they come here. So now I've just drawn a line around here. So why do you see two of them? Because these axons go around the caudal end of the internal capsule. They go around. They're the same axons you see here, you're seeing again up here. Just like if we were more caudal in the brain, we would see hippocampus going all the way down ventrally here, and with the axons coming out of that hippocampus are mostly collected in the fimbria of the fornix. And there's the fimbria of the fornix up here. And in this dorsal part of the hippocampus, the fornix fibers are there.

So there's fimbria with the other fornix. And all those fibers are collected there. Where else do we see those axons in this section? Yet those axons go around the internal capsule too. But then where they go? They come down in from of the thalamus, they go into the septal area, but some of them go more caudally.

Where are they going? This is Papez circuit. They're going to the hypothalamus. Where in the hypothalamus? I'm going to stop right here until one of you tells me. Where's the hippocampus project in the hypothalamus? It's Papez circuit. Come on.

Mammillary bodies. And there's the fornix fibers. And then those fibers from the amygdala are doing something similar. Here they are. There they are. Where are those down here? They're here in that light area around-- because the dendrites of the cells in the ventromedial nucleus protrude out into that light area where you see the clumped cells.

So the cells here in central nucleus go up, around the back of the internal capsule, then they follow around, come down through the basal forebrain, and run caudally just like the fornix. Here's our view of the-- medial view of the hemisphere where I've pretended it was transparent, so I can show you these structures.

There's the amygdala right in front of the ventral end of the hippocampus in a rodent. There come those axons. They're going around the internal capsule which I've cut off here. I've cut them right there, but they're terminating in the bed nucleus stria terminalis By definition, that's why we name it the bed nucleus, the stria terminalis, because those cells are right among the axons of the stria terminalis and they're getting connections from it.

And similarly, the hippocampal fibers-- you see there I left those in black-- the columns of the fornix are right there next to the stria terminalis. They go to mammillary bodies, runs from the stria terminalis, though mainly to the ventromedial hypothalamus.

There are other connections in the hypothalamus too of both those bundles of axons, but the major termination are in the mammillary bodies in the ventromedial hypothalamus.

So this whole area is basal forebrain. We'll talk a little more about that. In the frontal sections here, these are the levels through the embryonic brain and here we're in the mid thalamic region. There's the amygdala down in the temporal lobe.

And there's the stria terminalis coming around the internal capsule.

And there's the bed nucleus, the stria terminalis. I cut it here right at the rostral limit of the bed nucleus and the caudal limit of the nucleus accumbens. Nucleus accumbens has become very big in humans. It gets a lot of input from the amygdala and the hippocampus, especially the hippocampus and other limbic system structures.

And if we go forward, we just see nucleus accumbens, the major nucleus of the basal forebrain. Accumbens means leaning, because it's leaning against the septal which is here.

A couple more questions here. What sensory inputs come to the cortical and medial nucleus and the amygdala without passing through the neocortex? We know they're very ancient from comparative studies. We keep seeing it even in really ancient vertebrates, vertebrates that is, that are still living but are very similar to vertebrates that lives hundreds and thousands of years ago.

OK olfactory is certainly one of them. What's another one you'd expect? What was really important in getting animals to do one thing rather than another, is a source of reward important in feeding? Taste. Yes.

And there are taste inputs directed from the brainstem too. Olfaction and taste and the other one is pain-- all come in there. Those are the ancient inputs to the cortical and medial nuclei.

And then the question for us is the lateral nucleus the amydala receives various sensory inputs too, but they come by way of neocortical associations and a few of them directly from the thalamus but not from more caudal brainstem structures. And I showed you in the diagrams before those-- you think most about the visual system ones, but we know auditory system ones and we will come back to that. So here's what I just said. I also pointed out that basal, particularly the basal nucleus, the lateral somewhat, reciprocally connected to prefrontal cortex.

This is that picture when we talked about auditory system from experiments where they labeled the cells in the medial colliculus body, and they traced the axons not only to auditory cortex, there here in the opossum but in the opossum very heavily to that lateral nucleus in the amygdala. But it also occurs in the hedgehog, and the treeshrew and the rat and the mouse. Probably in us although the connection to neocortex in the primates is by far the most important.

I want to show you again what medical school illustrations can look like. To make it a little easier, just so you understand what they're doing here. They're trying to show too much, and because of the way the human brain's developed it's very distorted. This kind of picture is a lot easier to understand. This is the human, and I've shown inputs by labeling the cells red. The outputs I've shown the cells in the amygdala with a green dot so you can see. But a lot harder to understand than the way I'm presenting it.

Here I'm in a dissection-- a big slice so you can see the myelinated fibers really clearly. This is from a book, beautiful book, that shows-- I can bring it in class and let you go through it-- showing beautiful dissections, and then he also has some of these sections.

There you see the amygdala. This has to be-- I'll ask you what level this cut. This is thalamus here, and there's mammillary bodies there. If it were a rodent, this would be caudal thalamus, but in human, the way it's arranged, you can't count on that.

There's the putamen, the caudate nucleus, but there's that big amygdala. And you'll see it's a abutting the more anterior parts of the corpus striatum right there. That's all amygdala and if we went back just a little bit, then we'd be in hippocampus, because that's got to be the anterior most part of the temporal lobe here. See there's the [INAUDIBLE], so this is all temporal lobe down here. This is parietal lobe up here. Or if we're very far anterior, it might even be coronal cortex.

Now some of the early functional information around the amygdala functions came from electrical stimulation, and it's been done a lot in monkeys, but it's also been done in humans during surgical procedures. So I've taken this from human studies. You stimulate the cortical medial area, you get oral kinds of things, salivation, smacking of the lips, licking, chewing, and inhibitional involuntary movements as well. You can also get elimination behavior.

If you stimulate the basal lateral nuclei of the amygdala in a human you get arousal, attentiveness, sometimes you get fear or rage, or you get anxiety and another very strong emotions, and you also get effects on memory. The person will say, I've been here before, and of course he hasn't been here before, but it feels like he has. It's an experience that many of us have when we're growing up. It's called a deja vu experience. How many of you can recall having such experiences? You've been in a situation, you're sure you were there before, but you couldn't have been there before. You know? You're in a new place.

Well we think it probably happens through a minor seizure. It doesn't mean anything's wrong. It doesn't. Because these cells in the limbic system are prone to getting overly excited. People with temporal epilepsy can have deja vu preceding an actual seizure. But most of us, it doesn't go that far. I kept having those until, I think I was 30 years old.

In the research, negative emotions have been stressed. And when you hear talks about amygdala, it used to be they would only talk about learned fears. That's changing a bit now, and I know from the human literature that it had to change because, in fact, positive feelings, as well as negative, can be obtained from manipulating that area.

If you take the amygdala out, there's not much on humans, but occasionally there are humans with amygdala lesions, generally not bilateral though. It has been done on both sides with monkeys. And the first thing you notice-- monkeys, rhesus monkeys are pretty wild. We were getting rhesus monkeys that were wild caught. Then they started using only the ones that are bred in captivity, but even the ones bred in captivity, they grow up in these groups and they are not tame monkeys.

But after the amygdala lesions, they were remarkably tame. They lost their normal defensiveness and aggressiveness in social situations.

But the monkeys in social groups become socially isolated, because they're not sensitive, the way they should be, to dominance relationships. So you now have this monkey who's not a dominant animal at all, or he wasn't before the amygdala lesion. He will walk right up to the dominant male and get clobbered by him. So they get injured, they become isolated just because they lose their normal fears, especially in response to visual inputs.

They show loss of autonomic reactions that accompany fear and anxiety. Also, you see some other types of effects like altered dietary preferences. And in the animal work, this was true also, the cats get hypersexuality. There's a famous picture that used to be reproduced in all the physiological psychology or brain behavior books of a animal mounting another animal who's mounting another animal who's mounting another animal because they had amygdala issues.

The amygdala, like the striatum is involved in habit learning, but what kind of habits are we talking about now? Well, I just described learned fears and other kinds of learned [? effects. ?] That can be considered a habit.

Prejudices are learned. That is an emotional habit, something learned. It's a tag we put on certain things or even certain people. That depends on the amygdala and basal forebrain, particularly the amygdala because its close connections to the temporal lobe.

This is from a study of fear conditioning that was found to be dependent on the amygdala in rats. So here you have a rat in a box with a grid they can electrify and there's a speaker here and they're sounding a tone. And you can see in response to the tone-- the left graph here is blood pressure, doesn't change very much. This is the time he spends resting his behavior, freezing behavior, very little.

When the sound is [? novel, ?] you'll get a little bit of increase in blood pressure, a little bit of freezing right at the beginning, and then he gets used to it and you don't get in anymore.

But now, when you get the sound, you electrify the grid, as you'd expect, blood pressure goes way up. He freezes a lot because he can't get away. Of course, he tries to run, but he can't get away.

They don't shock them so much that they're really injured. It's not a super cruel experiment, and it's often been used to study fear condition. But anyway, after they learn that association, now you just give the sound, you don't electrify the grid. Blood pressure goes up. Freezing goes up. That's learned fear, and that's how it's measured.

[? Ablation ?] amygdala or just damaged pathway from the medial [INAUDIBLE] body of the thalamus to the amygdala, you get rid of that. There's pretty good evidence that depends on that auditory pathway from the thalamus to the amygdala to the lateral nucleus to the amygdala.

There's interesting lesion experiments that have been done in monkeys. I want to go through this quickly. How do you produce a monkey that shows all these bizarre effects of amygdala ablation only when he's looking with one eye but not when he's looking with the other.

AUDIENCE: [INAUDIBLE]

PROFESSOR: Louder.

AUDIENCE: [INAUDIBLE]

PROFESSOR: So you do a split brain operation meaning cut the commissures, cut the optic chiasm-- see, if you cut the optic chiasm, you still have ipsilateral fibers you haven't cut, the ones that don't cross. Like here, here's the chiasm, you cut around the midline. So now the right eye goes to the right side of the brain, the left eye goes to the left side. Of course, each eye sees only half the visual field.

But to get this to work you start just with the amygdala lesion in one side, the left hemisphere. I'm looking at the top, but I'm showing where the amygdala is down below in the temporal lobe.

So you have to not only cut the chiasm, you have to cut the commissures, because otherwise the visual input that reaches this visual cortex can go from the [? juxtastriat ?] areas here across to the other side, because these areas are interconnected across the chiasm. The reason they cut it all the way forward is they actually have to cut the interior commissure to get this to be complete, because the amygdala's connected to the amygdala area. Temporal lobe structures, even temporal unicortex is connected across the interior commissure too.

So they cut these commissures and now the animal has a totally different personality with one eye than with the other. We call that a disconnection syndrome. Disconnection syndromes have become well known in human neurology, because many lesions, human strokes, can eliminate a particular group of axons, a particular pathway.

So the person can lose his ability to read, for example, but he can see perfectly well and he can respond to objects, but if you've disconnected the pathway going to areas, the Wernicke's area of the temporal and posterior parietal cortex, then he won't be able to read.

You can read the rest of this, and I describe it in the chapter. Learn about how aggression-- these are all associated with amygdala functions-- we know aggression is also highly dependant on amygdala even though it's also generated through the hypothalamus of course.

The endbrain structure that's most closely connected to it, in fact, it's a major function of that connection from amygdala to the ventromedial hypothalamic nucleus. And you can stimulate the ventromedial nucleus hypothalamus and get aggressive behavior, but that is enhanced by testosterone. This is just an experiment that shows males, high aggressiveness-- it's low in females over here-- but if you castrate them, their aggressiveness goes down. This is studies of rats, but it's been done on a number of other species as well.

Then you start administering the testosterone which affects the amygdala. Then aggressiveness comes back unless you drop the level of testosterone you're administering to low levels.

Read about the basal forebrain. I think that we will have time to go through basal forebrain, and I want you to understand this hypothesis, because it relates to Chapter 13, studies of plasticity. | makes a prediction that has been verified indirectly in studies of humans after Hurley temporal lobe lesions in certain groups of schizophrenics. This is from one of my papers, a paper published in 1979 I think. We'll come back to that next time.